Circulation

RESPONSE TO LETTER TO THE EDITOR

Response by Bascom and Seder to Letter Regarding Article, "Derivation and Validation of the CREST Model for Very Early Prediction of Circulatory Etiology Death in Patients Without ST-Segment–Elevation Myocardial Infarction After Cardiac Arrest"

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In Response:

We appreciate the commentary by Dr Voicu et al about our recent manuscript[1] proposing a simple scoring system to assess the risk of circulatory etiology death after resuscitation from cardiac arrest. Identification of patients likely to die from circulatory etiology death is important, not only to identify those who may benefit from mechanical circulatory support, but also to select for early angiography and revascularization and to choose temperature targets, experimental neuroprotective measures, or other individualized treatments. Triage of resuscitated patients should be based on the competing risks of circulatory and neurological etiology death, maximizing the effectiveness of various treatments by offering them to the patients most likely to benefit. This early risk stratification also facilitates selection of appropriate subpopulations for clinical trials.

We were interested to hear of work predicting circulatory etiology death after resuscitation using admission pH and shock.[2] Our study population differs in that we excluded patients with ST-segment–elevation myocardial infarction. In the United States the treatment pathway for such patients is established,[3] unlike those with VT/VF and presumed cardiac etiology but no ST-segment–elevation myocardial infarction.[4] Additionally, their study included patients from a single center, likely treated under a single protocol, whereas ours involved 44 centers in multiple countries with associated variations in practices and outcomes that may have contributed to the lower c-statistic in our cohort.

We acknowledge potential to strengthen our model by the addition of pH, which was not available in the cohort we evaluated. In previous studies, however, low pH at admission is associated with both circulatory and neurological death,[5] and may not help distinguish between these poor outcomes. Furthermore, the timing of pH measurement is important; an (unpublished) analysis at our center showed that persistent or worsening metabolic acidosis at 4 hours after resuscitation was an independent predictor of poor outcome, usually because of circulatory collapse, and performed better than admission pH, which tracked closely with duration of ischemia.

The definition of shock used in our registry is derived from the American College of Cardiology (ACC) definition, and applied during the first 4 hours of admission. We agree that this reflects a severe phenotype, and is like the INTERMACS class 1 definition (Interagency Registry for Mechanically Assisted Circulatory Support), but we emphasize the assessment of competing risks before initiating mechanical circulatory support, as bleeding associated with advanced circulatory assist devices could outweigh potential benefits if the brain injury is severe. Clearly these competing risks are already being assessed informally at the bedside, as our cohort included no patients with shock and severe brain injury who received advanced circulatory support.

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In the CREST model (CAD, heart rhythm, ejection fraction, shock, ischemic time), echocardiography was included if performed within 6 hours of admission. We agree that contractility is in flux after resuscitation and depends on many factors. But in our cohort, low admission ejection fraction was strongly associated with circulatory etiology death, and we feel that echocardiography has an important role to play in early triage. The best techniques to assess circulatory and neurological risks after resuscitation will continue to evolve, but the development of individualized treatment pathways based on those data should be increasingly fundamental to postresuscitation care.

ARTICLE INFORMATION

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Disclosures

None.

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