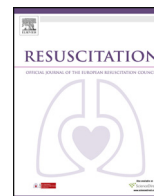




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## Letter to the Editor

**Rapid response ECLS after 140 min of refractory ventricular fibrillation following out-of-hospital cardiac arrest: Feasibility as bridge to primary PCI**


Sir,

A 42-year-old male with a history of smoking but no cardiovascular medical history, experienced a cardiac arrest at home. He has complained of angina the day before and in the early morning hours. His fiancé who witnessed the event immediately started with chest compressions and called the emergency service. Despite advanced cardiac life support, with tube ventilation, cardiac compressions, injections of adrenaline and amiodarone, external electric shocks, and attempt of lysis with recombinant tissue-type plasminogen activator (rt-PA; 100 mg), sustained spontaneous circulation could not be obtained. The patient was transported to our ICU under continuous CPR. At arrival, the ECG showed ventricular tachycardia/fibrillation, serum electrolytes were normal in the blood gas analysis with lactate measuring 7.7 mmol/L and pH 7.137 (please see [Table 1](#) for blood gas analysis and [Fig. 1A](#) for initial ECG). It was immediately decided to implant an extra-corporeal life support (ECLS) system (SCPC (Sorin); Hilite LT (Medos), initial flow 4.0L/min). Until now the patient was without return of spontaneous circulation (ROSC) for 140 min. The venous (Quickdraw (Edwards Lifesciences) (25 french)) and arterial ((Opti Site) (22 french) perfusion cannulae were implanted bed-site and the ECLS was connected within 20 min. In the consecutive performed coronary angiography a subtotal stenosis of the proximal LAD was seen and treated using a bare-metal-stent ([Fig. 1B](#)).

Removal of the ECLS support was performed following gradual reduction of the ECLS flow to 2 L/min at 0.5 L/day steps under echocardiography guidance. Mild doses of inotropic and phosphodiesterase inhibitor agents were initiated. ECLS removal was considered eight days after the event at central venous pressure

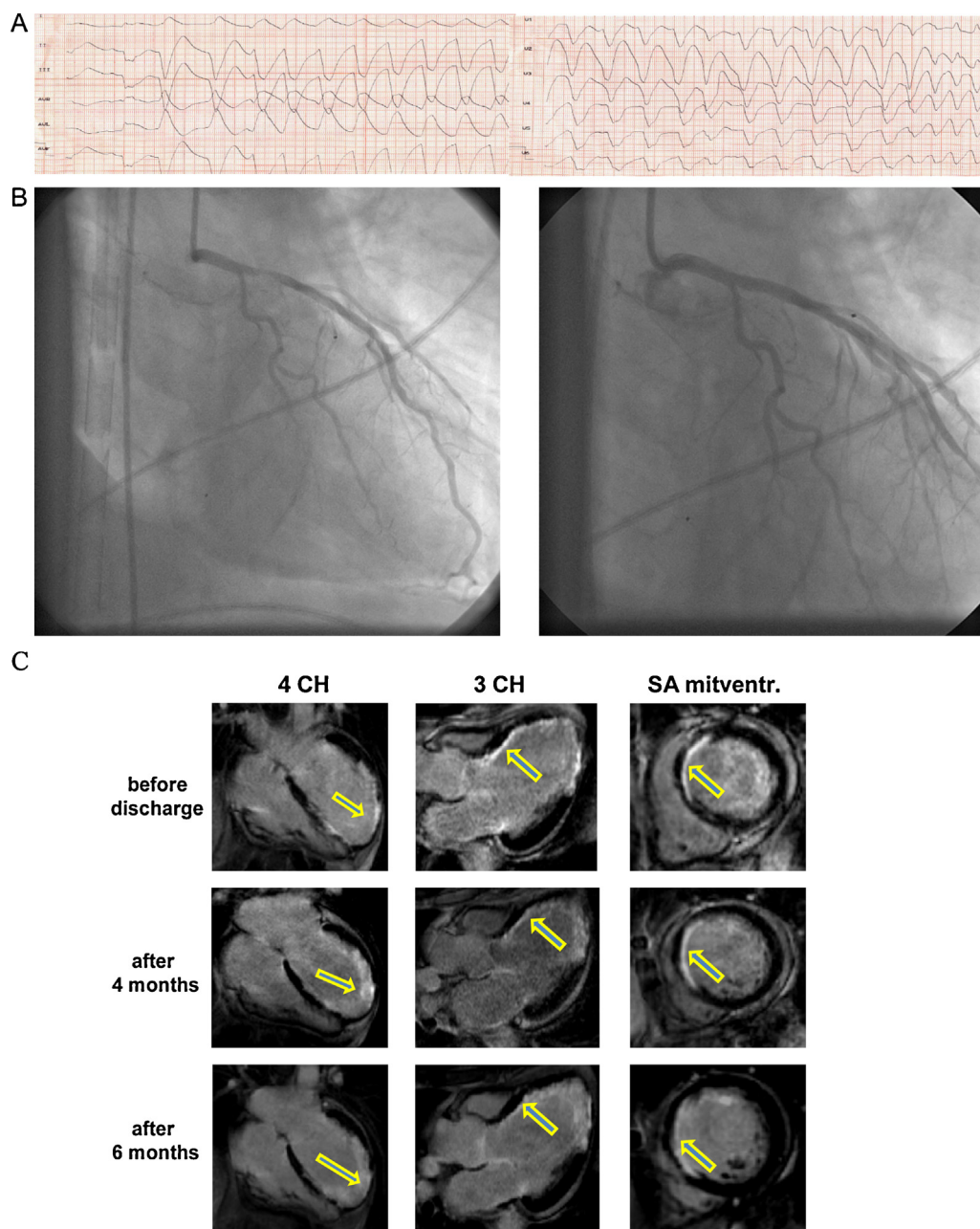
**Table 1**

Initial blood gas analysis on admission.

pO <sub>2</sub>	349 mm Hg
sO <sub>2</sub>	100%
pCO <sub>2</sub>	56.7 mm Hg
pH	7.137
HCO <sub>3</sub> <sup>-</sup>	15.7 mmol/L
BaseExc	-9.2 mmol/L
Anion gap	11.1 mmol/L
Na <sup>+</sup>	136 mmol/L
K <sup>+</sup>	3.9 mmol/L
Ca <sub>2</sub> <sup>+</sup>	1.22 mmol/L
Cl <sup>-</sup>	107 mmol/L
Lac	7.7 mmol/L
Glu	335 mg/dL
Hb	15.6 g/dL

<15 mm Hg, mean arterial pressure >60 mm Hg, and cardiac index remained >2.4 L/min/m<sup>2</sup>. The patient was discharged after 35 days without neurological deficits. Follow-up MRI exams showed a gradually improved ejection fraction up to 49% ([Fig. 1C](#)).

Despite medical advances, only 5–10% of patients survive after out-of-hospital cardiac arrest (OHCA).<sup>1</sup> Survival rates even decrease to around 2% when ROSC can not be achieved within 15 min.<sup>2</sup> Therefore, there is a strong medical need for alternative strategies. It is especially unclear how to treat younger patients with long periods of CPR after OHCA. In recent years technical innovations led to miniaturization of ECLS systems towards portable machines with the potential to be implanted under emergency situations bedside with limited delay. So-called “rapid-response ECLS” were associated with improved survival compared to conventional CPR in patients with in-hospital cardiac arrest.<sup>3</sup> This seems also be important for OHCA. Our case shows that we have to be open for novel approaches. Despite the potential cost and resource intensity of ECLS, younger patients with very long periods of cardiac arrest may benefit from such so far not standardized strategies. Future trials will have to identify which patients with OHCA may benefit mostly from “rapid-response ECLS”.



**Fig. 1.** (A) Initial ECG after hospital admission. (B) Coronary angiogram before (left) and after (after) percutaneous coronary intervention shows the culprit lesion in the proximal LCA. (C) MRI examination were performed before, and four and six months after discharge, respectively. Late gadolinium enhancement (LGE) delineated infarcted area.

#### Role of the funding source

TR is a Heisenberg professor funded by the German Research Foundation (DFG RA 969/7-2).

#### Conflict of interest statement

No conflicts of interest exist.

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18 December 2013