Commentary

Heart during acidosis: Etiology and early detection of cardiac dysfunction

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A R T I C L E   I N F O

Commentary on "Systemic Acidemia Impairs Cardiac Function in Critically Ill Patients" by Rodrigez-Villar et al. (EClinical Medicine, 2021)

Physiology can inform and help transform clinical practice. In turn, clinical practice demands physiological mechanisms to determine intervention and treatment. And so the spiral of fresh scientific discovery and its clinical application goes on.

Lactic acidosis is a common adverse finding in the critically ill patient. Physiological studies in preclinical animal models have long established that systemic acidemia can impair cardiovascular function, weakening cardiac contractility and reducing peripheral vascular reactivity to constrictor agonists [1]. However, underlying mechanisms have not been delineated, particularly in humans. Of greater importance, no human clinical trial until the work by Rodrigez-Villar et al. published in this issue of EClinicalMedicine [2] had established a threshold arterial pH, below which cardiac function becomes compromised. This commendable and difficult-to-execute study not only confirms a negative relationship between arterial pH and cardiac dysfunction in humans, but it demands questions with potential answers that have the power to transform the clinical management of patients with severe acidemia, such as those suffering from septic shock or cardiac arrest. What is so critical about a threshold arterial pH of 7.28 below which cardiac function deteriorates? What is the underlying physiological mechanism linking acidemia and cardiac dysfunction in humans? Is acidemia a cause or effect of cardiac dysfunction in critically ill men and women? Can respiratory versus metabolic acidosis differentially affect cardiac function? What is the best therapy for normalising pH and is there a statistical relationship between buffering pH and improved cardiac function in the ICU patient?

How acidemia affects cardiovascular function during fetal life before birth has also been an area of intense scientific interest for many years, largely because it is a common outcome of labour and delivery [3]. During labour, compression of the umbilical cord can lead to fetal hypoxia and trigger a carotid chemoreflex that reduces heart rate and redistributes cardiac output away from peripheral circulations towards the fetal brain, the so-called fetal brain sparing response [4,5]. Lactic acidemia in the fetus results from the anaerobic metabolism of glucose in tissues, particularly in those undergoing peripheral vasoconstriction as a result of the fetal brain-sparing reflex [6]. A study in late gestation, mature, fetal sheep showed that acidemia enhanced the magnitude of the fetal cardiovascular and endocrine defences to acute stress, but it reversed the increase in umbilical vascular conductance to vasoconstriction, during an episode of acute hypoxia. Therefore, it is clear that acidemia in fetal as well as adult life can significantly impact cardiovascular function, however underlying mechanisms remain to be fully explored. It is also clear that, whether in fetal or adult life, acidemia rarely occurs in isolation, often being accompanied by altered arterial blood gas status and increased levels of stress hormones in the circulation. If this is the case, then how can the clinician detect incipient cardiac decompensation either during complicated labour in the fetus or in the ICU setting in the critically ill to offer life-saving interventions such as ECMO [7]? This challenge has been tackled recently in preclinical studies where a machine learning algorithm leveraging continuous heart rate data has enabled more precise, individualized medicine with real-time prediction of cardiovascular decompensation [8].

Interestingly, the study of Rodrigez-Villar et al. does not present real-time cardiovascular data in the patients, such as the electrocardiogram (ECG) and arterial blood pressure (ABP), because these cardiovascular outcomes were not accessible to the researchers. This can no longer be the status quo in 21st century medicine. Hospitals spend considerable resources to be able to afford real-time monitoring of such indispensable physiological data in the ICU setting. Patients enrolled in the study have the right to these data, yet clinicians are often unable to access the raw data to conduct the critical clinical analysis. In light of the growing mass of evidence that unequivocally supports that access to such data can transform clinical care, this situation needs to change. Access to ECG and ABP in-depth data analysis would enable the development of real-time prediction of cardiovascular decompensation. In turn, this would lead to earlier interventions, reducing morbidity and mortality. Therefore, we call upon the manufacturers of the clinical ICU monitoring devices to remove their silos around accessing cardiovascular raw data. Doing so would boost badly needed clinical research and algorithm

development, both of which have the potential to help save thousands of lives.

Declaration of Competing Interest

M. G. Frasch has patents on aECG (WO2018160890) and EEG technologies for fetal monitoring (US9215999). The authors declare that the research was conducted in the absence of any other commercial or financial relationships that could be construed as a potential conflict of interest.

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