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Editorial

# The Changing Landscape of Cardiogenic Shock: One Step Closer to Speaking a Common Tongue

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"Progress is impossible without change, and those who cannot change their minds cannot change anything."

George Bernard Shaw (1944)

The clinical syndrome we recognize as cardiogenic shock (CS) traces back to seminal observations made over 100 years ago.[1] In 1912, Dr James B. Herrick reported on preclinical and human experiences wherein acute obstruction of an epicardial coronary artery did not result in immediate death, thereby contradicting the prevailing belief of the day.[1,2] Rather, patients lingered in varying states of hemodynamic compromise, descriptions of which chronicle progression from what is now known as SCAI stage C to stages D/E CS and ultimately death in the majority of patients. Dr Herrick also noted that occasionally, "functionally complete recovery ensues" and that "no simple picture of the condition can, therefore, be drawn."[2] The latter statement, which still holds true today, underscores the critical importance of ongoing work such as the SCAI SHOCK Clinical Expert Consensus Update.[3]

On the path to modern CS care, several notable historic milestones provide context for points specifically highlighted in the current consensus document. In 1942, Drs Stead and Ebert detailed 2 distinct phenotypes of a "shock syndrome produced by failure of the heart" using clinical, radiographic, and laboratory data.[4] In 1967, Drs Killip and Kimball described 4 distinct classes of "myocardial derangement" in patients with acute myocardial infarction (AMI), assigning for the first time specific blood pressure parameters to a shock diagnosis.[5] In the same year, the first human intra-aortic balloon pump (IABP) implant was performed by Kantrowitz et al in a young patient with AMI with CS (AMICS), successfully bridging the patient to recovery and discharge.[6] In 1970, Drs Swan and Ganz first described the use of their eponymous flow-directed catheter, ushering in the era of invasive hemodynamic assessment.[7] Despite numerous iterative advances, however, mortality in CS remained extremely high. Reports began to emerge in the mid-1980s of modest improvements in survival with early surgical revascularization and with fibrinolytic therapy.[1] In 1988, Lee and colleagues published a 10-year retrospective experience comparing serial cohorts of patients with AMICS, treated with or without percutaneous coronary intervention (PCI) and observed dramatic improvement in 30-day survival from 17% with conventional therapy to 50% with the addition of successful coronary angioplasty.[8] Definitive proof of this concept came in 1999 with the landmark SHOCK trial, the first prospective, randomized study in AMICS comparing a strategy of early revascularization to initial medical stabilization with drug therapy and IABP. Numerical improvements in 30-day mortality with early revascularization became statistically significant 6 and 12 months later, establishing emergent PCI as the default therapy for AMICS.[9] As the number of CS registries and RCTs grew, so too did variability in reported survival, ascribable in part to unquantified differences in baseline severity of illness, i.e., the proportion of patients who were already *in extremis* at the time of study entry.[1,3] Twenty years later and 107 years after the first credible description of CS, the 2019 SCAI clinical expert consensus statement on the classification of CS codified CS diagnosis and risk stratification, quickly gaining multisociety endorsement as well as wide recognition by clinicians.[10]

In this inaugural issue of the *Journal of the Society for Cardiovascular Angiography and Interventions (JSCAI)*, Naidu SS, on behalf of the SCAI SHOCK Writing Group, presents the revised SCAI SHOCK Stages Classification Clinical Expert Consensus Update. This document expands the scope of the original schema through validation of its discriminatory capacity, more granular recommendations for bedside, hemodynamic, and laboratory assessment of shock severity and by expanding the breadth of CS assessment along a proposed 3-axis model of evaluation and prognostication.

First, the updated statement offers broad validation of the original SCAI SHOCK schema across differing CS states and treatment environments, encompassing data from more than 25,000 patients studied in the context of 9 (predominantly retrospective) studies conducted since the 2019 statement. SCAI SHOCK staging reliably predicted mortality, although it should be noted that retrospective, single (rather than prospective and/or serial) assignment of stage was performed in the majority of studies with variability in study-specific criteria for assigning stages C-E, inconsistent differentiation between acute versus chronic renal

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dysfunction as a marker of hypoperfusion, and limited availability of hemodynamic data. Table 3 of the revised schema offers much-needed guidance for improving the precision of CS staging, vis-à-vis numerical cutoff values, categories broken down by typical clinical features, and therapy-based criteria to judge transitions between stages.[3] Despite this, it should be recognized that many aspects of CS staging still entail some degree of subjectivity and thus may result in variable staging, even within a given center. Similarly, timing and implementation of therapies that follow staging, such as mechanical circulatory support (MCS), are highly variable and will impact how the recovery/deterioration pathways outlined in Figure 5 are applied within a given system of care.[3] Greater visual clarity is given to the SCAI SHOCK pyramid figure (Figure 4), now depicted in gradations of color to reflect increasing severity within each stage. The inherent heterogeneity of cardiac arrest (CA) is acknowledged, and accordingly, the "A" modifier is now designated only for patients with CA with suspected anoxic brain injury. The revised statement also incorporates a 3-axis model (Figure 3) to aid in CS prognostication, separating out various modifiable and nonmodifiable factors that determine shock phenotype and, in turn, drive mortality.[3] While this construct appropriately recognizes the impact of cardiometabolic derangements, systemic inflammation, and congestion profile on outcomes, it is not immediately translatable to clinical decision-making. Furthermore, its applicability may vary based on CS etiology and chronicity of the preshock state, as in patients with chronic heart failure progressing to CS.

In summary, the 2021 SCAI SHOCK Stages update meaningfully advances CS risk stratification and prognostication without departing from the simplicity of the original model. That stated, many opportunities exist to further improve the utility of the model. The "A" modifier is now applicable only to CA with neurologic injury, but this determination usually takes time and is affected by numerous factors; moreover, end-organ damage sustained during the arrest often independently contributes to poor outcomes. The deliberate omission of age as a criterion is also problematic, as advanced age predicts poor outcomes across the spectrum of critical illness; further consideration of these 2 variables would be welcome. Similarly, incorporation of serial hemodynamic and laboratory assessments and standardization of the methodology and frequency of clinical assessment may sharpen the calibration of the model and may help inform optimal timing of therapies such as mechanical circulatory support. Ultimately, however, the clinical impact of this statement will less likely hinge on further refinements, but rather on broad adoption by the global community of cardiovascular clinicians and trialists as the new *lingua franca* for acute cardiogenic shock.

### *Declaration of competing interest*

Dr Nathan has served as a consultant to Abiomed, Cardiovascular Systems, Inc, and Getinge. Dr O'Neill has served as a consultant to Abiomed.

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