# REBOA in Severe Brain Injury: Fatal Combination or Another Treatment Alternative?

# Khan-Kessel Corner

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Resuscitate thoracotomy (RT) is considered as a last resort to save lives of massively bleeding patients in Class IV hemorrhagic shock. Aortic cross-clamping of the descending thoracic aorta redistributes the patient's limited blood volume to the vital organs: myocardium and brain. The outcome of this procedure is arterial hypertension above the clamp and hypotension below the clamp. This dramatic intervention in patient physiology has multiple consequences, including significant increased cerebral blood flow. During the last decade, a new technique - resuscitate endovascular balloon occlusion (REBOA) - has emerged as a viable alternative to open aortic clamping. The most significant advantage of this technique is that, in selected patients, its minimally invasive nature allows it to be proactively employed prior to critical hemodynamic deterioration. Due to increasing experience and the spread of the learning curve, multiple studies have already been published, but mortality data on REBOA use is conflicting. Actually, the rationale for both techniques is the same, with the only difference being in the way aortic control is obtained - extra or intraluminally. Interestingly, in the era of open thoracotomy and aortic clamping, suspected or proven severe traumatic brain injury (TBI) was not considered as an absolute contraindication for this procedure. However, the use of REBOA in these patients is severely criticized, most probably due

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This is an open access article published under the terms of the Creative Commons Attribution License (CC BY 4.0), which permits use, distribution and reproduction in any medium, provided the original work is properly cited. to the potential significant impact on brain physiology. The first expert opinion study on REBOA use in 2019 did not reach consensus on whether TBI patients may experience any benefit on REBOA use [1]. However, the accumulative data from recent years demonstrate that the issue is still under active investigation and answers are yet to be determined. Elkbuli et al., in a study on patients suffering TBI who underwent REBOA, showed that inpatient mortality with REBOA does not differ between patients with or without concomitant TBI [2]. Furthermore, in a large study spanning 8 years, Brenner et al., found that Zone 1 REBOA had better outcomes compared with RT in all patterns of injury, including TBI patients [3].

We raise several questions that should be addressed, and we will be happy to open discussion on our journal pages. Theoretically, supra-physiological blood pressure and increased carotid blood flow induced by aortic occlusion may worsen cerebral edema, increase intracranial pressure, or exacerbate intracranial hemorrhage. The question we need to ask is how dangerous is it to occlude the aorta in TBI patients? Will increasing cerebral blood flow (CBF) exacerbate the bleeding? Will increased CBF result in intra-cranial pressure (ICP) elevation and affect neurologic outcomes? Does this increase the risk of potential brain herniation?

The true effect of aortic occlusion on the injured brain is unclear. Only a few investigations have been performed in this area, all of which have been conducted on animals. In 1990, Shackford et al., in a study on four groups of animals, found that aortic clamping improved perfusion to the injured brain without a significant increase in ICP. In addition, this study demonstrated that aortic occlusion elevated mean arterial pressure and appeared to have no detrimental effect on ICP, CBF and cerebral blood pressure [4]. One may claim that an increase in CBF may exacerbate existing intracranial hemorrhage. Currently, in our opinion, such statements cannot be proved or excluded. In a single-animal study, Cralley et al. demonstrated that REBOA does not worsen brain injury [5]. Similar results were demonstrated in Johnson et al.'s study, which did not demonstrate any differences in the percentage of animals with hemorrhage progression on CT after REBOA. Another very interesting finding of this study was that in the animal model, rapid blood resuscitation, and not REBOA, resulted in the largest increase in ICP [6]. In addition, the impact of multiple brain autoregulation mechanisms in "artificially" changed brain physiology is completely unclear. In summary, we feel that, as clear conclusions regarding REBOA are unlikely to be established in animal models, larger randomized investigations utilizing human subjects are urgently needed.

## **Ethics Statement**

- (1) All the authors mentioned in the manuscript have agreed to authorship, read and approved the manuscript, and given consent for submission and subsequent publication of the manuscript.
- (2) The authors declare that they have read and abided by the JEVTM statement of ethical standards including rules of informed consent and ethical committee approval as stated in the article.

# **Conflicts of Interest**

The authors declare that they have no conflicts of interest.

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