Published in final edited form as:

Crit Care Med. 2017 November ; 45(11): e1194–e1195. doi:10.1097/CCM.0000000000002663.

Cardiac Dysfunction Following Traumatic Brain Injury

Vijay Krishnamoorthy, MD, PhD^{1,2,8}, Ali Rowhani-Rahbar, MD, MPH, PhD^{2,8}, Edward F. Gibbons, MD^{3,8}, Frederick P. Rivara, MD, MPH^{2,4,8}, Nancy R. Temkin, PhD^{5,6,8}, Nopphanan Chaikittisilpa, MD⁸, Kevin Luk, MD, MS¹, and Monica S. Vavilala, MD^{1,8}

¹Department of Anesthesiology and Pain Medicine, University of Washington

²Department of Epidemiology, University of Washington

³Department of Medicine, Division of Cardiology, University of Washington

⁴Department of Pediatrics, University of Washington

⁵Department of Neurosurgery, University of Washington

⁶Department of Biostatistics, University of Washington

⁷Georgetown University School of Medicine

⁸Harborview Injury Prevention and Research Center, University of Washington

Keywords

traumatic brain injury; echocardiography; cardiac

We would like to thank Longhi, et al for their thoughtful comments (1) regarding our manuscript recently published in *Critical Care Medicine* (2), which demonstrated that early systolic dysfunction is common and reversible following moderate-severe traumatic brain injury (TBI). We agree with the authors that TBI-induced cardiac dysfunction likely shares a common underlying pathophysiology with cardiac dysfunction after other neurologic injuries (i.e. subarachnoid hemorrhage), namely a massive release of catecholamines. To illustrate this, recent work by our group has demonstrated that patients with TBI-induced cardiac dysfunction have a distinctive early hemodynamic profile that is likely secondary to a maladaptive catecholamine-excess state (3).

Longhi, et al expressed important points regarding the indication for vasopressors in our cohort, the need for more detailed measures of systolic function, and the relationship between cardiac dysfunction and radiographic TBI severity. In our study, vasopressor use was mainly driven by systolic blood pressure (SBP) and was initiated when the SBP was less than 90 mmHg and unresponsive to fluid therapy. We agree with Longhi, et al that future studies on TBI-induced cardiac dysfunction must consider a more nuanced evaluation of

Corresponding Author: Vijay Krishnamoorthy, MD PhD, Assistant Professor, Anesthesiology and Pain Medicine, University of Washington, 1959 NE Pacific Street, BB-1469, Seattle, WA 98195, Phone: 206-543-2673, Fax: 206-543-2673, vkrish@u.washington.edu.

Conflicts of Interest: None

The remaining authors have disclosed that they do not have any potential conflicts of interest.

cardiac dysfunction, including measures that are less dependent on loading conditions. While fractional shortening and ejection fraction are traditional measures of systolic cardiac performance, recent advances in cardiac imaging using speckle tracking have led to earlier detection of systolic dysfunction in critical illnesses, including sepsis (4) and subarachnoid hemorrhage (5). Speckle tracking is a novel technology that quantifies myocardial strain in the circumferential, radial, and longitudinal planes. Global longitudinal strain is more sensitive than ejection fraction in detecting impaired systolic function (6) and may allow for early detection of subclinical systolic dysfunction following TBI. Lastly, while the small sample size of our study did not allow a detailed evaluation of specific markers of clinical/radiographic severity and systolic dysfunction, prior work from our group has suggested that clinical TBI severity may be more predictive of systolic dysfunction than radiologic severity (7).

We hope that our paper will inform future studies that aim to personalize hemodynamic management after acute brain injury, rather than the current “one size fits all” approach. Our data suggests that the cardiac response to brain injury is heterogeneous, and potentially needs to be considered when making decisions regarding fluid management and pharmacologic manipulation of hemodynamics following TBI. Future studies on this topic are urgently needed.

Acknowledgments

Funding: NIH – L30 NS084420

Copyright form disclosure: Dr. Krishnamoorthy received funding from the National Institutes of Health (NIH). Drs. Krishnamoorthy and Vavilala received support for article research from the NIH.

References

1. Longhi L, Ferri F, Cavalleri G, et al. Cardiac Function Following Traumatic Brain Injury. *Crit Care Med.* 2017 in press.
2. Krishnamoorthy V, Rowhani-Rahbar A, Gibbons EF, et al. Early Systolic Dysfunction Following Traumatic Brain Injury: A Cohort Study. *Crit Care Med.* 2017; 45:1028–36. [PubMed: 28398926]
3. Krishnamoorthy V, Rowhani-Rahbar A, Chaikittisilpa N, et al. Association of Early Hemodynamic Profile and the Development of Systolic Dysfunction Following Traumatic Brain Injury. *Neurocritical care.* 2017; 26:379–87. [PubMed: 28000133]
4. Sanfilippo F, Santonocito C, Panarello G, Arcadipane A. The role of speckle tracking echocardiography for prognostication in patients with severe sepsis or septic shock. *Crit Care.* 2016; 20:284. [PubMed: 27616201]
5. Cinotti R, Piriou N, Launey Y, et al. Speckle tracking analysis allows sensitive detection of stress cardiomyopathy in severe aneurysmal subarachnoid hemorrhage patients. *Intensive care medicine.* 2016; 42:173–82. [PubMed: 26499884]
6. Leung DY, Ng AC. Emerging clinical role of strain imaging in echocardiography. *Heart Lung Circ.* 2010; 19:161–74. [PubMed: 20149727]
7. Urdaneta AE, Fink KR, Krishnamoorthy V, Rowhani-Rahbar A, Vavilala MS. Radiographic and Clinical Predictors of Cardiac Dysfunction Following Isolated Traumatic Brain Injury. *J Intensive Care Med.* 2017; 32:151–7. [PubMed: 26584593]