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Editorial

Echocardiography in post-resuscitation care: “Always look on the right side”



Bedside echocardiography has been incorporated as a valuable non-invasive diagnostic and monitoring tool in the management of critically ill patients, including victims of cardiac arrest (CA). Point-of-care ultrasound during resuscitation might be useful to detect potential reversible causes of CA (cardiac tamponade, massive pulmonary embolism, hypovolemia . . .), identify true absence of cardiac mechanical activity and predict short-term outcome,¹ although attention must be paid to not increasing hands-off time.² In addition, the role of echocardiography in the post-resuscitation period to detect and quantify the degree of myocardial dysfunction, monitor haemodynamic conditions and guide therapeutic interventions is well-established.³

Traditionally, ventricular function assessment in post-resuscitated patients has been largely focused on the “left side” of the heart. In this sense, the prognostic value of left ventricular (LV) dysfunction has been widely described.^{4–7} However, in the last decade there has been increasing interest in the study of the right ventricle, as we have gone deeper in the understanding of its distinctive physiology, closely dependent on haemodynamic conditions and indivisibly related to LV performance. Right ventricular (RV) dysfunction has been recognised as a strong predictor of mortality in various contexts, including myocardial infarction (MI),⁸ heart failure,⁹ pulmonary hypertension,¹⁰ congenital heart disease¹¹ and cardiothoracic surgery.¹² In the CA setting, RV morphology and/or systolic function alterations are common within the first 24 h, regardless of the aetiology of arrest.¹³ However, their clinical significance and impact on patient outcomes has been less characterized.

The study conducted by Patel et al. in the current issue of *Resuscitation*, *Impact of right ventricular dysfunction on mortality in adults with cardiac arrest undergoing coronary angiogram*, attempts to answer some of these questions. This observational retrospective cohort study including 147 adult patients admitted with CA sought to assess the association between RV dysfunction, obstructive coronary artery disease (CAD) and clinical management, and identify the impact of RV dysfunction on all-cause in-hospital mortality.

One of its principal strengths lies in the thorough collection of relevant clinical data in a cohort of CA patients with high suspicion of underlying myocardial ischemia. This included patients’ characteristics, type of MI, extent and severity of CAD, revascularization strategy -including coronary artery intervened, use of aspiration thrombectomy, fibrinolytics and glycoprotein IIb/IIIa inhibitors-, as well as use of vasopressors and inotropes, mechanical support and therapeutic hypothermia. Thus, the authors provide a valuable clinical

snapshot of CA patients with and without RV dysfunction. Patients with RV dysfunction had a higher prevalence of previous history of CAD, multivessel, left main and right coronary artery involvement at the index event, lower LV ejection fraction and higher haemodynamic instability. Furthermore, the multivariable analysis conducted pointed at RV dysfunction as the strongest independent predictor of higher in-hospital mortality [odds ratio (OR) 4.71, 95% confidence interval (CI) 1.27–17.50] independently of LV ejection fraction and accounting by age, gender, use of vasopressors, mechanical support and therapeutic hypothermia.¹⁴

In a clinical scenario where LV dysfunction has been in the spotlight for decades, Patel et al. make a valuable contribution to previous research exploring the role of the “right side” of the heart in the clinical course and prognosis of CA patients. A previous study by Ramjee et al. using data from the Penn Alliance for Therapeutic Hypothermia (PATH) registry investigated the prevalence of RV dysfunction among CA victims within the first 10 h from the event, and its potential impact on patients’ survival and neurological outcome.¹⁵ In this cohort, RV dysfunction was associated with poorer outcome after accounting by LV dysfunction, duration of CA, initial rhythm, targeted temperature management and dose of epinephrine. Interestingly, this relationship was strengthened when considering RV dilation. However, the PATH registry lacked relevant clinical data about patients’ baseline characteristics, aetiology of arrest, type of MI and management details, including revascularization, inotropes and vasopressors use and mechanical support, all of which were considered in the study by Patel et al.

Prognostication of survival and neurological outcomes of resuscitated victims is a field in constant development. In this regard, patient clinical information (such as concurrent severe disease) and event-related facts (witnessed event, early CPR, defibrillable initial rhythm . . .) are well-known prognostic factors.^{16,17} However, final outcome depends on many other circumstances, making necessary the combination of clinical parameters, biomarkers, brain imaging and electrophysiological examination to estimate the degree of brain injury and the risk of mortality.^{3,18} In this multidimensional approach emphasizing neurological predictors cardiac markers have been less explored, which is consistent with the fact that once spontaneous circulation is restored and the patient is haemodynamically stable, hypoxic-ischaemic brain injury will be the principal determinant of prognosis and the main focus of critical care.³ However, in the same way that commonly used cardiac markers such as high-

sensitivity troponin have shown to be independently associated with all-cause mortality after CA,¹⁹ accessible tools such as bedside echocardiography could add valuable prognostic information at a low cost and with no harm to the patient.

There might be some areas for improvement for studies in this field. The first is the lack of a standardized definition and quantification of RV dysfunction. RV contractility is determined by three factors: inward movement of the free wall, longitudinal shortening towards the apex and traction on the free wall secondary to LV contraction. Moreover, RV systolic global function is strongly influenced by preload (RV filling) and afterload (pulmonary vascular resistance), heart rhythm, synchrony of ventricular contraction and ventricular interdependence.²⁰ A comprehensive evaluation of RV function should assess these components and consider factors such as heart valve disease, arrhythmia or shunts. Qualitative “visual” assessment by an expert operator is considered valid to guide quick clinical decisions, but speaking a common language might be needed when staging dysfunction for prognostication purposes, for the sake of reproducibility. Different criteria may well explain the striking differences in the prevalence of RV dysfunction between series, varying from 13.6% in the cohort of Patel et al.,¹⁴ 59% in the PATH registry¹⁵ and 96.6% in the study by Wardi et al.,¹³ (the only to provide a definition based on quantitative parameters). Secondly, physiopathology and clinical implications of acute RV dysfunction (due to ischaemia, abrupt changes in loading conditions or use of mechanical support devices) differ significantly from chronic dysfunction (secondary to LV failure, pulmonary hypertension, heart valve disease . . .). For instance, pre-arrest LV dysfunction was found to predict poor outcomes after CA,⁴ and its recovery was proven to have more impact than its initial severity on long-term survival. Hence, considering pre-arrest RV function, accounting for changes over time and characterising mechanisms should be considered by future studies to refine results and draw more reliable conclusions.⁶

In summary, the valuable work of Patel et al. presents new and consistent data pointing at RV dysfunction as an important determinant of poor in-hospital survival in a cohort of patients with highly suspected ischaemic cause for CA undergoing coronary angiogram. This study further expands previous research exploring the usefulness of echocardiography as a non-invasive and accessible tool in post-resuscitation care. Moreover, it contributes to the slow “rightward shift” in the study of myocardial dysfunction in this setting, usually focused on the left ventricle and now integrating the right ventricle in the complex equation of prognostication after CA.

Conflicts of interest

None.

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