

current practice of emergency medicine. Recommendations for education, training experience and quality improvement in the emergency setting are well described in the literature. POCUS can reliably be used to identify reversible causes of CA [1]. However, POCUS use during cardiac arrest management may be associated with prolonged CPR pauses [2]. TEE could be used to diagnose reversible causes of collapse, optimize the quality of chest compressions, guide therapies, and identify cardiac activity [3]. Despite potential advantages during resuscitation, TEE is not currently widely adopted in the routine practice of ED. As described in the « 2025 European Resuscitation Council Guidelines », further studies are needed to better define the diagnostic value of TEE in identifying reversible pathology and myocardial contractility in cardiac arrest patients [4]. Development of a comprehensive guide is also essential to define the minimum training requirement for an effective implementation of TEE into emergency medicine practice. In the meantime, developing local CPR protocols in collaboration with TEE-proficient specialists may offer crucial information to physicians involved in cardiac arrest management (Figure 2). Conclusion: Focused cardiac ultrasound during resuscitation represents an increasingly important skill for the modern critical care physician. Understanding cellular physiology and its integration into the management of cardiac arrest may facilitate decision-making during resuscitation. Our experience demonstrates that point-of-care TEE can be instrumental in the diagnosis and treatment guidance of cardiac arrest and acute aortic syndrome. Further studies are required to validate the clinical benefit and feasibility of implementing TEE during CPR.

Sodium bicarbonate for hospitalized patients with metabolic acidosis: a systematic review and meta-analysis of randomized controlled trials

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Introduction: Intravenous bicarbonate is a commonly used agent in the management of acute metabolic acidosis. However, its clinical benefit and safety is largely uncertain. We summarized the existing randomized controlled data and examined whether the use of intravenous bicarbonate, as compared to control or an alternate type of fluid without

bicarbonate, leads to improvements in patient-important outcomes in those hospitalized with acute metabolic acidosis. Methods: We searched Ovid MEDLINE, Embase, the Cochrane library, and Clinicaltrials.gov, from inception until Jan 2nd 2026 and included randomized controlled trials of hospitalized adult participants who received intravenous bicarbonate, as compared to control or an alternate type of fluid without bicarbonate, for metabolic acidosis. We excluded studies examining oral bicarbonate. We pooled data using a random-effects model and present risk ratios (RRs) or mean differences (MDs), along with 95% confidence intervals (CIs). We assessed individual study risk of bias using the modified Cochrane tool, and certainty of evidence using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) approach. Results: Of 1066 citations, we included 9 RCTs (n = 1331 patients), three of which focused on patients exclusively with severe diabetic ketoacidosis, with initial pH values ranging from 7.00 -7.06. Pooled analysis showed that bicarbonate may be associated with decreased mortality (RR 0.93, 95% CI: 0.82–1.05, low certainty), and decreased need for renal replacement therapy (RR 0.69, 95% CI 0.60 to 0.80, low certainty) compared to not using bicarbonate. Bicarbonate may have no effect on length of hospital stay (MD of 0.41 days longer, 95% CI 3.54 fewer to 4.36 days longer, low certainty). Conclusion: Bicarbonate may reduce mortality and need for renal replacement therapy in hospitalized patients with metabolic acidosis, but further data is needed to increase precision in effect estimates and further assess the impact of bicarbonate on other patient-important outcomes.

Physiology at the edge: septic shock in BiVAD-supported patients. What happens to shock physiology when the heart is no longer the pump?

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Introduction: Mechanical circulatory support devices have become an integral component of modern cardiac intensive care, significantly improving survival in advanced heart failure. Implantation of a biventricular assist device (BiVAD) profoundly alters cardiovascular physiology, creating a state of device-dependent circulation in which pump flow, rather than intrinsic myocardial performance, determines cardiac output. With Frank–Starling mechanism no longer functioning adequately, and the presence of concomitant autonomic and endothelial dysfunction, the heart's capacity to respond to physiological stressors is substantially compromised. In this context, the onset of septic shock may generate metabolic and hemodynamic demands that exceed the adaptive capacity of device-supported circulation. Methods: We analyzed the pathophysiological mechanisms underlying septic shock in BiVAD supported patients, with particular focus on hemodynamic responses to pump flow adjustments and on laboratory parameters reflecting tissue perfusion, inflammation, and organ dysfunction. In order to respond