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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Accepted for publication March 24, 2017.

Funding: Departmental.

The authors declare no conflicts of interest.

Reprints will not be available from the authors.

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DOI: 10.1213/ANE.0000000000002150

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 postremoval 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

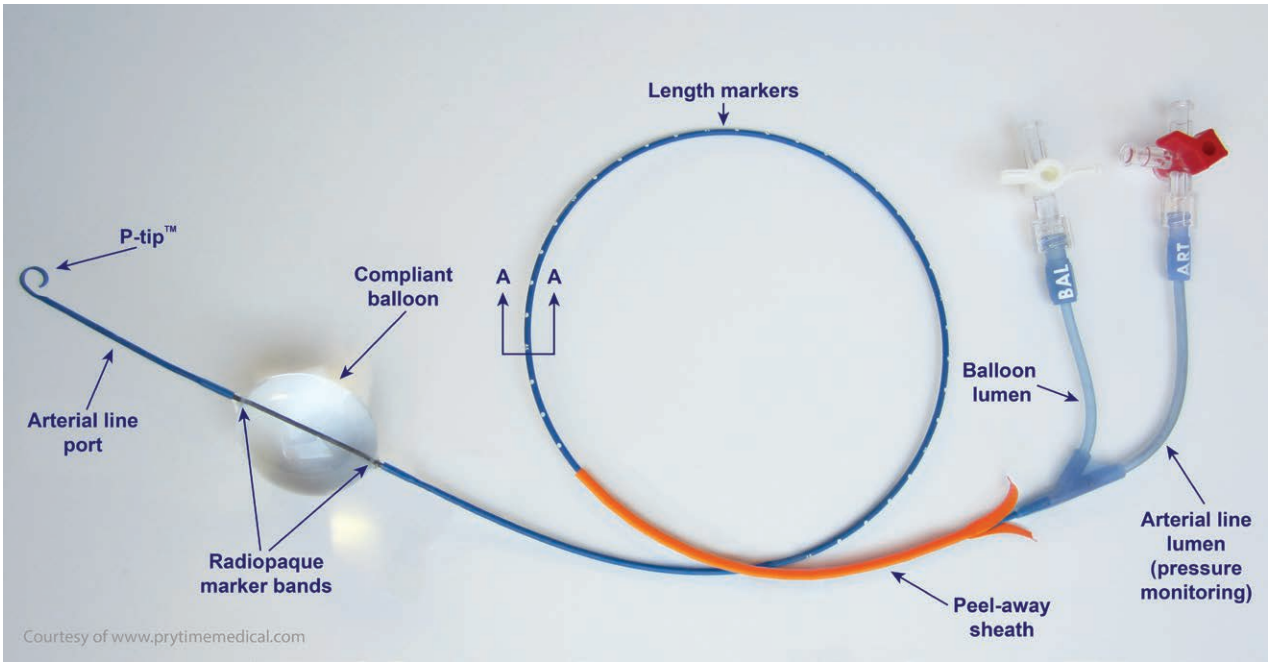

Figure 1. Catheter and balloon example (PryTime Medical ER-REBOA Catheter). REBOA indicates resuscitative endovascular balloon occlusion of the aorta.

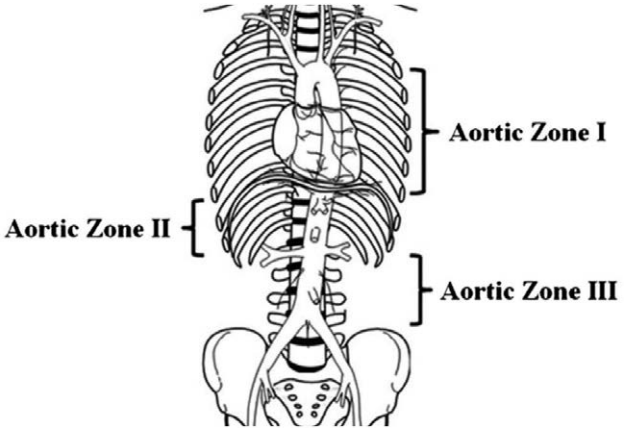

Figure 2. REBOA zones (Maryland.CCproject.com). Zone I: From origin of the left subclavian artery to the celiac artery (infradiaphragmatic). Zone II: Abdominal aorta from the celiac to lowest renal artery. Zone III: The infrarenal abdominal aorta between the lowest renal artery and the aortic bifurcation.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

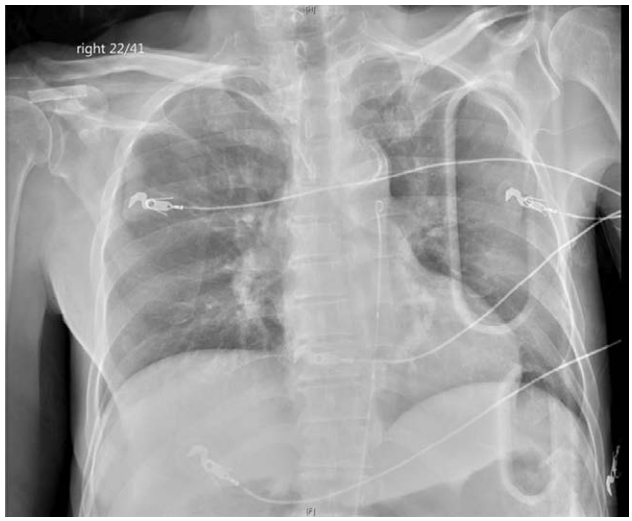

Figure 3. Chest x-ray showing REBOA catheter tip location in zone I distal to the aortic arch. REBOA indicates resuscitative endovascular balloon occlusion of the aorta.

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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