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Letter to the Editor

## ECMO therapy after thrombotic left main occlusion bridges prolonged cardiac arrest

*Sir,*

A 53 year old patient was admitted February, 8th to our hospital with ST-segment elevation myocardial infarction and severe cardiogenic shock. Because of cardiopulmonary instability, the patient was treated with catecholamins and was already intubated in the trauma room. He then underwent immediate coronary angiography and was treated with drug-eluting stenting because of a thrombotic occlusion of the left main coronary artery. After implantation of an intra-aortic balloon pump (IABP), the patient was transferred to our intensive care unite (ICU). Laboratory findings showed massive increase of cardiac enzymes (CK 14,465 U $l^{-1}$ ($\leq$190), CK-MB 1146 U $l^{-1}$ ($\leq$25) Troponin $I$ > 50.0 ng $ml^{-1}$ ($\leq$0.05)). Despite rapid and successful recanalization of the left main coronary artery and IABP support, the patient was on high-dose catecholamins (2.8 mg $h^{-1}$ at admission on ICU). At 8:00 p.m. the cardiopulmonary situation was complicated by ventricular fibrillation and cardiopulmonary rescuscitation (CPR) was needed for 5 min. In the course of the following hours, cardiac shock progressed very rapidly leading to application of excessing dosages of catecholamines (cumulative dosage of arterenol and epinephrine >5 mg $h^{-1}$). Thus, we decided to perform extra-corporal membrane oxygenation (ECMO) 24 h after stent-implantation. Shortly after initiation of ECMO therapy (blood flow 4.5 l $min^{-1}$), the heart showed electromechanical decoupling with no contractions at all. This condition lasted for approx. 24 h until spontaneous heart action returned. In the course of the following days, left ventricular ejection further improved to LVEF of 25–30% and cardiac output monitored by pulmonary artery catheter (PAC) increased from 1.2 l $min^{-1}$ up to 5.2 l $min^{-1}$, signalizing a now hyperdynamic circulatory state. This, togheter with increasing inflammatory markers announced an initiating catheter sepsis and we decided to change central venuous catheters and end ECMO therapy. Therefore, IABP was re-implantated and ECMO therapy could be successfully weaned under protection of IABP. On February, 20th IABP could be removed and in the course of the following days, catecholamins could be further reduced. On May, 7th the patient could be discharged from our hospital without significant neurological sequelae.

The usefulness of extra-corporal membrane oxygenation in cardiopulmonary rescuscitation, post-acute myocardial infarction complicated by cardiogenic shock and rapid recovery from cardiac arrest using ECMO have been shown previously.[1,2,3] Here we show, that even after prolonged cardiac arrest (>24 h) following acute myocardial infarction, ECMO therapy can lead to restitution of cardiac function. We therefore propose early application of ECMO therapy in progressive cardiac shock after stent-implantation for bridging of cardiac arrest.

### Conflict of interest statement

The authors have nothing to disclose.

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