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;XXX:00–00)

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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Table 1

Title: Physiology of balloon occlusion at different aortic levels. Data is from human studies [14,15,16]

Physiologic Variable	Zone 1	Zone 3
Mean arterial pressure	↑↑ (38-54%)	↑ (2-8%)
Pulmonary capillary wedge pressure	↑↑↑ (90-190%)	No change
Central venous pressure	↑↑ (35%)	No change
End-systolic area	↑↑ (69%)	↑ (11%)
End-diastolic area	↑↑(28%)	↑ (9%)
Ejection fraction	↓↓ (38%)	No data
Cardiac index	↓↓ (29%)	No data
Cardiac wall motion abnormality	Significant (detected in 92% patients)	Limited or none

Legend: ↑= increase; ↓= decrease;

Table 2

Title: Comparison of Complete REBOA (C-REBOA) with Partial-REBOA (P-REBOA) – Data from swine model[18]

Variable	C-REBOA (n=5)	P-REBOA (n=5)
Proximal mean arterial pressure	Supraphysiologic (>110mmHg) augmentation throughout occlusion	Absence of supraphysiologic augmentation
Pressure gradients between proximal and distal systolic pressures (average)	90%	50%
Blood lactate at 90 minutes (average)	9.3 mmol/L	3.2 mmol/L
Evidence of duodenal ischemic necrosis at 90 minutes	60% of animals	20% of animals
Evidence of acute tubular necrosis at 90 minutes	80% of animals	No animals

Legend: mmHg = millimeters of mercury; mmol/L = millimoles per liter