

# In-hospital resuscitation of Covid-19 patients is impeded by serious delays, but the problem is obscured by poor time data

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**This commentary refers to ‘Cardiac arrest in COVID-19: characteristics and outcomes of in- and out-of-hospital cardiac arrest. A report from the Swedish Registry for Cardiopulmonary Resuscitation’, by P. Sultanian et al., doi:10.1093/eurheartj/ehaa1067 and the discussion piece ‘Handling time elements for in-hospital cardiac arrest’, by P. Sultanian et al., doi:10.1093/eurheartj/ehaa163.**

The recent article about cardiac arrest survival for Covid-19 patients<sup>1</sup> gives useful information about the pandemic’s impact but has a major weakness: it presents time data from in-hospital cardiac arrests (IHCA) that are simply not believable. This is hardly a new phenomenon, but it takes on added significance in the setting of Covid-19.

The lack of accurate time data from IHCA is a longstanding problem that represents a major impediment to quality improvement and research,<sup>2</sup> and the problem is even more acute in the Covid-19 era. The Covid-19 pandemic requires significant delays in responding to cardiac arrests in hospitals, due largely to the time needed to don personal protective equipment (PPE).<sup>3</sup> As is common in the medical literature, the authors presumably get their numbers from retrospective chart reviews relying on handwritten records made during the events. In these source documents, apparent intervals are artificially shortened by the practice of using the time of first recorded observation/intervention as the time of arrest. Much time can go by before a recorder is designated and begins writing, effectively rendering any treatment delays due to Covid-19 invisible.

The article reports that pre- and post-pandemic time intervals showed ‘no differences in time to alert, time to CPR, or time to defibrillation in IHCA’. The reported median intervals, dubiously short to begin with (0 min to start of CPR, 2 min to defibrillation), were unchanged, suggesting that delays for donning PPE or other reasons related to Covid-19 simply did not occur. This obscures a major factor that undoubtedly decreases survival, leading to an inaccurate impression of the inherent lethality of Covid-19.

Quantifying these delays is essential to addressing the problem. Another recent study found wide disparities in survival among hospitals.<sup>4</sup> How much of these differences is due to variations in times to resuscitation interventions? This is unknown (to their credit, the authors mention this as a study limitation), and the question will remain unanswered without better time data. Similarly, the effectiveness of innovative ways to address delays, such as ILCOR’s recommendation to prioritize defibrillation,<sup>3</sup> will remain unknown.

The prevailing view appears to be that capturing accurate time data from in-hospital codes is essentially impossible—as evidenced by the fact that the most recent revision of the Utstein Guidelines all but ignores the issue.<sup>5</sup> However, improving the situation may not actually be difficult. An easy starting point might be to look at monitored tachyarrhythmic arrests: typically, the cardiac monitor record clearly shows arrest onset and distinctive artefacts marking periods of chest compressions and defibrillatory shocks. Tracking at least these intervals could be accomplished by virtually any hospital.

Dealing with treatment delays is perhaps the biggest immediate challenge to resuscitation efforts in the Covid-19 era. Quantifying the delays with accurate time data is essential if we are to ameliorate the problem and thereby improve survival.

**Conflict of interest:** J.A.S. is the sole author and reports no relevant conflicts of interest, affiliations, or grant support.

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## CARDIOVASCULAR FLASHLIGHT

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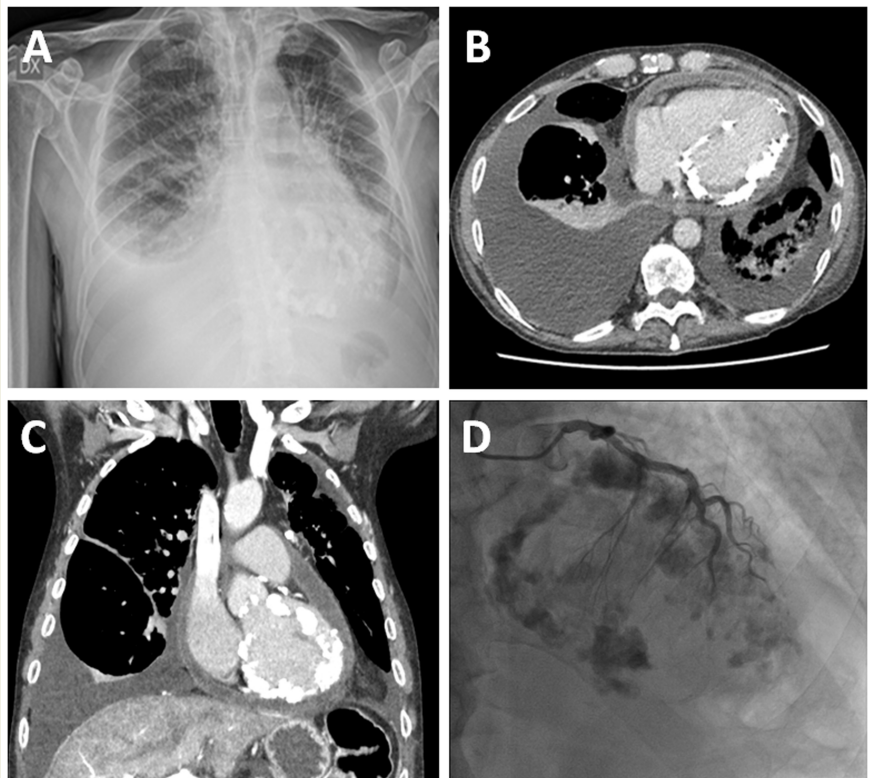
### ‘Heart of stone’: an unusual post-actinic sequela

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A 60-year-old man with a history of Hodgkin’s lymphoma was admitted to our institution for congestive heart failure. Twenty years earlier, he had received chemotherapy and radiotherapy and had developed late post-actinic sequela involving the lungs and heart. Chest X-ray and echocardiographic examination showed pulmonary congestion and fibrosis, pleural, and pericardial effusion along with reduced left ventricular ejection fraction (Panel A). However, the clinical picture was predominantly characterized by evidence of severe left ventricular calcifications extending inward from the epicardium and involving the interventricular septum and mitral apparatus, as documented on computed tomography chest scan (Panels B and C; [Supplementary material online, Video S1](#)). These impressive calcifications were also the prevailing features on coronary angiography which revealed the absence of significant coronary stenoses (Panel D; [Supplementary material online, Video S2](#)). Chest radiation exposure is associated with a substantial risk for the subsequent development of pulmonary and cardiovascular disease; however, massive calcifications that penetrate deep into the myocardial layers of the left ventricle have rarely been reported.



[Supplementary material](#) is available at *European Heart Journal* online.

**Conflict of interest:** The authors have submitted their declaration which can be found in the article [Supplementary Material online](#).