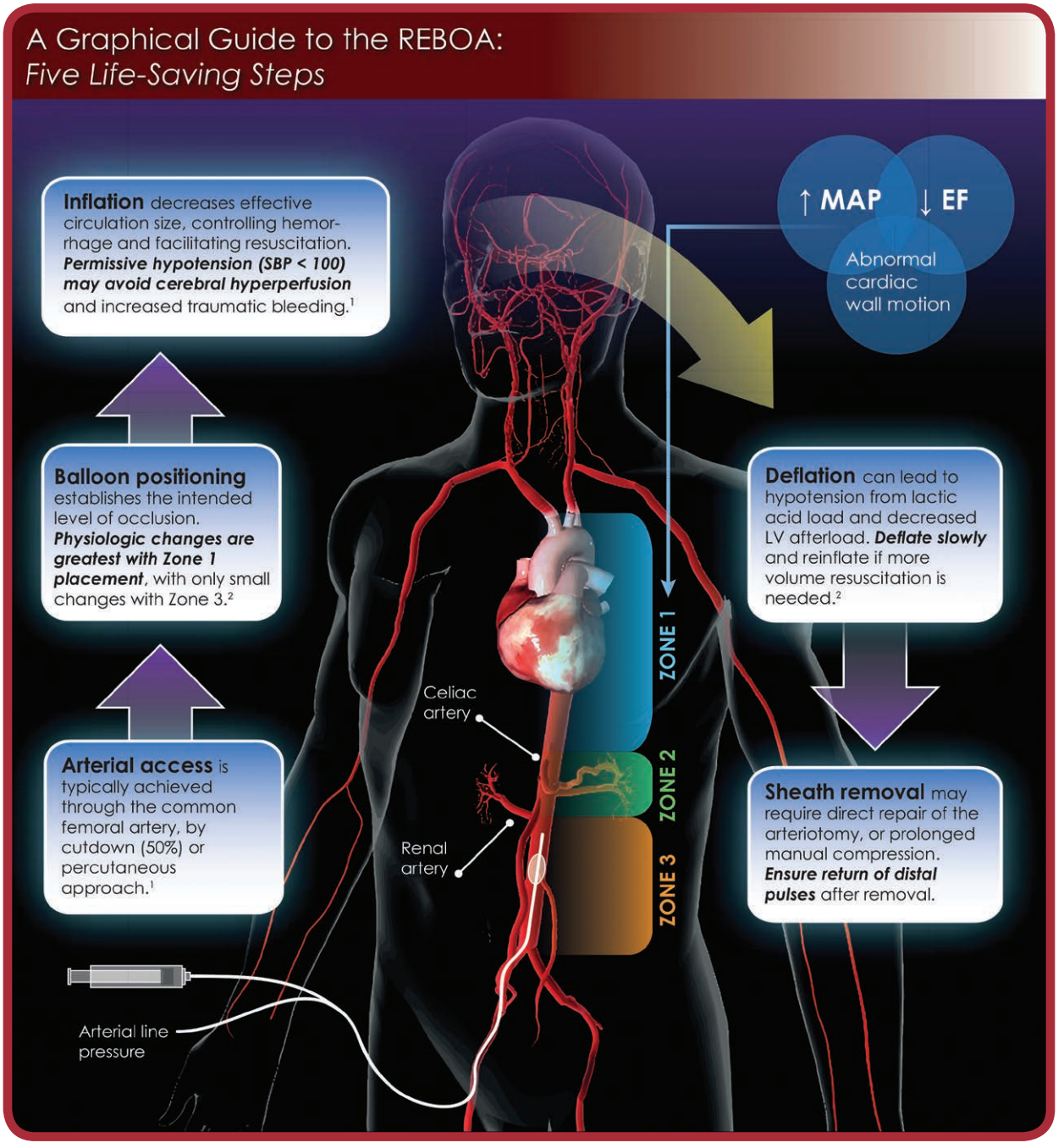
ANESTHESIA & ANALGESIA® Infographic



The introduction of resuscitative endovascular balloon occlusion of the aorta has the potential to dramatically affect the management of noncompressive torso hemorrhage. Early results from clinical application of this new tool have been promising, although high-quality data are still needed to understand the best clinical management strategies. In this infographic, we review the essential steps for resuscitative endovascular balloon occlusion of the aorta usage and the associated considerations for anesthetic management of this patient population.

EF indicates ejection fraction; LV, left ventricle; MAP, mean arterial pressure; SBP, systolic blood pressure.

The Infographic is composed by Jonathan P. Wanderer, MD, MPhil, Vanderbilt University School of Medicine (jon.wanderer@vanderbilt.edu), and Naveen Nathan, MD, Northwestern University Feinberg School of Medicine (n-nathan@northwestern.edu). Illustration by Naveen Nathan, MD.

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2. Qasim Z, Sikorski R. Physiologic considerations in trauma patients undergoing resuscitative endovascular balloon occlusion of the aorta. *Anesth Analg*. 2017;125:891–894.

IARS International Anesthesia Research Society

Resuscitative Endovascular Balloon Occlusion of the Aorta: A New Weapon to Combat Exsanguinating Hemorrhage

Richard P. Dutton, MD, MBA,* and Frank Herbstreit, DrMed†

Hemorrhage is a leading cause of death from trauma, as well as from obstetrical emergencies and misadventures in elective surgery. While visible bleeding is easily managed by compression and vessel ligation even under battlefield conditions, noncompressible torso hemorrhage remains a lethal problem. Noncompressible torso hemorrhage can result from pelvic or retroperitoneal trauma, uterine disease or injury, or misplaced hardware in orthopedic procedures. Surgical visualization of injuries in the deep pelvis is difficult, and profuse bleeding can occur from a network of veins that offers no easy site for vascular control. Angiographic embolization is possible in modern facilities, but even in the best-equipped centers it can take time to mobilize the necessary resources. Too often the patient bleeds to death or sustains a lethal dose of shock before hemorrhage can be controlled.

During active bleeding, time is the enemy. Two review articles in this issue of *Anesthesia & Analgesia* describe a new technique for temporizing lethal hemorrhage, one that can minimize critical hypoperfusion during the time needed to definitively address the source of bleeding through surgery or invasive radiology.[1,2] Resuscitative endovascular balloon occlusion of the aorta (REBOA) has emerged in recent years as an adjunctive therapy in major trauma cases and has been supported by development of new technology and protocols.

Briefly, REBOA consists of rapid access to the femoral artery, with placement of a balloon-tipped catheter in the descending aorta. Inflation of the balloon leads to partial or complete obstruction of the aorta, with significant reduction in lower body arterial pressure. The resulting low-flow state facilitates native hemostatic mechanisms and prevents washout of fragile early blood clots. The aortic level at which the balloon is inflated can be estimated initially, then fine-tuned by ultrasound or fluoroscopy as time and patient condition allow. Unlike the all-or-none thoracic aortic cross-clamp of the open chest cardiac arrest, often as physiologically damaging as the trauma itself, the REBOA balloon can be adjusted in both location and inflation to minimize bleeding while preserving some perfusion.

The review articles on REBOA in this issue of *Anesthesia & Analgesia*, from 2 of America's busiest trauma centers, provide more details on indications for REBOA, technical considerations in placement and maintenance, physiologic changes induced by balloon inflation and deflation, and early clinical evidence of effectiveness. A parallel article in this month's *Anesthesia & Analgesia Case Reports* illustrates the use of REBOA as part of damage control resuscitation in the first minutes after a severe injury.[3] Each of these articles emphasizes the importance of close communication among the members of the trauma team during use of REBOA—the anesthesiologist is integral to good management. Physiologic changes with balloon inflation and deflation can be dramatic—if the team is not coordinating then wide swings in vital signs are likely, with the potential for cardiac arrest on the one hand and pressure-induced rebleeding on the other. As a properly integrated component of hemostatic ("damage control") resuscitation, REBOA will save lives that are being lost today. Future studies will determine the best indications, further improve available devices, and compare the endovascular technique to the traditional open approach to aortic occlusion.

Use in the trauma population may be just the beginning. With equipment on hand and experience in our heads, REBOA could be applied to patients with abdominal or pelvic bleeding from many different causes, including unanticipated obstetrical hemorrhage, vascular surgery catastrophes, and inaccessible spinal tumors. As such, every anesthesiologist should be aware of the principles and physiology of REBOA, and every large hospital should have the necessary equipment on hand.

Perhaps more notable than the technique itself is what these articles represent: the increasing recognition in the United States of the subspecialty of trauma anesthesiology. While all anesthesiologists must have familiarity with the basics of resuscitation and life support, trauma specialists bring added value through their deeper understanding of mechanisms of injury, trauma patient physiology and the logistics of team-based care during both the golden hour of early resuscitation and the subsequent days or weeks of critical care. In European countries, of course, anesthesiologists have always been involved in prehospital and emergency care, but this tradition has only just been rediscovered in America. The Trauma Anesthesiology Society (www.tashq.org) was founded in 2011, and now boasts

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an international membership of several hundred enthusiastic clinician scientists, including the authors of these reviews. The Society has been instrumental in establishing and supporting a Trauma Section in *Anesthesia & Analgesia*, and we are proud to have served as the Executive Section Editor and Senior Editor for its inaugural year. We hope to bring to light many more examples of emerging science in trauma care, with particular emphasis on the role that anesthesiologists can play in delivering good outcomes for our patients.

DISCLOSURES

Name: Richard P. Dutton, MD, MBA.
Contribution: This author helped review the literature and write the editorial.

Name: Frank Herbstreit, DrMed.
Contribution: This author helped review the literature and write the editorial.
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NARRATIVE REVIEW ARTICLE

CME

Resuscitative Endovascular Balloon Occlusion of the Aorta: Principles, Initial Clinical Experience, and Considerations for the Anesthesiologist

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Resuscitative endovascular balloon occlusion of the aorta (REBOA) is an endovascular technique that allows for temporary occlusion of the aorta in patients with severe, life-threatening, trauma-induced noncompressible hemorrhage arising below the diaphragm. REBOA utilizes a transfemoral balloon catheter inserted in a retrograde fashion into the aorta to provide inflow control and support blood pressure until definitive hemostasis can be achieved. Initial retrospective and registry clinical data in the trauma surgical literature demonstrate improvement in systolic blood pressure with balloon inflation and improved survival compared to open aortic cross-clamping via resuscitative thoracotomy. However, there are no significant reports of anesthetic implications and perioperative management in this challenging cohort. In this narrative, we review the principles, technique, and logistics of REBOA deployment, as well as initial clinical outcome data from our level-1 American College of Surgeons–verified trauma center. For anesthesiologists who may not yet be familiar with REBOA, we make several suggestions and recommendations for intraoperative management based on extrapolation from these initial surgical-based reports, opinions from a team with increasing experience, and translated experience from emergency aortic vascular surgical procedures. Further prospective data will be necessary to conclusively guide anesthetic management, especially as potential complications and implications for global organ function, including cerebral and renal, are recognized and described. (Anesth Analg 2017;125:884–90)

Despite numerous advances in the management of trauma victims with significant hemorrhage, early mortality due to exsanguination remains high, with lethal hemorrhage identified as the most common cause of mortality. In patients with potentially survivable injuries, noncompressible truncal hemorrhage (NCTH) is the most common source of bleeding.[1] These findings exist in both the civilian and military populations despite differences in mechanism of injury.[2] Although innovative approaches to management are well described, including prehospital treatment and rapid transport, damage control resuscitation, and accompanying surgical concepts, morbidity and mortality remain high. Aggressive management of perturbations in temperature, acid–base status, and the coagulopathy associated with trauma have been mainstays of current therapy.[3–7] Simultaneously, there has been a rapid increase in the development, utility, and success of endovascular techniques, initially in the cardiac surgery, cardiology, and interventional radiology arenas.[8–10] This concept has translated into algorithms for the trauma population, and anesthesiologists will increasingly encounter use of resuscitative endovascular balloon occlusion of the aorta (REBOA)[11] as local studies continue under the guise of the American Association for the Surgery of Trauma.[12] REBOA registry data investigation is also underway in Asia (eg, Japan Trauma Data Bank) and Europe (Endovascular Hybrid Trauma bleeding Management, based at Orebro University, Sweden).

In this narrative, we describe the principles, technique, initial clinical experience/data, and implications for anesthesiologists caring for patients with REBOA, as there are insufficient available data to conduct a robust systematic review or meta-analysis. We will primarily discuss the use of REBOA in the trauma population for this review, but endovascular aortic occlusion (AO) has become more common in other scenarios of NCTH, including aortic aneurysm rupture, cancer, and obstetric surgery.

REBOA PRINCIPLES

Noncompressible hemorrhage in the thorax, abdomen, or pelvis usually requires prompt access to the operating room (OR) and all damage control resuscitation components alluded to above. The goal of REBOA is to assist resuscitation with temporary restoration of aortic blood pressure via an endovascular balloon inserted via a femoral arterial approach. Inflation of the balloon in the descending aorta has been shown to maintain cerebral and myocardial organ perfusion, allowing for temporal correction of physiologic, coagulation, and blood volume abnormalities while surgical hemostasis is being achieved. Depending on the location of the endovascular balloon inflation, bleeding itself from a vessel or solid organ injury may be decreased secondary to a decrease in arterial inflow. This is a similar concept to the traditional aortic cross-clamp maneuver, which is performed via a left anterolateral resuscitative thoracotomy. Initial success in animal models[13] led to technical descriptions in humans,[11] a specific national training curriculum,[14]

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and increased clinical reports.[15,16] Specific catheters for use in the United States are approved by the Food and Drug Administration (FDA). Despite enthusiasm, only recently have larger patient cohorts been described, and there is some debate in the surgical literature as to the optimal utilization of REBOA in patients with exsanguinating torso hemorrhage arising from below the diaphragm.[17] This emphasizes the need for further prospective evaluation.

REBOA TECHNIQUE

Description of the REBOA technique includes the following accepted steps.[18]

Arterial Access and Sheath Insertion

Arterial access is the critical initial step in the deployment of REBOA. We advocate early ultrasound-guided placement of an arterial line into the common femoral artery (CFA) rather than radial artery if REBOA is a possibility. At our institution, a systolic blood pressure (SBP) <90 mm Hg in a partial or nonresponder to resuscitation is a trigger for femoral access. This can be used as a conduit for the larger sheath required for REBOA deployment if chest x-ray eliminates an obvious aortic injury.

Sheath placement in the CFA can be via percutaneous, open cut down, or the described guidewire exchange over an existing femoral arterial line, with ultrasound guidance. There are a variety of sheath diameters and lengths, and available and advancing technology of manufacturers is leading to decreased sheath size from the initial iterations of 12 to 13 French (Fr).[19] We utilize a 7-Fr sheath, of which multiple manufacturers are compatible with currently used catheters, including Medtronic (Minneapolis, MN), Input Introducer Sheath, Cordis (Milpitas, CA), Avanti+ Sheath Introducer, Terumo (Somerset, NJ), Pinnacle R/O II Radiopaque Marker Introducer Sheaths and Arrow (Morrisville, NC), and Super Arrow—Flex Sheath Introducer (Teleflex, Morrisville, NC).

Balloon Selection and Position

There are several FDA-approved endovascular AO balloons currently available in the United States. Some systems such as the Cook CODA balloon (Cook Medical, Bloomington, IN) were initially developed for use in the setting of ruptured abdominal aortic aneurysm (AAA) management and are deployed via a 12 Fr sheath. We currently use the FDA-approved Prytime ER-REBOA (Prytime Medical Devices, Inc, Boerne, TX) system, which is also approved for use in Europe (Figure 1).

With REBOA, to decide on the optimal site of balloon inflation, the aorta is divided into 3 anatomical zones (Figures 2 and 3):

> Zone I: Descending thoracic aorta (between origin of the left subclavian and celiac arteries). Balloon inflation at zone I would physiologically resemble application of an aortic cross-clamp during a resuscitative left anterolateral thoracotomy.
> Zone II: Paravisceral aorta (origin of the celiac artery to the most distal renal artery). This is considered a less viable occlusion zone due to the presence of the celiac, superior mesenteric and renal arteries in this zone.
> Zone III: Infrarenal abdominal aorta (between the lowest renal artery and aortic bifurcation). Inflation here is optimal for patients with hemorrhage arising from severe pelvic fractures or junctional hemorrhage not amenable to application of either a junctional or lower extremity tourniquet.

After the optimal zone of occlusion has been determined based on the most likely source of hemorrhage, the catheter is inserted with radiographic, ultrasound, or clinical confirmation of position in the appropriate zone before balloon inflation. Fluoroscopy is not used in our current practice.

Balloon Inflation and Stabilization

After confirmation of appropriate catheter and balloon position, the balloon is inflated to approximate against the wall of the aorta. Balloon inflation volume varies with catheter and position of occlusion. With balloon inflation, there should be a concomitant rise in SBP. Adjuncts to appropriate balloon inflation include loss of pulse or Doppler signal in the contralateral CFA. Although not yet described, transesophageal echocardiography could assist with balloon placement in zone I, as described for intraaortic balloon pump placement.[20] Stabilization of the catheter after balloon inflation is critical, especially with wireless catheters that have a tendency to migrate with aortic pulsation if not secured. Documentation of the time of balloon inflation should be performed and communicated with the resuscitation team.

Balloon Deflation

Once hemorrhage control and initial volume resuscitation have been achieved, balloon deflation should occur with effective team communication, given anticipated changes in afterload, perfusion pressure, and physiologic milieu. At our institution, balloon deflation is slow by removing a few milliliters of saline sequentially and monitoring hemodynamic changes. Several cycles of partial deflation and reinflation may be required before complete deflation. Once the balloon has been deflated and stability has been achieved, the catheter can be removed. In devices with arterial pressure monitoring, removal and loss of monitoring necessitates placement of an alternate, usually upper-limb, arterial catheter.

Sheath Removal

The process for sheath removal is dependent on the size of the sheath utilized. Larger 12- Fr sheaths require a femoral artery cut down with direct repair of the arteriotomy. Smaller 7-Fr sheaths may be removed without surgical repair, but manual compression on the insertion site should be applied for a minimum of 30 minutes. It is imperative that confirmation of distal arterial perfusion be verified immediately post-removal and thereafter by clinical and/or ultrasonographic means. Larger 12-Fr sheaths are typically occlusive, especially in younger patients with smaller diameter femoral/iliac vessels. Therefore, sheath removal should occur expeditiously to limit the risk of distal ischemia and potential limb loss.

INITIAL CLINICAL EXPERIENCE

A recent report of multicenter national experience from the American Association for the Surgery of Trauma

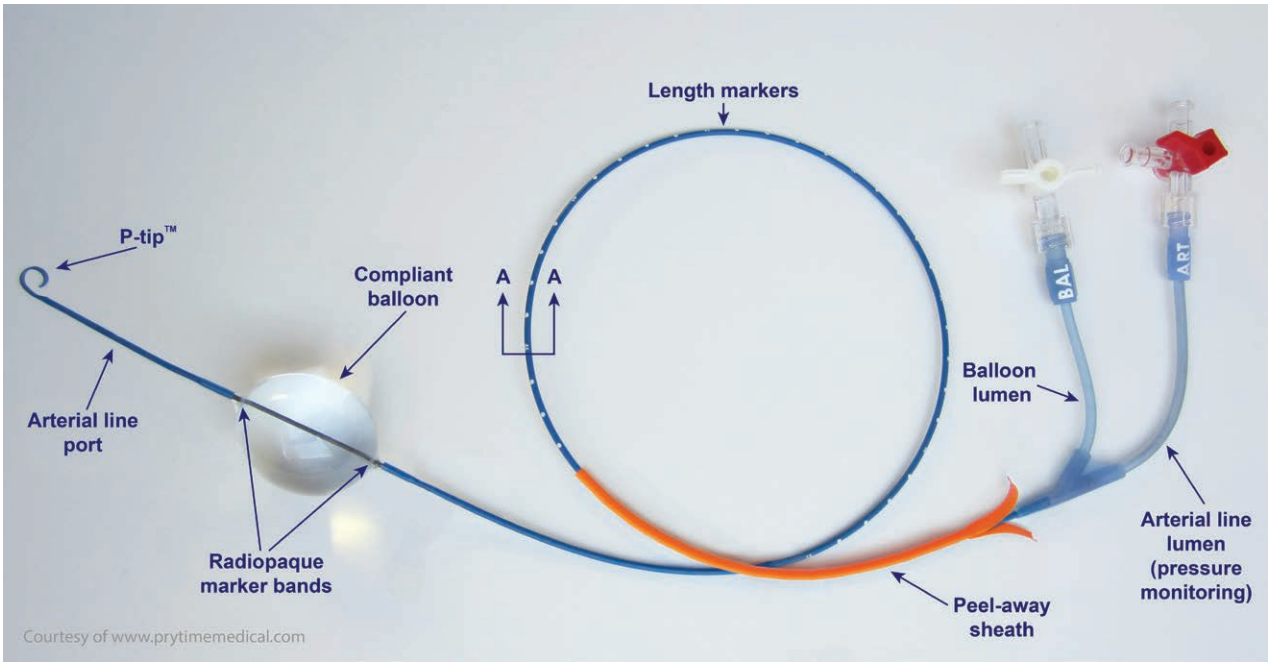

Figure 1. Catheter and balloon example (PryTime Medical ER-REBOA Catheter). REBOA indicates resuscitative endovascular balloon occlusion of the aorta.

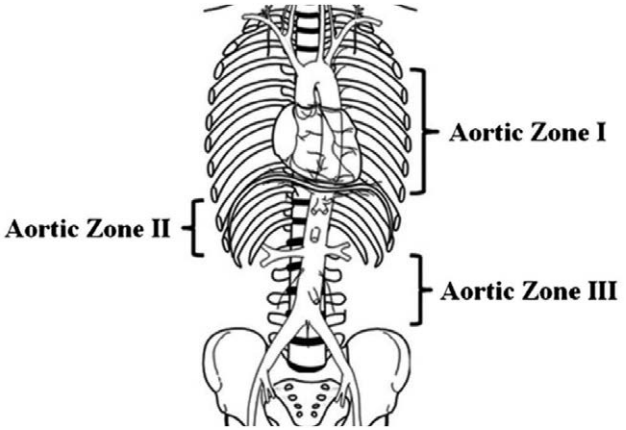

Figure 2. REBOA zones (Maryland.CCproject.com). Zone I: From origin of the left subclavian artery to the celiac artery (infradiaphragmatic). Zone II: Abdominal aorta from the celiac to lowest renal artery. Zone III: The infrarenal abdominal aorta between the lowest renal artery and the aortic bifurcation. REBOA indicates resuscitative endovascular balloon occlusion of the aorta.

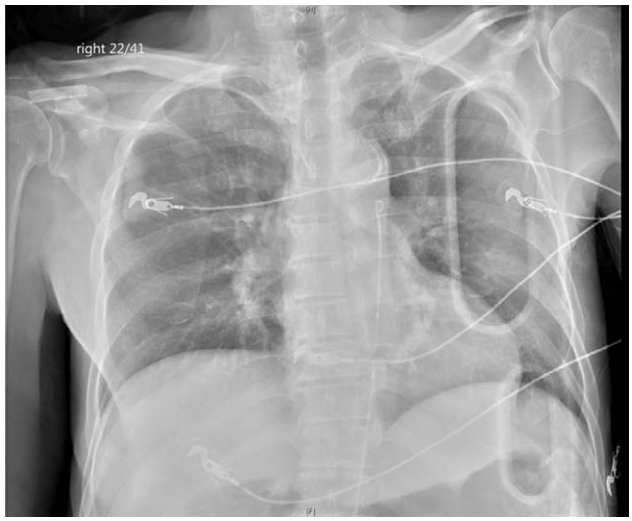

Figure 3. Chest x-ray showing REBOA catheter tip location in zone I distal to the aortic arch. REBOA indicates resuscitative endovascular balloon occlusion of the aorta.

registry[12] supplements earlier outcome data from a systematic review.[16] However, in that review, only 15 of the included 41 studies were for trauma victims, which limits its application to the current narrative. Additional data remain retrospective at this time.[19,21] An overwhelming majority of the trauma registry data are from 2 centers, including ours. We emphasize that initial enthusiasm from these registry and observational reports must be evaluated in the absence of prospective randomized controlled trials, which are required to confirm and validate early outcomes. A recent single-center retrospective data collection is also a lower level of evidence.[22] Details of anesthesia technique, management, and implications are limited in these reports to date.

Deployment

Registry data through February 2015 from 8 participating centers confirmed earlier reported trends.[12] In the report, 46 patients underwent REBOA compared to 68 patients with open AO. AO occurred in the emergency department more commonly than the OR (73.7% vs 26.3%). In a single-center retrospective study, through September 2015, 31 patients underwent REBOA with significant mean injury severity (ISS -34) and 10 of the cohort (32%) undergoing cardiopulmonary resuscitation (CPR) at the time of placement.[22] In this report, REBOA access was primarily via femoral cut down (50%), followed by percutaneous without imaging (28.3%) and ultrasound guided (10.9%).[12] As anticipated, balloon deployment

was mostly in zone I (78.6% registry, 45% single-center), followed by zones III (19% registry, 55% single center) and II (2.4%).[12,22] A second AO attempt was required in 2 REBOA subjects (4.3%), less frequent than with open AO (13.2%). Of note, there was no time difference to successful AO (REBOA, 6.6 ± 5.6 minutes, $P = .8$ compared to open). Average deployment time has been 18 to 26 minutes.[11,21]

Efficacy

Hemodynamic improvement after AO was observed in 67.4% of REBOA patients (no difference to open), with more achieving stability (SBP consistently >90 mm Hg for >5 minutes; 47.8%) compared to open (27.9%, $P < .05$).[12] Others have reported median increases of 55 to 62 mm Hg,[11,21] with recent suggested trends in the increase from zone I (median 55 mm Hg) to zone III (45 mm Hg).[22] Of note, in the 10 patients undergoing CPR, 6 had return of spontaneous circulation.[22] As emphasized, trends to improved overall survival with REBOA (28.2%) in the registry is not level-1 evidence and did not reach statistical significance ($P = .12$). In the 2 highest-enrolling centers in the registry, fewer early deaths and improved overall survival were significant compared with open resuscitative thoracotomy and aortic clamping (37.5% vs 9.7%, $P = .003$).[21] A significant portion of the mortality appeared to be related to nonsurvivable head injuries, which are confirmed beyond 24 hours of hospitalization and not in the immediate postinjury phase.[22] As anticipated, worst survival benefit was in the cohort with CPR in progress and a zone-I occlusion.[22]

Safety and Complications

Complications of REBOA in these initial series were uncommon given the severity of injury (pseudoaneurysm 2.1%, embolism 4.3%), with no detected limb ischemia. Of concern, an external iliac artery injury and lower limb ischemia, necessitating amputation, has been reported in a different series (12.5%).[23]

Case reports exist for possibly implicated complications, and in 1 patient, increased blood pressure with REBOA is purported to have exacerbated a preexisting cerebral injury (contusion and subarachnoid hemorrhage), leading to a fatal outcome.[24] The current dilemma of optimal cerebral perfusion pressure (CPP) in a polytrauma patient with a cerebral injury and who is a candidate for REBOA will only be confirmed in a prospective investigation.

IMPLICATIONS FOR ANESTHESIOLOGISTS

Given the relative novelty of the resuscitative potential of REBOA, it is imperative that anesthesiologists, especially those dealing with the acutely injured trauma victim, be familiar with use of the device and the physiologic trespass related to balloon inflation and, more importantly, deflation. This would complement recent suggestions for the "acute care" anesthesiologist, which includes trauma care.[25] There is currently an absence of published perioperative or anesthetic data, so recommendations are made based on experience, physiologic principles, and a similar mechanism in aortic vascular procedures. Our goal is to provide a practical approach to anesthetic management of these critically ill patients and identify areas in which further study is necessary to facilitate formal guidelines. We address considerations for patient preparation, REBOA deployment, hemodynamic and resuscitative goals, potential complications related to AO and mitigation/prevention, and issues related to deflation of the balloon and subsequent management.

Predeployment Phase

From the anesthesiologist's perspective, a large proportion of patients undergoing AO will do so before arrival to the OR. However, given the trend toward lower thresholds for REBOA deployment,[12] patients requiring exploration for hemorrhage but not yet in extremis may present with planned balloon deployment. This affords time to optimize preparation as far as possible before the procedure. A comparable model for REBOA is AO for ruptured AAA, where endovascular AO is becoming more common. Many recommend prophylactic balloon placement in that cohort before any maneuvers that may change hemodynamic status, such as induction of anesthesia or initiation of surgery.[26,27] This is supported by animal data models[28] and with use of REBOA prophylactically during resection of pelvic tumors.[16] The benefit of early deployment to prevent hemodynamic compromise is balanced with risks of duration of AO, which can be addressed by protocol-guided decision making.[27]

Vascular access should be obtained as soon as possible, ideally before anesthesia induction, so that resuscitation can be continuous. As discussed, we recommend femoral arterial access with an 18- or 20-gauge catheter. Theoretically, REBOA may be deployed from upper extremity arterial access, but brachial access may be associated with more complications and is not recommended.[27] Venous access should be with large bore central access for transfusion and use of necessary resuscitative medications.

For expected zone-I REBOA placement, transesophageal echocradiography, which may already be in use to guide resuscitation and assessment of cardiac function, may aid in balloon placement.[20] In addition, it may impact planned REBOA in a patient with undiagnosed severe atherosclerosis, traumatic aortic dissection, or aortic aneurysm. Transesophageal echocradiography can also be used to monitor position of the balloon, as the use of smaller sheaths for placement may have increased propensity for balloon migration with vigorous cardiac activity.[27]

Hemodynamic and Resuscitative Concerns During AO

Resuscitation of patients with NCTH after REBOA placement should mimic management without a balloon in place, but there are several additional considerations. Most trauma centers use massive transfusion protocols to guide therapy in patients with massive hemorrhage, and this is appropriate in the REBOA population as well. Inflation of the REBOA balloon often results in immediate and dramatic increase of blood pressure,[12,16] which while helpful, may be hazardous in a critically ill patient. A sudden increase in afterload produced by AO increases left ventricular work and may lead to left ventricular dilation, increased pulmonary artery pressure, and myocardial ischemia,[28,29] especially in patients with preexisting heart disease. Temporary central hypertension may lead to cerebral hyperperfusion, which may be problematic in the setting of intracranial trauma or bleeding.[24,30,31] While there is decreased or absent arterial inflow during a truncal hemorrhage, central

hypertension and overaggressive volume administration may exacerbate venous hypertension with potentially worsened bleeding from abdominal viscera. With these concerns, it may be prudent to continue permissive hypotension as recommended in trauma patients, with a goal SBP ≤100 mm Hg[29] and close vigilance on volume resuscitation. Although these concerns will be especially evident with zone-I deployment, they may also occur with distal AO. It is reassuring that endovascular occlusion data from the cardiac surgical literature does not appear to increase risk compared to clamp occlusion.[32]

Duration of AO is critical, given continued ischemia distal to the site and impending reperfusion. Although there are no current recommendations for maximum duration of REBOA, registry data showed AO between approximating 25 minutes. The anesthesiologist must use this time of relative hemodynamic stability to prepare for reperfusion.

Mitigation of Complications

Renal. Renal perfusion is decreased regardless of the location of AO.[33] Concomitant with reduced renal blood is increased renal vascular resistance. The Aortic Occlusion for Resuscitation in Trauma and Acute Care Surgery (AORTA) registry showed a low rate of acute kidney injury, resulting in need for renal replacement therapy.[12] However, the rate of acute kidney injury that does not require dialysis and potentially complicates recovery may be significant, as patients receive dual insults of ischemia and reperfusion.[34] Renal protection is accomplished by limiting AO time, as postoperative renal failure in AAA repair is minimized with <20 minutes of ischemic time and increases 10-fold with AO times >50 minutes.[33,35] Although theoretically attractive, results of pharmacologic therapy to limit renal injury with reperfusion remains disappointing in the cardiovascular literature. Data are inconclusive for mannitol (scavenging reactive oxygen species [ROS], inhibiting neutrophil-induced tissue damage and reduced inflammatory mediators), dopamine, and dopamine receptor agonists such as fenoldopam.[29,34] The best recommendation for the anesthesiologist is to optimize hemodynamic status and preserve circulating blood volume for favorable renal perfusion.

Pulmonary. The cardiac literature suggests that there is an increase in pulmonary vascular resistance created by AO, and this may be significant in a trauma patient with unknown cardiopulmonary disease.[29] There is also potential for pulmonary edema from increased capillary permeability created by inflammatory mediators associated with ischemia reperfusion and increased hydrostatic pressure after AO. In the population with acute trauma injury, there are several other contributing factors to pulmonary injury, acute lung injury, and respiratory failure. Of note, the AORTA registry showed a nonstatistically significant reduction in pulmonary complications with use of REBOA over resuscitative thoracotomy.[12]

Intestinal and Visceral. Distal AO has the potential for hyperemia and congestion of the splanchnic circulation, and proximal occlusion leads to ischemia and reperfusion after restoration of blood flow. Transmural intestinal ischemia, which increases mortality significantly, is primarily a concern with prolonged ischemic times.[29,34] In AAA surgery, colon ischemia results in almost 2% mortality and up to 9% mortality in ruptured AAA patients.[33] Other than obvious ischemic sequelae, bacterial translocation may be a concern with bacteremia and potential sepsis, especially in a compromised patient.[33] Minimizing AO time is critical for the visceral and intestinal contents.[29]

Neurologic. Patients with NCTH who require REBOA may frequently have other significant injuries that cannot be fully evaluated before arrival in the OR, especially traumatic brain injury (TBI). During AO, cerebral blood flow will increase and may be detrimental in the setting of unrecognized intracranial bleeding. Current guidelines for management of patients with TBI are for a CPP goal of 60 mm Hg, with >70 mm Hg not benefiting recovery.[31] With REBOA in place, CPP will often be unknown, but hemodynamic goals should prevent excessive perfusion when TBI is suspected.

Occlusion of the proximal aorta impedes radicular artery flow to the spinal cord (SC), with potential detriment to anterior SC perfusion.[29] In patients undergoing aortic surgery, occlusion times of up to 30 minutes are unlikely to create permanent deficit.[36,37] The AORTA registry had no patients in either arm with SC deficit,[12] so the likelihood of injury appears small unless REBOA deployment time is prolonged. Current strategies in vascular surgery for mitigating SC ischemia related to AO involve increasing SC perfusion pressure (similar to CPP) by removing cerebrospinal fluid or increasing blood pressure when spinal hypoperfusion is likely. With the emergent trauma patient with unknown injuries, placement of a cerebrospinal fluid drain is unlikely, and CPP will be cautiously managed as described above. One target for anesthesiologists will be control of hyperglycemia, which has the potential to ameliorate cerebral and SC ischemic injury.[29]

Temperature. Hypothermia is one of the lethal triad in trauma patients and a significant factor in mortality. Literature supports grading active therapies based on the level of hypothermia,[38] with even more aggressive correction indicated in REBOA patients with impending balloon deflation. Passive and active measures include raising the ambient temperature in the OR, infusion of warm fluids, convective or radiant warming, minimized evaporative cooling,[38] and prehospital warming if and when possible.[39]

REBOA Deflation and Management of Reperfusion

Deflation of the balloon is a critical time in care of these severely injured patients. Keys to limiting complications are balloon inflation for the shortest possible time and excellent communication and anticipation from all members of the operative and anesthesia teams. Anticipated derangements include the following[28]:

- Sudden decrease in circulating volume;
- Increasing metabolic acidosis from reperfusion of distal tissue beds;
- Release of inflammatory mediators, complement, and ROS secondary to anaerobic metabolism;
- Possible release of embolic material into the circulation;
- Myocardial rhythm and contractility changes from electrolyte, temperature, and acidosis; and

- Hemorrhage from restoration of blood flow to an injured area where surgical hemostasis still has to be achieved.

These potential problems require active management before balloon deflation, although complete mitigation may not be possible in a severely injured patient. Caution is required for a period after deflation, as in aortic surgery, metabolic derangement and presence of inflammatory markers and ROS may continue for up to 30 minutes after reperfusion.[33] These manifestations may occur after as brief an occlusion of 15 minutes.[34]

The concept of ischemic postconditioning with gradual deflation and partial reinflation requires prospective study in REBOA but may be of theoretical benefit for several organ systems, including the kidney.[33,40] Preparation for reperfusion should include pharmacologic and electrical means for managing cardiac arrest and associated arrhythmias, with active participation of the OR team.[27]

CONCLUSIONS

REBOA is a recent addition to the available strategies in a trauma victim with life-threatening truncal hemorrhage. Although prospective level-1 evidence is still awaited, early retrospective and registry data support the physiologic principles and appear promising. Given increased utilization, it is incumbent on anesthesiologists to be familiar with the technique, the logistics of balloon inflation, and the inevitable pathophysiologic changes associated with balloon deflation, similar to traditional aortic vascular maneuvers. Although there are very limited data to guide perioperative management, the intent of this narrative is to facilitate anesthesiologist knowledge of the technique. Suggestions and recommendations for intraoperative management are based on expert recommendations from one of the highest users in the US registry, physiologic principles, and more evidence-based strategies from aortic vascular procedures.

DISCLOSURES

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Physiologic Considerations in Trauma Patients Undergoing Resuscitative Endovascular Balloon Occlusion of the Aorta

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Resuscitative endovascular balloon occlusion of the aorta is a new procedure for adjunctive management of critically injured patients with noncompressible torso or pelvic hemorrhage who are in refractory hemorrhagic shock, ie, bleeding to death. The anesthesiologist plays a critical role in management of these patients, from initial evaluation in the trauma bay to definitive care in the operating room and the critical care unit. A comprehensive understanding of the effects of resuscitative endovascular balloon occlusion of the aorta is essential to making it an effective component of hemostatic resuscitation. (Anesth Analg 2017;125:891–4)

Resuscitative endovascular balloon occlusion of the aorta (REBOA) is a novel catheter-based approach to aortic interruption based on knowledge gained from the vascular surgical literature as well as translational research from animal studies.[1,2] The technique is intended for use in trauma patients at high risk for death from exsanguinating hemorrhage due to noncompressible torso hemorrhage, in place of traditional open thoracotomy and cross-clamping of the descending thoracic aorta. A balloon catheter is positioned in the aorta via a sheath inserted in the common femoral artery and inflated to achieve partial or complete aortic obstruction. REBOA represents a means of temporary control of bleeding and provides a bridge to definitive surgical care in the operating room or angiography suite.[1–3]

The anesthesiologist, as a part of the trauma team, is intimately involved in the care of the critically injured patient from the trauma bay to the operating room and onto the critical care unit. They need to understand the purpose and function of REBOA, its physiologic implications, and how to incorporate it into a comprehensive protocol for hemostatic resuscitation. In this report, we aim to provide an understanding of critical physiologic and team issues unique to the patient who has had a REBOA catheter placed.

ADMISSION AND INITIAL PATIENT MANAGEMENT

REBOA is most commonly performed in the trauma bay, in a severely injured patient with progressive hemorrhagic shock due to a noncompressible injury below the diaphragm. Prehospital use of REBOA has been advocated in Emergency Medical Systems with physicians present at the scene of injury.[4]

The critically injured patient requiring REBOA needs early airway management to facilitate definitive care. Before proceeding with rapid sequence induction (RSI), large bore IV access must be established if possible, and fluid resuscitation commenced in accordance with the principles of hemostatic resuscitation.[5] REBOA before intubation may optimize hemodynamics to better facilitate tolerance of RSI, though this concept has not been prospectively studied. In a given trauma admission, the specifics of individual presentation, available resources, and team coordination will likely dictate the sequencing of REBOA and RSI. Recommended dosing of induction drugs and techniques for intubation have been extensively covered in the literature.[6–8]

Positive-pressure ventilation may have a profound effect on preload in the volume-depleted patient.[9] This effect may be exaggerated in patients undergoing REBOA and should be accompanied by active infusion of blood products, including uncrossmatched "emergency-release" red blood cells and plasma in accordance with the institutional massive transfusion protocol. As REBOA immediately reduces the size of the effective circulation, balloon occlusion is an alternative to early blood product administration if blood is not available; there is evidence from controlled experiments in swine that equivalent survival can be achieved.[10]

THE PHYSIOLOGY OF REBOA

The REBOA catheter may be inflated in different anatomic zones of the descending aorta depending on the presumed location of injury. Zone 1 is in the descending thoracic aorta, above the celiac trunk; zone 2 is from the celiac trunk to the lowest renal artery; and zone 3 extends from the lowest renal artery to the aortic bifurcation. For abdominal exsanguination, the balloon is placed in zone 1. For primarily pelvic bleeding, a zone 3 occlusion is indicated. The specifics of the procedure have been well described elsewhere.[2]

Balloon occlusion in the setting of continuing fluid resuscitation leads in our experience to a marked and almost immediate improvement in hemodynamics and a profound rise in left ventricular (LV) afterload.[11] The response will be more dramatic with more proximal occlusion. Although it seems likely that more distal occlusion will be better tolerated, early studies in a swine model did not demonstrate

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a difference in mortality or neurologic injury (paraplegia), based on zone 1 versus zone 3 occlusion.[12]

Zone 1 balloon occlusion produces acute changes in cardiovascular physiology and organ perfusion, as predicted by the existing body of literature related to aortic cross-clamping for vascular surgery. The physiologic response to zone 1 balloon occlusion, in a patient with a previously normal ventricle, is an increase in LV afterload, wall tension, and mean central aortic pressure, with associated increase in subendocardial oxygen demand. In a swine model of hemorrhagic shock, these changes were variable over time, based on the duration of balloon occlusion.[13] In elective vascular surgery patients, Roizen et al[14] described greater changes in physiology with more proximal aortic occlusion: occluding the descending thoracic aorta increased mean arterial pressure (MAP) by 35% to 84%, pulmonary capillary wedge pressure by 90% to 190%, and central venous pressure by 35%. Conversely, supraceliac occlusion increased end-diastolic and end-systolic areas pressures by 28% and 69%, respectively, with decreases in ejection fraction and cardiac index of 38% and 29%, respectively. Wall motion abnormalities were detected in 92% of patients after occlusion.[14]

Zone 3 occlusion is not associated with the same profound cardiovascular changes as seen in zone 1 occlusion. In vascular surgery patients, infrarenal aortic cross-clamping leads to a mild increase in MAP (2%–8%), no change in pulmonary capillary wedge pressure or central venous pressure, and increases in end-diastolic and end-systolic area of 9% and 11%, respectively.[14] There is limited evidence of wall motion abnormalities in patients undergoing infrarenal occlusion, in contrast to those with more proximal occlusion.[14,15]

Supplemental Digital Content 1, Table, http://links.lww.com/AA/B804, summarizes key physiologic changes at different levels of aortic occlusion.

Aortic cross-clamping induces damage to the vascular endothelium.[15,16] Compared to aortic cross-clamping, REBOA may be a favorable alternative.[17] In particular, the concept of partially occluding the aorta is being explored. Russo et al demonstrated that complete resuscitative balloon occlusion of the aorta (C-REBOA) compared to partial resuscitative balloon occlusion of the aorta (P-REBOA) was accompanied by supraphysiologic proximal pressures in the C-REBOA group.[18] Supraphysiologic pressure was defined as a MAP >110 mm Hg for the duration of balloon occlusion. Pressure gradients were maintained consistently between proximal and distal systolic pressures in each group. Pressure gradients averaged 90% in the C-REBOA group, 50% in the P-REBOA group, and 10% in the control group (no intervention). Lactate concentrations rose more rapidly in the C-REBOA group, and histologic changes in the duodenum (ischemic necrosis) and renal parenchyma (acute tubular necrosis) were observed.[18]

Supplemental Digital Content 2, Table, http://links.lww.com/AA/B805, summarizes the animal data of physiologic parameters with C-REBOA and P-REBOA.

In a different animal model of hemorrhagic shock, the maximum tolerated duration for zone 1 balloon occlusion was about 60 minutes.[19] Both organ ischemia and mortality increased as the occlusion time approached 90 minutes. Maintaining proximal MAP near the normal physiologic target with P-REBOA may decrease the incidence of cerebral edema, respiratory failure, and cardiac dysfunction when compared to sustained aortic occlusion, but this hypothesis has not yet been prospectively studied.

Hemodynamic instability after balloon deflation is reduced in patients undergoing P-REBOA versus C-REBOA,[20,21] with less duodenal ischemia in the P-REBOA group despite equivalent visceral MAP. This effect may be attributed to a phenomenon similar to ischemic preconditioning.[22]

As with traditional aortic cross-clamping for vascular surgery, increase in MAP with aortic occlusion does not imply correction of hypovolemia or resolution of distal bleeding. Full resuscitative measures should continue until the surgeon has identified and controlled the source of hemorrhage. While case-controlled data are limited, it seems clinically prudent to deliberately increase preload during aortic occlusion, in anticipation of balloon release.

INTRAOPERATIVE MANAGEMENT AND MONITORING

Patients with torso hemorrhage who have undergone REBOA require immediate surgical or angiographic exploration, often concurrently. If available, hybrid operating rooms or RAPTOR (resuscitation with angiography, percutaneous techniques, and operative repair) suites allow the performance of all interventions, open and endovascular, in one physical location.[23]

In addition to standard resuscitative measures, we suggest the use of arterial lines and echocardiography will additionally assist the anesthesiologist in their approach to the patient who has REBOA.

Placement of an upper extremity arterial line as soon as it is feasible to do so is recommended. This will allow the anesthesiologist to anticipate and adjust for rapidly changing hemodynamic parameters. Newer REBOA catheters such as the ER-REBOA device (Prytime Medical, Denver, CO) include a proximal port for pressure monitoring during balloon occlusion, but removal of the REBOA catheter (and the femoral sheath) is recommended as soon as possible to avoid ischemic complications of the lower extremity.

Close assessment of cardiovascular status is recommended. Dynamic 2-dimensional transesophageal echocardiography (TEE) has numerous benefits if it is available, and the operator is proficient in its use. TEE allows for the rapid intraoperative diagnosis of wall motion abnormalities, measurement of ejection fraction, and can assist with volume management. Roizen et al[14] demonstrated that in patients undergoing aortic cross-clamping, myocardial stresses varied with the level of occlusion, and intraoperative TEE may reveal myocardial dysfunction not easily detectable by other conventional monitoring modalities. TEE can also be used to confirm positioning of the REBOA balloon in zone 1 when that is the desired location.

CONSIDERATIONS SURROUNDING BALLOON DEFLATION

REBOA balloon deflation is a tenuous time. The balloon may be deflated when all hemostasis is achieved or to check for ongoing hemorrhage. Deflation is akin to releasing an aortic

cross-clamp. A lactic acid load will accumulate in direct relation to the duration of occlusion. Along with the accompanying myocardial depression, release may result in decreases of LV afterload of 70% to 80% with resultant drops in MAP.[14,19] Coronary blood flow and LV end-diastolic volume also decrease by as much as 50%. This combination of lactic acid load, change in cardiac function, and ongoing hemorrhagic shock can lead to profound hypotension at the time of balloon deflation. The anesthesiologist should be prepared to administer further blood products and calcium as needed.

Clear team communications are required. Balloon deflation should be gradual, allowing the surgeon to check for potential continued hemorrhage while the anesthesiologist monitors perfusion. If rapid deterioration occurs, the balloon can be reinflated to allow additional time for reassessment and planning. Once all hemorrhage is controlled, and the patient is hemodynamically stable with the balloon deflated, resuscitation should be completed in accordance with normal practice. Monitoring of serum lactate is recommended as a guide to successful reversal of shock.[24,25]

SPECIAL CONSIDERATIONS FOR VULNERABLE POPULATIONS

REBOA may produce exaggerated physiologic effects or unintended consequences in specifically vulnerable populations. Elderly patients will be less tolerant than younger ones to abrupt changes in preload and cardiac stress.[26]

Trauma patients may have concurrent injuries to the brain and body and may be suffering from both hemorrhagic shock and traumatic brain injury, with a synergistic increase in prospective mortality. Abrupt changes in MAP and preload may adversely affect cerebral perfusion pressure. This may inadvertently lead to worsened secondary brain injury after the primary insult.[17,27] If there is suspicion of traumatic brain injury, the anesthesiologist, in conjunction with the trauma or neurosurgical team, may wish to consider early invasive intracranial pressure monitoring to allow more informed management of systemic and cerebral pressures during and after REBOA. A concurrent neurosurgical procedure may be required while the trauma team is addressing the source of hemorrhage.

ADJUNCTIVE PROCEDURES

REBOA may create the need for additional procedures. The large sheath in the common femoral artery will require removal by a surgeon skilled in vascular techniques and may include a perfusion study of the lower extremity. This procedure will add to the overall operative time. Newer REBOA catheters are being designed for placement through smaller sheaths.[28] Their use may mitigate these concerns.

TEAM CONSIDERATIONS

A key component to the management of any critically ill patient is efficient team dynamics, and this applies equally to patients undergoing REBOA. Key concepts in effective trauma team performance include situational awareness, closed-loop communication, mutual performance monitoring, and avoiding task overload.[29,30]

The management of the REBOA patient should be dynamic. The entire trauma team must understand the mechanism of injury and its implications. In the US trauma center, the trauma surgeon will often assume the role of team leader. If the surgeon is also the proceduralist for REBOA, they risk becoming task focused and losing situational awareness.[31] Depending on the individual hospital's response system, either an anesthesiologist or an emergency physician, and sometimes both, will form the nonsurgeon physician component of the resuscitation team. This individual should assume and direct resuscitative efforts for the period of time that the surgeon is performing the procedure.[32] The anesthesiologist can further continue this direction as the patient is transported to the operating room and prepared for surgery. In the intraoperative period, the anesthesiologist should remain in close communication and coordination with the surgeon and continue to direct the medical resuscitative therapy of the patient.

CONCLUSIONS

REBOA is a potentially life-saving technique in previously unsalvageable trauma patients; however, effective application requires a thorough understanding of the technology, the physiology of aortic occlusion, and the role of REBOA in hemostatic resuscitation.

DISCLOSURES

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Resuscitative Endovascular Balloon Occlusion of the Aorta and the Anesthesiologist: A Case Report and Literature Review

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The most common preventable cause of death after trauma is exsanguination due to uncontrolled hemorrhage. Traditionally, anterolateral emergency department thoracotomy is used for temporary control of noncompressible torso hemorrhage and to increase preload after trauma. Resuscitative endovascular balloon occlusion of the aorta is a minimally invasive technique that achieves similar goals. It is therefore imperative for the anesthesiologist to understand physiologic implications during resuscitative endovascular aortic occlusion and after balloon deflation. We report a case of a patient with significant pelvic and lower-extremity trauma who required acute resuscitative endovascular balloon occlusion of the aorta deployment, aggressive resuscitation, and extensive intraoperative hemorrhage control. (A&A Case Reports. 2017;9:154–7.)

To control noncompressible torso hemorrhage (NCTH), particularly below the diaphragm, anterolateral emergency department thoracotomy (EDT) traditionally is performed to facilitate aortic cross-clamping.[1] Ideally, this results in increased preload, temporary control of hemorrhage, and facilitates internal cardiac massage and defibrillation when needed. Endovascular aortic occlusion has been used to control hemorrhage in patients with ruptured abdominal aortic aneurysms.[2] The original case series using an intra-aortic balloon by the U.S. military described its use in the Korean War on 3 injured soldiers[3] and was slow to gain acceptance in trauma. With recent improvements, however, the use of resuscitative endovascular balloon occlusion of the aorta (REBOA) has become more common in the management of patients after traumatic hemorrhage.

REBOA is a supportive resuscitation technique, which, similar to EDT, should minimize distal hemorrhage while maintaining cerebral and myocardial perfusion.[4] After cannulation of the common femoral artery and insertion of a vascular sheath, a guidewire is advanced retrograde to the distal aortic arch. A balloon catheter is then advanced over the wire to a specified anatomic position (zone 1: origin of the left subclavian artery to the celiac artery or zone 3: lowest renal artery to the aortic bifurcation), which is determined based on the likely source of hemorrhage (Figure 1). The balloon is then inflated to provide aortic occlusion, confirmed by radiographic images, and secured in place with a clamp so as not to migrate distally. Although the surgical approach for placement of the REBOA has been outlined, the anesthetic perspective of prolonged aortic occlusion has not been described. This case study describes REBOA use with an occlusion time of approximately 110 minutes.

Written Consent Statement

The patient and family have given written permission for this published report.

DESCRIPTION OF THE CASE: CLINICAL CARE

A 40-year-old, 75-kg male unrestrained driver who was ejected from a motor vehicle presented to the trauma center with a Glasgow Coma Score (GCS) of 15. He had an obvious deformity of his left upper extremity, bilateral open femur fractures, and an unstable pelvis. He had no palpable pulse in any extremity but was able to move his left lower extremity and was awake, alert, oriented, and conversant. His heart rate varied from 112 to 137 beats per minute, with an oxygen saturation of 100% on nasal cannula. Initially, the patient continued to demonstrate an appropriate and reassuring mental status (the inability to obtain peripheral pulses was likely because of the extent of extremity injuries). An automatic blood pressure recording could not be obtained, and, therefore, manual sphygmomanometry was used with the first reading of 74/48 mm Hg approximately 15 minutes after presentation. The focused assessment with sonography for trauma was negative for pericardial, abdominal, or pelvic fluid.

Fluid resuscitation, including red cell and thawed plasma transfusion was initiated. An arterial line was placed in the left femoral artery, and a pelvic binder was applied. Admission lactate was 8.4 mmol/L and the base deficit was 18.3 mmol/L. Because of continued hypotension and concern for hemorrhage from severe pelvic injury, a REBOA device was deployed in zone 3 (infrarenal) through the left femoral arterial line site 23 minutes after presentation. The patient ultimately was intubated with etomidate and rocuronium 30 minutes after arrival as the result of declining mental status. His pelvic X-ray showed an extensively

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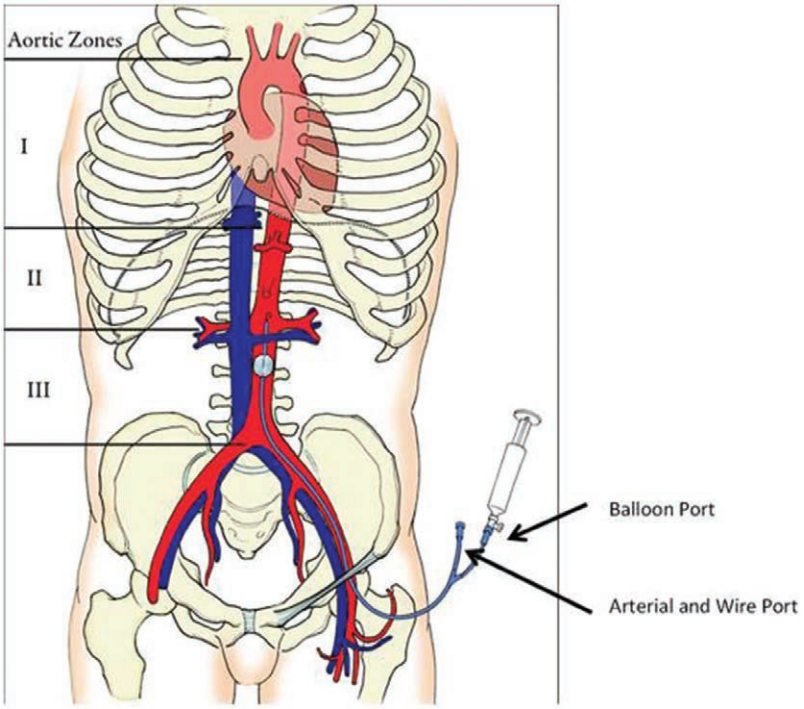

Figure 1. Anatomic landmarks of REBOA placement. REBOA indicates resuscitative endovascular balloon occlusion of the aorta.

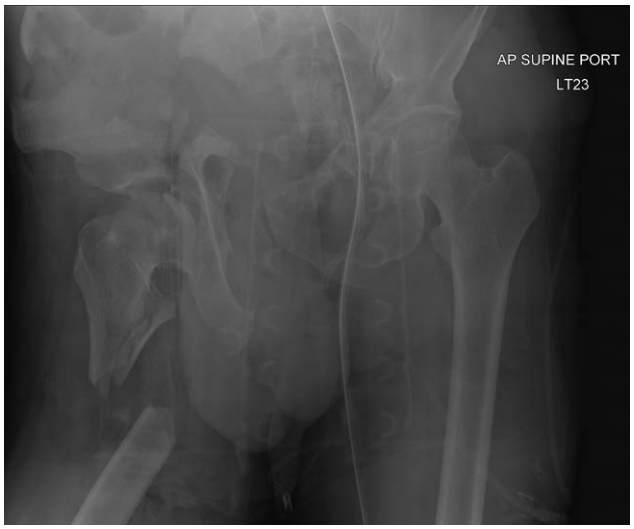

Figure 2. Anteroposterior radiograph of the pelvis demonstrating severe disruption of the pelvic ring, a right iliac wing fracture that extends into the right acetabulum, and a right subtrochanteric femur fracture. REBOA catheter is visualized over the left hemipelvis (arrow). REBOA indicates resuscitative endovascular balloon occlusion of the aorta.

displaced combined pelvic ring and acetabular fracture (Figure 2). The patient was transported emergently to the hybrid operating room (OR). Total balloon aortic occlusion time on presentation to the OR was 51 minutes.

Intraoperative Course

A right brachial endovascular angiogram demonstrated a "blush" of the right proximal internal iliac artery, which was coil embolized. The right external iliac artery was occluded, and an external iliac-to-common femoral artery bypass was performed. Spanning external fixation was applied to the bilateral lower extremities and pelvic stability was maintained by internal rotation of bilateral femurs and bridging the femoral frames to reduce pelvic volume. The REBOA was removed and the femoral artery repaired. Total REBOA inflation time, including preoperative deployment, was 110 minutes (Figure 3). Throughout the 9-hour operative course, the patient required ongoing hemodynamic resuscitation that included 32 units of packed red blood cells, 28 units of plasma, 24 units of platelets (4 "6 packs"), 180 mL of cryoprecipitate, 1 g of tranexamic acid, 1 L of 5% albumin, 4 L of Plasma-Lyte® (Baxter Healthcare Corp, Deerfield, IL), intermittent boluses of phenylephrine, vasopressin, calcium chloride, sodium bicarbonate, and infusions of norepinephrine and vasopressin. His analgesic requirement consisted of a total of 800 µg of fentanyl, which was administered in 50- to 100-µg aliquots throughout the 9-hour operation. He also received 4 mg of midazolam: 2 mg before the start of the operation and 2 mg before the final deflation of the REBOA. The inhalation anesthetic concentration of isoflurane ranged from 0.2% to 0.5% throughout the operation. The Table outlines the intraoperative acid–base status via arterial blood gas analysis. Estimated blood loss in the OR was 4.5 L, although this did not account for preoperative estimated blood loss, nor the volume of blood and clots on the OR table.

In subsequent days, the patient underwent multiple operations for debridement of the right lower extremity as he had a combination of direct muscle injury and ischemia resulting in muscle necrosis. However, he ultimately developed acute kidney failure requiring continuous renal-replacement therapy and, to obtain source control of necrotic tissue, required a right lower extremity amputation, followed by a right hemipelvectomy. Two months after injury, the patient was discharged to a rehabilitation facility with a GCS of 15. The patient was seen in trauma clinic 3 months later: he was with a GCS of 15 and cognitively intact.

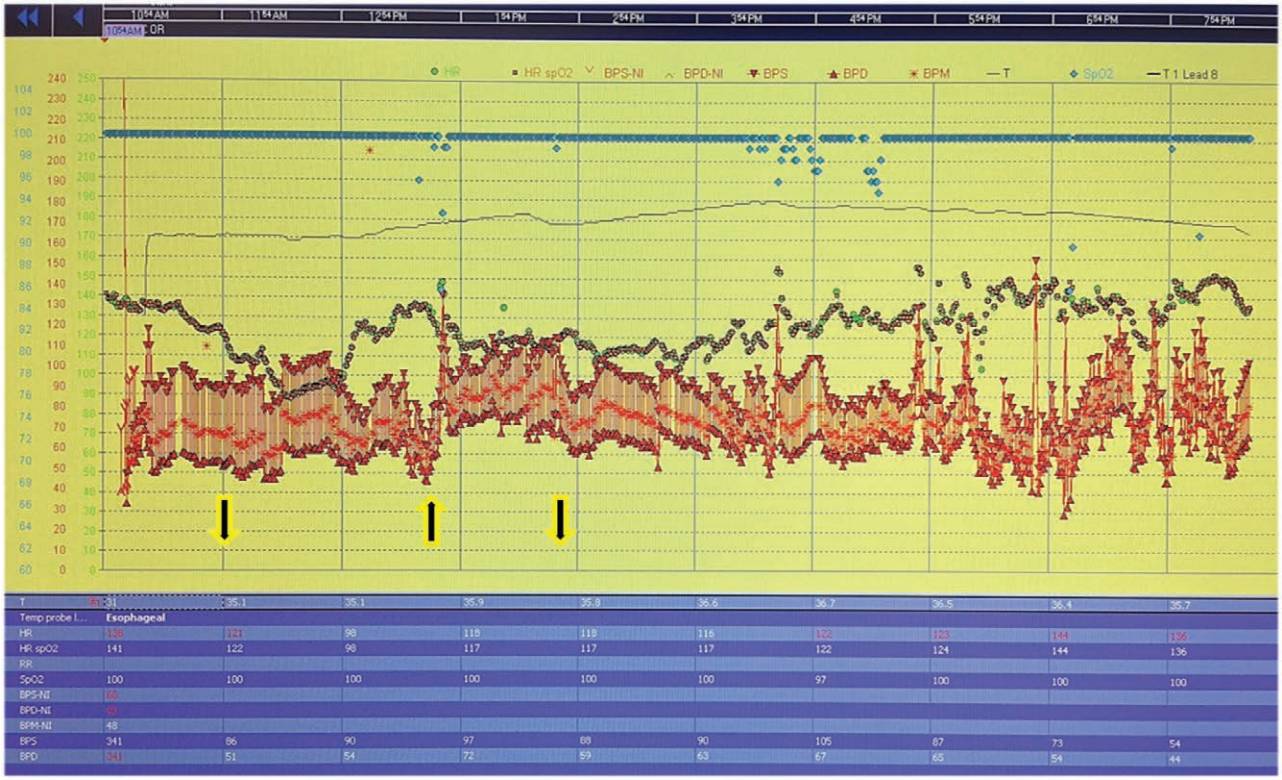

Figure 3. Time-compressed anesthetic electronic medical record (MetaVision) of intraoperative physiology. Inflated REBOA is in situ on arrival to OR; deflation and reinflation events are noted by arrows. REBOA indicates resuscitative endovascular balloon occlusion of the aorta.

Table. Arterial Blood Gas Values

	09:55	11:45	13:10	14:40	15:44	18:50
pH, Arterial	7.21	7.22	7.24	7.29	7.38	7.19
$Paco_2$ (mm Hg)	21	53	46	53	45	57
Pao_2 (mm Hg)	233	163	248	255	242	177
HCO_3^- (mmol/L)	8	21	19	24	26	21
Base excess (mmol/L)	−18.3	−6.2	−8.0	−1.8	1.6	−7.0

The initial values are immediately after insertion of arterial catheter in the resuscitation bay. Values beginning at 11:45 are from the intraoperative period. Final REBOA deflation occurred before the 15:44 time mark.

Abbreviations: HCO_3^-, biocarbonate; REBOA, resuscitative endovascular balloon occlusion of the aorta.

DISCUSSION

Uncontrolled hemorrhage represents the leading cause of preventable death after injury.[5] Despite recent advances in blood transfusion therapy,[5] NCTH remains a common cause of death due to hemorrhagic shock. NCTH results from injuries that cause vascular disruption of axial torso vessels, solid organ damage (ie, spleen, liver, kidney), and/or injuries to the bony pelvis. Although EDT has been the standard therapeutic approach to temporize NCTH,[1] it is morbid and the open chest is a source of heat and blood loss. Recent advances and availability of endovascular techniques, particularly the REBOA, have demonstrated promising results in the management of certain patients with NCTH after traumatic injury.[4]

The role of the anesthesiologist in the management of hemorrhagic shock is multifaceted, with attention paid to multiple organ systems. Deployment of the REBOA device and inflation of the occlusive balloon serve to augment coronary and carotid artery blood flow to maintain vital perfusion to the heart and brain, respectively. With proximal aortic occlusion, there is an increase in cardiac afterload and mean arterial pressure.[6] Physiologically, this increase in afterload, while supporting coronary perfusion, may also increase myocardial transmural wall tension and cardiac pressure work.[7] In a swine model of controlled hemorrhagic shock, Morrison et al[6] demonstrated that there was no significant difference in vasopressor support or cardiac filling pressures among animals with increasing duration of aortic balloon occlusion; however, the exact clinical significance on subsequent cardiac performance after balloon deflation has yet to be elucidated in humans. It is paramount for the resuscitation team to remember that REBOA is a temporizing measure with dynamic physiologic changes, which aims to support vital organ perfusion but does not provide definitive hemorrhage control.

At present, there are few recommendations for optimal hemodynamic management during the period of aortic occlusion. Considering the increase in systemic vascular resistance and mean arterial pressure during balloon inflation,[6,8] a balanced anesthetic technique that achieves

judicious analgesia and appropriate sedation seems wise.[9] Of note, our patient received a predominately narcotic-based anesthetic (approximately 10 mcg/kg of fentanyl) with an inhalational anesthetic of less than one-half of minimum alveolar concentration and supplemental midazolam. Aggressive hemodynamic resuscitation also must continue with transfusion of blood products and attention to the acute coagulopathy of trauma.[9] With prolonged duration of aortic occlusion, there is a significant increase in lactate concentration and volume of resuscitative fluid requirements compared with nonballoon occlusive therapy.[8] Similar to unclamping of the aorta during abdominal aortic aneurysm's repair, reperfusion distal to the occlusion site may result in a washout of metabolites with subsequent hyperkalemia, lactic acidosis, and profound hypotension.[7] Therefore, restoration of intravascular volume should be achieved before balloon deflation.

It is imperative that the anesthesiologist be in constant communication with the surgical operative team before and during the period of aortic balloon deflation to allow distal reperfusion without profound hemodynamic changes, and to rapidly reinitiate aortic occlusion should such decompensation occur. Similar to gradual release of an aortic cross-clamp, the REBOA balloon can be slowly deflated—and reinflated if necessary—while volume replacement or vasoactives are titrated to an optimal level.

Our patient sustained devastating anatomic injury. This injury pattern is associated with a high rate of morbidity and mortality related to hemorrhagic shock. Recent algorithms have been published to guide the decision-making process in patients with hemodynamically significant pelvic fractures, and the roles of angiography and external fixation.[10] The addition of REBOA for temporary hemodynamic support has also been described recently in patients with hemorrhagic shock secondary to pelvic injuries and is now considered part of the armamentarium for the resuscitation team until definitive hemorrhage control is achieved.[10] Considering the potential role of endovascular therapy in severe pelvic trauma, it would benefit the anesthesiologist to be well versed in the concepts of damage control resuscitation, damage control orthopedics, and endovascular aortic balloon occlusion. The concept of damage control anesthesia is also evolving to guide the anesthetic requirements in the setting of severe hemorrhagic shock.[9]

CONCLUSIONS

Profound life-threatening hemorrhage due to NCTH remains a leading cause of death after certain injuries. Endovascular therapy is an emerging technique that is applicable to trauma patients with significant vascular injuries. Considering the role of the anesthesiologist in the preoperative and intraoperative resuscitation period, a thorough understanding of hemorrhage control and temporary supportive therapies, such as REBOA, is necessary to be fully engaged in the dynamic resuscitation process. Future research with REBOA techniques will further define the role of this technology in the multidisciplinary management of severe hemorrhagic shock. ■

DISCLOSURES

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