Circulation Cardiovascular Case Series

Assisted Beating of the Ischemic Heart

How to Manage the Pulseless ST—Segment-Elevation Myocardial Infarction Patient

Christopher Lotz, MD; Oliver Ritter, MD, PhD; Ralf Michael Muellenbach, MD, PhD, MHBA

Forward

Information about a real patient is presented in stages (boldface type) to expert clinicians (Dr Oliver Ritter and Dr Ralf Michael Muellenbach), who respond to the information, sharing their reasoning with the reader (regular type). A discussion by the authors follows.

A 63-year-old white male was admitted with recurring chest pain of increasing intensity for ≈3 days. He had a known history of arterial hypertension and was obese, with a body mass index of 29.4 kg/m^2. After seeking help at his family physician he was immediately transferred to our hospital using emergency medical services because of ST-segment elevations. On arrival he had severe chest pain with dyspnea and a lowered blood pressure of 90/60 mm Hg. On auscultation moist rales could be heard over both lungs, whereas heart sounds were normal. The abdomen was nondistended with normal bowel sounds and no palpable organomegaly. His extremities were warm and exhibited peripheral edemas on both sides. The ECG displayed sinus tachycardia at 102/min with posteroinferior ST-segment elevations and reciprocal ST-segment depressions in leads I, aV_L, V_2 and V_3. Bedside echocardiographic assessment showed severely depressed biventricular function with a left ventricular ejection fraction of ≈15% and a dilated right ventricle measuring a tricuspid annular plane systolic excursion (TAPSE) of ≈1 cm. All heart valves were normal, and no signs of pericardial effusion could be observed.

Dr Ritter: The presentation of patients with severe chest pain and impaired hemodynamic parameters is always suggestive of myocardial infarction. However, intense pain accompanied by vegetative symptoms such as nausea, vomiting, dyspnea, or sweating should always prompt rapid diagnostic evaluation of an array of possible diagnoses. Life-threatening conditions include an acute myocardial infarction as well as aortic dissection, pulmonary embolism, Boerhaave syndrome, or pneumothorax. The first step of diagnostic evaluation is performing an ECG. It is important to emphasize that ST-elevations are not a must-see on the initial 12-lead ECG, especially in posterior myocardial infarction or total occlusion of the main left artery, whereas the presence of ST-elevation indicates a myocardial infarction in 80% to 90%. ST-elevation on the ECG has also been related to a high rate of early case fatality. The presence of type A aortic dissection cannot be excluded at this point, because it is able to mimic thromboembolic myocardial infarction via coronary ostium obstruction. Rapid transthoracic echocardiographic evaluation is the next diagnostic step. Echo may show regional dyskinesia and can simultaneously help to exclude aortic dissection, as well as provide information regarding pulmonary embolism.

Subsequent laboratory testing should include the assessment of cardiac serum biomarkers. In particular measurements of high-sensitive troponin enables the early detection of myocardial necrosis with high sensitivity if values exceed the 99th percentile decision limits. However, high-sensitive troponin is not entirely specific for myocardial infarction, and a long list of other causes may generate elevations of this cardiac biomarker. Serial testing is an option in stable patients, as a dynamic rise of high sensitive troponin is characteristic for acute myocardial damage, mostly caused by acute myocardial infarction.[1]

Once the diagnosis of an acute myocardial infarction is set, reperfusion therapy should be initiated as soon as possible with primary percutaneous coronary intervention (PCI) as the method of choice. All in all, door to balloon time should be <90 minutes. The current guidelines of the American Heart Association (AHA)/American College of Cardiology Foundation (ACCF) recommend reperfusion therapy for all eligible patients with ST—segment-elevation myocardial infarction (STEMI) with symptom onset within the previous 12 hours, as well as those with ongoing symptoms within the previous 12 to 24 hours. In the unstable patient with STEMI and cardiogenic shock or acute heart failure primary PCI should be performed irrespective of the time delay.[2]

As highlighted in the current case, the patients themselves often cause the biggest delay of treatment, as many of them do not realize the gravity of the situation because of gradual symptom onset. At time of admission, our patient already experienced

From the Department of Anesthesia and Critical Care (C.L., R.M.M.), Department of Internal Medicine I, Division of Cardiology (O.R.), and Deutsches Zentrum für Herzinsuffizienz (O.R.), University of Würzburg, Germany.

The online-only Data Supplement is available with this article at http://circ.ahajournals.org/lookup/suppl/doi:10.1161/CIRCULATIONAHA.114.009270/-/DC1.

Correspondence to Ralf Michael Muellenbach, MD, PhD, MHBA, Department of Anesthesia and Critical Care, University of Würzburg, Germany, Oberduerrbacher Str. 6 97080 Würzburg, BY, Germany. E-mail muellenbac_r@ukw.de

(*Circulation*. 2014;130:1095-1104.)

***Circulation* is available at http://circ.ahajournals.org** **DOI: 10.1161/CIRCULATIONAHA.114.009270**

cardiogenic shock with hypotension and peripheral and pulmonary edema. ECG and echocardiographic findings clearly pinpoint toward an acute STEMI infarction. Rapid actions are key, targeting urgent myocardial reperfusion above all.

Patient presentation (continued): **The patient's condition rapidly declined, resulting in respiratory insufficiency with an arterial partial pressure of oxygen (PaO_2) of 37 mm Hg and an arterial oxygen saturation (saO_2) of 77% while breathing supplemental oxygen. After endotracheal intubation cardiogenic shock further progressed, soon resulting in ventricular fibrillation, refractory to several defibrillation attempts. Cardiopulmonary resuscitation (CPR) was started using a LUCAS chest compression system (LUCAS, Physio-Control Inc, Lund, Sweden).**

Dr Ritter: Cardiogenic shock is a self-perpetuating process as the activation of neurohumoral systems and a systemic inflammatory response worsen the clinical picture. Secondary ventricular fibrillation is indicative of rapidly progressing damage and a risk factor for increased early mortality,[3] even in the modern PCI era.[4] The same applies to severely impaired left ventricular ejection as recently shown in the Harmonizing Outcomes With Revascularization and Stents in Acute Myocardial Infarction (HORIZONS-AMI) trial. Patients with left ventricular ejection fractions <40% had higher 1-year rates of major adverse cardiovascular and clinical events, noncoronary artery bypass graft major bleeding, and cardiac death.[5]

The necessary therapeutic actions are nearly self-explanatory and consist of CPR according to the current AHA/European Resuscitation Council (ERC) guidelines,[6,7] including ventricular defibrillation. At this juncture, one needs to particularly emphasize that high-quality CPR is essential to achieve a favorable outcome with subsequent revascularization. However, manual CPR is often performed at insufficient rates[8] and is tiring, when performed over longer periods of time.[9] Mechanical automated chest compression devices represent a promising alternative and have recently been shown to achieve higher mean regional cerebral oxygen saturation levels.[10] Different types of mechanical devices exist, working as pneumatic vests, load distributing bands, or pistons. The LUCAS chest compression system is a compressed air– or battery-driven piston working at a rate of 100 per minute with equal compression and decompression times. The system allows defibrillation without interruption, and its safe use has been reported during PCI percutaneous transluminal coronary angioplasty.[11] Experimental and clinical studies pinpoint toward improved outcomes after in-hospital-resuscitation[10,12,13]; nevertheless, current evidence is insufficient to allow any definite conclusion regarding the super- or inferiority of mechanical chest compressions compared to manual CPR.[14] Current AHA guidelines recommend the use of mechanical devices, either load distributing bands or pistons in specific settings with a class IIb, level of evidence (LOE) B or a class IIb, LOE C recommendation, respectively.[15]

We use mechanical automated chest compression in situations in which prolonged CPR must be expected and high-quality manual CPR may encounter difficulties. This includes the start of extracorporeal life support or further diagnostic evaluation and therapeutic intervention (ie, percutaneous transluminal coronary angioplasty). All of those were necessary in the current case.

Patient presentation (continued): **After 10 minutes CPR without a return of spontaneous circulation, the in-house extracorporeal cardiopulmonary resuscitation (E-CPR) team was alarmed.**

Dr Muellenbach: The success of CPR after in-hospital cardiac arrest is best when the event is witnessed or monitored and highly trained personnel are immediately at hand.[16] However, in the presence of cardiogenic shock and severely impaired left ventricular function, conventional measures often do not suffice. In such cases the early use of additional measures should be considered. E-CPR is defined as extracorporeal support initiated during conventional resuscitation or when repetitive cardiac arrest occurs without the return of spontaneous circulation for >20 minutes. The success rate of extracorporeal support depends on timing, whereas the greatest survival benefit could be observed when conventional CPR had been performed for <30 minutes.[17] The possibility of E-CPR should be integrated into the thought process early on, always having a door to balloon time of less than 90 minutes in mind. When in-house protocols are in place, the E-CPR team can get started in <10 minutes. However, when low- or no-flow times are minimized via high-quality CPR, patients may even benefit from extracorporeal life support after prolonged periods of time. Peripheral venoarterial (va)- extracorporeal membrane oxygenation (ECMO) is clearly the method of choice, because it is easily applicable at the bedside with procedural completion times of <30 to 45 minutes. In patients experiencing cardiogenic shock after an acute myocardial infarction va-ECMO represents an excellent means of tissue oxygenation and bridging toward early revascularization. Nevertheless, E-CPR is resource-intensive and the respective expertise as well as equipment needs to be installed. This includes the final decision making of whether or not to start extracorporeal life support based on clearly defined exclusion criteria. The Table depicts our in-house criteria on which the decision was based in the current case.

Patient presentation (continued): **On arrival, CPR was ongoing. The decision was made to start va-ECMO (Permanent Life Support Set and Rotaflow-console, Maquet GmbH, Rastatt, Germany), and cannulae were implanted via the right femoral vein and the left femoral artery, respectively. ECMO settings included a blood flow of 3 to 4 L/min and a sweep gas flow of 1 to 2 L O_2/min. The procedure was completed 35 minutes into the CPR, and hemodynamics immediately improved with mean arterial blood pressures ranging between 70 to 80 mm Hg.**

Dr Muellenbach: Va-ECMO is the prime mode of support in patients receiving active CPR, although improved myocardial function has also been described with venovenous cannulation in patients with combined lung and heart failure. As a general rule, venovenous cannulation primarily supports pulmonary gas exchange and depends on a sufficient function of the native heart. Va-ECMO is an incomplete cardiopulmonary bypass whereas blood that passes through the artificial

Table. In-House E-CPR Exclusion Criteria

Advanced age
Unobserved cardiac arrest with a delayed start of conventional CPR>5min
Prolonged multi organ failure
Long-term diabetic syndrome
End-stage renal failure
Severe brain damage
Liver cirrhosis CHILD B or C
End-stage heart failure
COPD GOLD IV
Life expectancy < 1 year
Living will

The respective leader of the extracorporeal membrane oxygenation team at the bedside makes the final decision regarding the start of extracorporeal membrane oxygenation therapy after a maximum of 20 min into conventional CPR. COPD indicates chronic obstructive pulmonary disorder; CHILD, Child-Pugh class; and E-CPR, extracorporeal cardiopulmonary resuscitation.

lung mixes with the blood ejected via the left ventricle into the aorta. A ratio of 80% to 20% is desired; generating blood flows approximately within the range of a normal cardiac index. The cannulae are easiest inserted percutaneously into the femoral vessels during ongoing CPR. If possible, insertion of the cannulae should be performed with ultrasound guidance. However, possible vascular complications of the femoral access need to always be taken into account including leg ischemia and bleeding. During cannulation, it is important to rigorously continue chest compression because low- or no-flow times must be minimized, as they are likely associated with a poor outcome. Once in place, venous blood is drained from the inferior vena cava and returned to the femoral artery with the chosen flow settings determining the perfusion toward the aortic root (Figure 1). Both oxygen delivery and CO_2 elimination can be fully maintained via the extracorporeal membrane lung. Improved hemodynamics result from a number of effects: (1) Flow and perfusion pressure are ameliorated per se, contributing to an improved coronary blood flow; (2) Venous drainage reduces right ventricular preload and subsequent left ventricular filling pressures assuming the absence of any large interventricular communication; and (3) The reduction of left ventricular end-diastolic pressure lowers cardiac oxygen demand and increases coronary blood flow, overall breaking a vicious cycle of self-perpetuating ischemic injury. As a caveat, va-ECMO inherently increases afterload and the reduction in left ventricular pressure is dependent on the generation of sufficient force by the native heart to open the aortic valve, allowing left ventricular decompression. Close monitoring of the saO_2 at the right hand, as well as frequent arterial blood gas analyses utilizing an arterial line placed into the right A. radialis helps to detect a so called "Harlequin syndrome." This denotes flow competition between the native heart and the ECMO circuit and subsequent deoxygenation of the upper part of the body in case of deteriorating pulmonary function (Figure 2). The right upper extremity is furthest away from the oxygenated blood supplied by the ECMO circuit, thus providing an indirect measure to evaluate cerebral oxygenation. ECMO blood flows can then be adjusted to guarantee sufficient oxygen supply to avoid hypoxic brain damage. Advanced monitoring of cerebral oxygenation via near-infrared spectroscopy may be an additional valuable tool, measuring regional oxygen saturation as an indicator of hemodynamic and metabolic alterations.[18]

All in all, the importance of E-CPR as a valuable bridging option of the unstable STEMI patient toward PCI or coronary artery bypass grafting (CABG) surgery cannot be overstated, just as the need of its early consideration within the therapeutic process. Beneficial effects of ECMO therapy may not even be limited to the pulseless patient, as its implementation in the catheter laboratory during cardiogenic shock without the particular need for CPR leads to a reduced 30-day mortality.[19]

Patient presentation (continued): **Defibrillation and amiodarone administration subsequently terminated ventricular fibrillation. However, after discontinuation of chest compressions no signs of sufficient left ventricular**

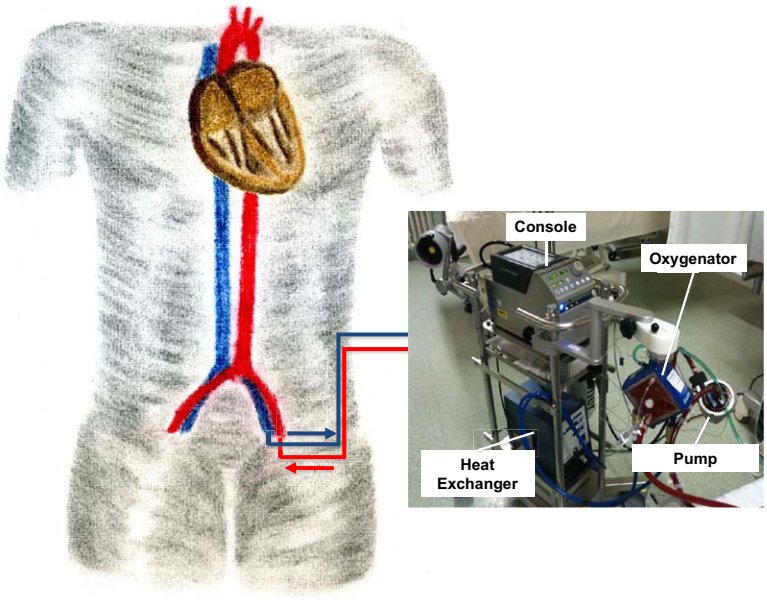

Figure 1. Peripheral venoarterial (va)-ECMO therapy breaks a vicious cycle of self-perpetuating myocardial ischemic injury. The extracorporeal membrane oxygenation supports the failing heart by draining blood from the inferior vena cava and returning oxygenated blood via the femoral artery (coronary perfusion). Beneficial effects unloading the heart include a reduced right ventricular preload, subsequently decreasing left ventricular filling and end-diastolic pressure, allowing reduced myocardial oxygen consumption as well as an increased coronary blood flow. ECMO indicates extracorporeal membrane oxygenation.

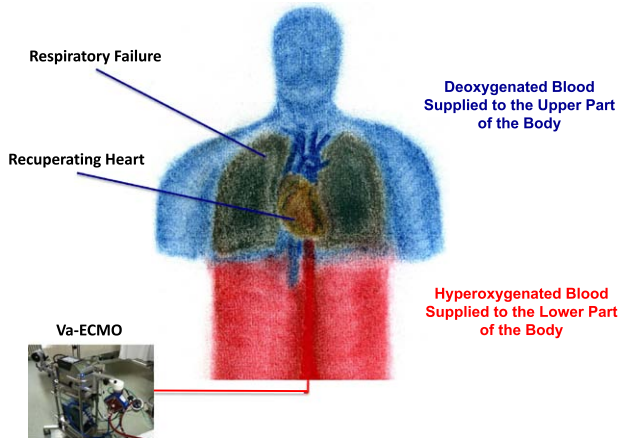

Figure 2. In case of respiratory failure, flow competition in the aorta between the recovering native heart and the extracorporeal circuit can lead to a "Harlequin" or "North–South" syndrome. Deoxygenated blood is supplied to the upper part of the body, whereas only the lower part benefits from the hyperoxygenated blood supplied by the venoarterial (va)-ECMO. The solution to this serious problem is additional venovenous (vv)-ECMO support (eg, via cannulation of the internal jugular vein). ECMO indicates extracorporeal membrane oxygenation.

ejection could be observed. The ECG showed organized pulseless electric activity. Administration of inotropes did not improve ventricular function, prompting the immediate resumption of chest compressions via the LUCAS system.

Dr Muellenbach: The va-ECMO generates a nonpulsatile blood flow, maintaining sufficient blood pressures and perfusion of all vital organs over long periods of time. As such the absence of a pulse wave would not be alarming per se. However, because the native heart contributes to the overall flow a pulse is normally perceptible. As depicted in the current case, its absence subsequent to the termination of ventricular fibrillation is equivalent to secondary pulseless electric activity during conventional CPR. Although the installed va-ECMO provides sufficient systemic oxygen delivery, the native heart still needs to generate enough force to open the aortic valve to avoid left ventricular overdistension via continuous filling through bronchial and thebesian veins, as well as an increased afterload proportional to va-ECMO flow. Movie I in the online-only Data Supplement depicts the echocardiographic presentation of this dilemma. Venting of the left ventricle is mandatory at this point. If one neglects to do so, a vicious cycle of electromechanical dissociation develops, leading to obstruction of coronary blood flow, pulmonary edema, hemorrhage, and even intracavity clot formation. Understanding the pathophysiology of this dilemma is one of the pivotal aspects of the current case, because the erroneous assumption that nonpulsatile flow alone suffices will lead to the death of the patient. An additional caveat is that with peripheral va-ECMO the oxygenation of the head is also determined via left ventricular outflow.

Venting options start by transiently reducing va-ECMO flow to differentiate hypokinesia from akinesia of the left heart. However, va-ECMO flow is not only directly correlated with increases in left ventricular afterload, but also inversely correlated with cardiac preload. This means that by decreasing

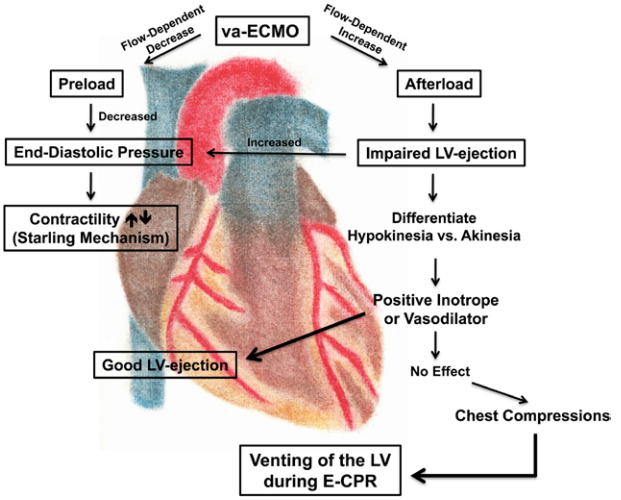

Figure 3. Circulatory changes associated with venoarterial (va)-ECMO therapy during cardiogenic shock requiring immediate venting of the left ventricle. Sophisticated pathophysiological mechanisms including a ECMO blood flow–dependent decrease in cardiac preload accompanied by a corresponding increase in afterload demand a higher contractile force from the left ventricle (LV) to open the aortic valve. If the failing LV is unable to exert sufficient pressure obstruction of coronary blood flow, pulmonary edema, hemorrhage, and even intracavity clot formation develops. Reducing va-ECMO flow may alleviate this problem and helps to distinguish left ventricular hypokinesia vs akinesia. However, because decreased afterload is accompanied by enhanced preload during va-ECMO, increased left ventricular filling pressures may worsen the situation in the failing heart via the Starling law. Inotropic or vasodilator support represents the next therapeutic step, respectively. Nevertheless if none of these measures suffice, immediate venting of the left ventricle is mandatory. At the bedside, this can be easiest accomplished via the resumption of chest compressions. ECMO indicates extracorporeal membrane oxygenation; and E-CPR, extracorporeal cardiopulmonary resuscitation.

va-ECMO flow, end-diastolic volume will be enhanced, thus ventricular ejection change dependent on the contractility of the left ventricle (via the Starling law). The use of a positive inotropic or vasodilator (reducing myocardial afterload) represent other viable choices to support left ventricular contraction at this juncture, because systemic vascular resistance remains independent of ECMO blood flow. We chose to administer epinephrine to enhance myocardial contractility, however, without effect, making mechanical venting of the left ventricle imperative as the next step within the cascade. Invasive measures to vent the left ventricle include the insertion of an additional cannula into the left atrium or atrial septostomy,[20] as well as the installation of a catheter based microaxial pump directly unloading the left ventricle (eg, the Impella). However, in case of planned emergency PCI the only suitable maneuver for continuous ventricular unloading at the bedside is the resumption of chest compressions (Figure 3). These were immediately resumed and maintained while referring the patient to the catheter laboratory.

***Patient presentation (continued)*: The patient was transferred to PCI via the right radial artery under continued va-ECMO support. Chest compressions were shortly paused during the percutaneous transluminal coronary angioplasty to facilitate the intervention, revealing the return of pulsatile blood flow. The right coronary artery was**

identified as the culprit vessel via the ECG. Angiography actually revealed a complete occlusion of the vessel and reperfusion was subsequently ensured via the placement of a bare metal stent. Subsequent diagnostic evaluation of the left coronary artery identified an 80% stenosis of the left main coronary artery, a complete occlusion of the left anterior descending artery, as well as a 60% stenosis of the proximal left circumflex coronary artery. After interventional reperfusion the patient was subjected to CABG surgery. Successful surgical revascularization consisted of grafts from the left internal thoracic artery onto the left anterior descending artery, venous grafts onto the ramus marginalis, as well as the ramus intermedius. After surgery the patient was transferred to the intensive care unit with an over time decreasing need for vasopressor support.

Dr Ritter: Timely myocardial reperfusion represents the hallmark of acute myocardial infarction therapy. Immediate coronary angiography is mandatory in STEMI, whereas the subsequent intervention depends on the diagnostic findings with either PCI or CABG surgery as viable options. As a primary goal, emergency PCI aims to identify and stent the infarct-causing lesion. However, in the presence of cardiogenic shock the intervention must not be limited to the infarct vessel, but also focus on maximizing coronary blood flow to improve hemodynamic stability. In case of a significant left main coronary artery stenosis, as well as significant stenoses in 3 major coronary arteries or in the proximal left anterior descending artery plus 1 other major coronary artery, CABG is the method of choice according to the current guidelines.[21] We performed a PCI and bare metal stent implantation of the right coronary artery to immediately restore perfusion of the posterior wall. Further diagnostic angiography revealed advanced coronary artery disease with a significant stenosis of the left main coronary artery, total occlusion of the left anterior descending artery, as well as a significant stenosis of the left circumflex coronary artery. Unfortunately hemodynamics did not improve as desired after PCI of the right coronary artery. Therefore it was decided to complete revascularization by subjecting the patient to CABG surgery. Multiple PCIs before CABG have been shown increase in-hospital mortality as well as the incidence of major adverse cardiac events in the perioperative period[22]; nevertheless, the rationale in the current patient was based on an individual risk–benefit analysis considering the favorable effect of immediate reperfusion of the infarct-determining vessel with an anyway high surgical risk. The synopses of complex lesions with a Synergy between PCI and Cardiac Surgery (SYNTAX) score of 43 and a mortality risk of 24.3% with a combined risk of morbidity or mortality of 70.6% as calculated by the Society of Thoracic Surgeons risk score, respectively, offers no clear-cut best practice. Current evidence suggests that 1- to 5-year mortality is similar after CABG surgery or PCI in patients with left main disease or 3-vessel disease solely showing higher repeat revascularization rates with PCI while offering a lower incidence of stroke. The optimal reperfusion strategy in severe cardiogenic shock remains basically unknown and CABG surgery is only favored to improve long-term outcomes with SYNTAX scores of >22 in the absence of a high risk of surgery, defined as a risk score of <5%.[23,24]

In patients without hemodynamic compromise the decision between culprit-only and multivessel revascularization is even more challenging. The 2011 ACCF/AHA/Society for Cardiovascular Angiography and Interventions (SCAI) guideline for PCI recommends a primary culprit lesion angioplasty,[25] with clinical practice often differing from these recommendations. Multivessel disease per se leads to a higher mortality,[26] and failed cardiac improvement often forces the attending physician to proceed with a multivessel procedure. The HORIZONS-AMI showed a quadrupled 1-year mortality when multivessel PCI was the primary intervention; nonetheless, 18.5% of the patients received multivessel PCI with only 1.5% experiencing cardiogenic shock.[27] The Assessment of Pexelizumab in Acute Myocardial Infarction (APEX-AMI) trial indicated an increased 90-day mortality when nonculprit interventions were performed at the time of primary PCI,[28] and Hannan et al further corroborated the current guideline recommendations via lower in-hospital mortality. However, staged multivessel PCI within 60 days after the initial intervention led to a significantly reduced 1-year mortality,[29] and a more aggressive interventional strategy immediately targeting all angiographic lesions has been advocated by the Preventative Angioplasty in Myocardial Infarction (PRAMI) trial.[30] The investigators showed that patients with a STEMI and concomitant multivessel disease clearly benefited from preventive PCI via a reduced rate of cardiac death, nonfatal myocardial infarction, or refractory angina. However, it is important to emphasize that no comparison between immediate versus staged multivessel PCI was conducted. Hence, although the optimal timing remains to be determined in the absence of cardiogenic shock, it has certainly become clear that all diseased vessels need to be addressed after a STEMI. To evaluate the best revascularization strategy for patients in cardiogenic shock the European multicenter CULPRIT SHOCK trial is currently recruiting patients (Culprit Lesion Only PCI Versus Multivessel PCI in Cardiogenic Shock, ClinicalTrials.gov Identifier: NCT01927549).

Dr Muellenbach: Fortunately, contractile function recovered allowing the heart to create sufficient force to open the aortic valve. Postoperatively, we refrained from implanting a left ventricular assist device, because left ventricular ejection fraction was improving with an ejection fraction of ≈20%.

Continued va-ECMO support follows successful completion of myocardial revascularization, ideally as a bridge to recovery. In this context it always needs to be considered that although indispensible for survival, myocardial reperfusion injury contributes up to 60% to the overall myocardial damage.[31] This often necessitates the need for prolonged extracorporeal support. Weaning from the extracorporeal support device is initiated according to each individual's progress in ventricular recuperation. The process is guided by daily echocardiographic monitoring of left and right ventricular function, optimized volume management, as well as maximized inotropic support. ECMO flows are gradually reduced and decannulation may follow as the functionality of the native heart permits hemodynamic stability (Figure 4). It is important to recognize that in the presence of pulmonary edema, arterial oxygenation may transiently worsen with improving cardiac contractility as a result of increased blood flow through the native lung compared with the va-ECMO circuit. Moreover, a short-term increase in vasopressor support is not uncommon after successful va-ECMO

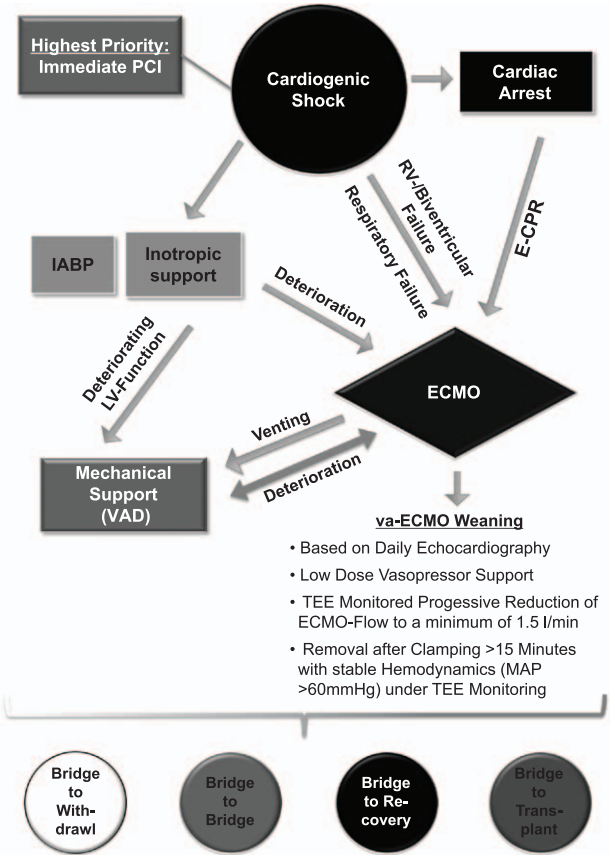

Figure 4. Support to the failing heart during cardiogenic shock must be considered early on to facilitate immediate revascularization as the number 1 priority. In this regard, extracorporeal cardiopulmonary resuscitation (E-CPR) represents the primary therapeutic option during cardiogenic shock requiring CPR. ECMO must also be considered early on during all cases refractory to inotropic support, in particular when right- or biventricular, as well as respiratory failure is impending. The extracorporeal circuit can be easily combined with other forms of left ventricular support, such as IABP, an Impella pump, or a Tandem Heart. The latter are prime choices in case of isolated left ventricular failure. All serve as bridges toward full recovery or definite therapeutic options depending on neurological and cardiac function. ECMO indicates extracorporeal membrane oxygenation; IABP, intra-aortic balloon pump; LV, left ventricle; and VAD, ventricular assist device.

decannulation. It is further important to emphasize that weaning from the ventilator is independent of va-ECMO therapy and may be completed before ECMO decannulation, as well as the initiation of additional postresuscitation measures. For the last decade postresuscitation care relied on therapeutic hypothermia as a proven strategy to reduce neurological damage in comatose patients. The 2010 AHA guidelines recommend the use of 32 to 34°C body temperature in comatose patients for 12 to 24 hours after out-of-hospital cardiac arrest and return of spontaneous circulation if the presenting rhythm was ventricular fibrillation (Class I, LOE B). It is further stated that the protocol may also be considered for comatose adult patients with return of spontaneous circulation after in-hospital cardiac arrest of any initial rhythm or after out-of-hospital cardiac arrest with an initial rhythm of pulseless electric activity or asystole (Class IIb, LOE B). Therapeutic hypothermia should be initiated as soon as possible,[32] using surface or endovascular cooling while slow rewarming with 0.25°C per hour is advised.[6] European guidelines state that therapeutic hypothermia should include comatose survivors of cardiac arrest associated initially with shockable and nonshockable rhythms.[33] The recommendations are largely based on 2 randomized studies from 2002, both providing valuable information only if ventricular fibrillation was the primary cause of cardiac arrest. The study by the Hypothermia After Cardiac Arrest Study Group is further limited by a prolonged interval of 5 to 15 minutes until the start of CPR and enrollment of just 8% of all patients assessed for eligibility.[34] Bernard et al[35] on the other hand excluded patients with cardiogenic shock subsequent to the return of spontaneous circulation. A recent trial including shockable and nonshockable rhythms could not find a significant difference when comparing target temperatures of 33°C and 36°C.[36] The conflicting results may be explained by a number of reasons, including specific patient subgroups, issues related to optimal timing and duration, as well as the best target temperature.

Nevertheless, in patients with an initial nonshockable rhythm a case-by-case decision should be made acknowledging the individual pros and cons. Although concurrent PCI and hypothermia seem to be safe, potential complications include coagulopathy, arrhythmias, and hyperglycemia. Moreover, immune function is compromised. The intervention is contraindicated in the presence of severe hemorrhage, intracranial hemorrhage, septic shock, pregnancy, and hypotension refractory to multiple vasopressors.[37] In our case we refrained from utilizing therapeutic hypothermia before CABG surgery because of an increased risk of bleeding, as well as the continued need of high-dose vasopressor support. Further, considering a 20% increase in mortality for every hour cooling is delayed, no benefit could be expected after completion of the surgical procedure.

In case of long-term refractory cardiogenic shock, para- or intracorporeal mechanical assist device techniques have a role within the treatment paradigm, as reflected in the current guidelines of the AHA/ACC with a class IIB/LOE C indication.[21] Va-ECMO support then acts as a bridge toward the implantation of the respective ventricular assist device or artificial heart. The mechanical ventricular assist device is subsequently used as a long-term solution (bridge to destination) or followed by cardiac transplant surgery (bridge to transplant). Naturally, unfortunate criteria of va-ECMO discontinuation also need to be considered, such as multiorgan failure, as well as the absence of cardiac recovery with concomitant contraindications for the implantation of a ventricular assist device.

Discussion

The pulseless STEMI patient represents a daily clinical challenge at the crossroads of life and death. The incidence of cardiogenic shock complicating an acute myocardial infarction is ≈5% to 15%[38] with atrocious mortality rates of ≈40%.[39,40] Usually the full clinical picture develops within the first few hours with a massive myocardial infarct size and consecutive ventricular dysfunction perpetuating themselves via the maladaptive activation of the sympathetic and renin-angiotensin systems. Both systolic and diastolic failure develops, leading to tissue hypoperfusion and pulmonary fluid retention. The primary therapeutic goal is to guarantee oxygen supply to all vital organs, whereas inotropic and vasopressor support results

in increased myocardial afterload and tachycardia. A vicious cycle of increased myocardial oxygen demand and decreased coronary perfusion pressure develops with malignant cardiac arrhythmias as the subsequent step within the shock paradigm. Nevertheless, the inevitability of 2-edged therapeutic options is 1 of the pivotal dilemmas of acute myocardial infarction therapy and includes causal therapy via revascularization and subsequent myocardial reperfusion injury.[31]

Ventricular fibrillation is still regarded as the number 1 lethal arrhythmia, although recent evidence has shown a rise in pulseless electric activity.[41] The outcome of in-hospital cardiac arrest remains poor, whereas ventricular fibrillation and secondary pulseless electric activity need to be interpreted as consecutive steps within the dying process. Post-defibrillation pulseless electric activity reflects massive myocardial injury and is seldom associated with residual left ventricular contraction. Irrespective of the type of arrhythmia, high-quality conventional CPR must immediately limit low- or no-flow times. Unfortunately, the achieved blood flow during conventional CPR is mostly insufficient.[42] Mechanical devices such as the LUCAS promise an improved quality of chest compressions, as optimal frequency, depth, and decompression via the integrated suction cup suggest a simplified practice. Laboratory studies on pigs showed a significantly higher cardiac output, carotid artery blood flow, and coronary perfusion, as well as improved cerebral blood flow when using the device.[13] The incidence of CPR-related injuries does not seem to differ between manual and mechanical compressions,[43] nevertheless, efficiency may vary between in-hospital versus out-of-hospital use. The LINC-Trial comparing the outcome of out-of-hospital cardiac arrest did not find a significant difference in 4-hour survival between patients treated with a mechanical device or those with manual CPR.[44] The in-hospital setting offers different conditions, in particular if diagnostic or interventional procedures are planned. High-quality manual CPR in the catheter laboratory is very difficult and mechanical help eases and speeds up the revascularization procedure.[43] However, careful inspection of device efficiency must be guaranteed on a case-by-case basis, as insufficient chest compressions due to inadequacy of the preset depth have been reported.[45]

Regardless of whether manual or mechanical versions are used, novel concepts are required to improve current CPR practice.[46] With standard CPR as a poor bridge toward reperfusion therapy va-ECMO is rapidly emerging as a lifesaving option providing hemodynamic support. Chen et al[47] impressively demonstrated a survival benefit in patients with witnessed in-hospital cardiac arrest. The prospective observational study included only patients with a cardiac cause and compared the use of extracorporeal life-support with conventional CPR with duration of >10 minutes. Both short-term as well as long-term survival favored E-CPR. These data were corroborated by a recent retrospective analysis, whereas E-CPR granted a significantly higher 6-month survival rate with minimized neurological impairment.[48] The procedure is particularly effective with cardiac causes of arrest, in younger patients, and if rapid implementation after the start of conventional CPR is warranted.[17] However, although a great therapeutic option, it is accompanied by profound circulatory changes and a thorough understanding of the pathophysiology and potential pitfalls is indispensible. This is highlighted in the current case, illustrating the importance of left ventricular venting during refractory cardiogenic shock. Nonpulsatile va-ECMO flow generates stable systemic hemodynamics, a benefit nevertheless accompanied by a flow-dependent increase in left ventricular afterload. Although negligible during normal cardiac function, this may be the tip of the iceberg in the failing heart preventing the opening of the aortic valve. Moreover, although the concomitantly reduced preload helps to decrease myocardial oxygen demand by alleviating end-diastolic pressure and end-diastolic volume, myocardial contractility may be further impaired according to the Starling law.[49] If these highly dynamic mechanisms are missed, the immediate death of the patient is imminent as a result of the obstruction of coronary blood flow, pulmonary edema, and finally intracavity clot formation. Left ventricular venting strategies include surgical decompression via transseptal puncture and placement of a drain,[50] as well as percutaneous insertion of a rotary blood pump[51–53] or placement of a venous cannula into the pulmonary artery.[54] Obviously none of these techniques are expeditiously applicable at the bedside during ongoing E-CPR with indicated early PCI and the goal to minimize door to balloon times. Hence, the fastest and easiest venting option is the immediate resumption of chest compressions until a definite method can be subsequently installed; always assuming that a concomitant return of pulsatile flow is still missing.

In our case myocardial recovery allowed revascularization without additional left ventricular support or venting. However, at this juncture the implantation of a percutaneous left ventricular assist device is an excellent option if either or both are required. The Impella or the TandemHeart both offer additional short-term support via left ventricular volume unloading again as bridges to recovery, the implantation of a long-term left ventricular assist device, device or cardiac transplant. The Impella pump is a microaxial rotary pump inserted into the left ventricle via the aortic valve offering hemodynamic support with blood flows of up to 5 l/min (Impella 5.0). Ventricular unloading is achieved, subsequently decreasing left ventricular wall stress and reducing myocardial oxygen consumption. The Prospective Feasibility Trial Investigating the Use of IMPELLA RECOVER LP 2.5 System in Patients Undergoing Huigh Risk PCI (PROTECT I) trial evaluating the safety and feasibility of the Impella 2.5 system during high-risk PCI concluded that the system is safe, easy to implant, and provides excellent periprocedural hemodynamic support in patients with poor left ventricular function.[55] Comparison of prophylactic Impella or intra-aortic balloon pump (IABP) use in the Prospective, Multi-center, Randomized Controlled Trail of the IMPELLA RECOVER LP 2.5 System Versus Intra Aortic Balloon Pump (IABP) in Patients Undergoing Non Emergent High Risk PCI (PROTECT II) trial indicated superior hemodynamic support via the Impella without increasing the incidence of major adverse events.[56] However, patients with a recent STEMI or cardiogenic shock were excluded from the PROTECT trials, and major studies in hemodynamic unstable patients experiencing a STEMI (Impella versus IABP reduces infarct size in STEMI patients treated with primary PCI (IMPRESS), Trial Using Impella LP 2.5 System in Patients With Acute Myocardial Infarction Induced Hemodynamic Instability (RECOVER II)) were stopped

because of slow patient inclusion. Moreover, comparisons with IABP therapy are a limited proof of its effectiveness, as benefits of the balloon pump have been doubted.[40,57] Thiele et al[40] showed that IABP use did not improve 30-day survival after an acute myocardial infarction with early revascularization, casting doubt on its usefulness during cardiogenic shock, although recommended in the current guidelines. The respective 6- and 12-month follow-up further corroborated this result.[57] However, concomitant IABP and ECMO therapy post CABG has been suggested to improve coronary graft flows by Madershahian et al.[58] The TandemHeart is a left ventricular assist device functioning as a centrifugal pump. Cardiac output is augmented and the left ventricle decompressed by aspirating blood from the left atrium and returning into the femoral arteries. Blood flow reaches up to 5.0 L of oxygenated blood per minute. Compared with IABP, the TandemHeart achieved significantly greater increases in cardiac index and mean arterial blood pressure.[59] Currently ongoing is The TandemHeart to Reduce Infarct Size (TRIS) Trial, evaluating a reduction in myocardial infarct size in comparison with conventional therapy along with PCI. Nevertheless, a major disadvantage of the device is that placement requires transseptal puncture, a maneuver nearly impossible during ongoing resuscitation.

In summary, both the Impella and the TandemHeart are valuable choices of short-term mechanical support during isolated left ventricular failure. Right ventricular function and pulmonary oxygenation are not improved, requiring ECMO therapy. The combined use of va-ECMO and Impella is an intriguing option, because the devices cover one another's weaknesses and the Impella pump has been successfully used for left ventricular decompression during va-ECMO.[51,52] Figure 4 provides an overview of the approach to ventricular support during cardiogenic shock requiring high-risk PCI.

Conclusion

In summary, the current case highlights the management of a pulseless STEMI patient using E-CPR as means of tissue oxygenation and bridging toward early revascularization. The procedure is ideally integrated in a step-wise approach, whereas it needs to be considered early on during cardiac arrest to achieve optimal results. The case further illustrates the importance to carefully scrutinize any therapeutic success. Stable hemodynamics after the initiation of va-ECMO therapy can be deceiving as a vicious cycle of increased afterload and further obstruction of coronary blood flow, pulmonary edema, and even intracavity clot formation develops if the native heart is unable to open the aortic valve. Venting of the failing left ventricle must be ensured with the fastest and easiest option being the resumption of chest compressions during CPR. Additional support may be provided further down the cascade via a left ventricular assist device, overall rigorously pursuing the goal of early revascularization via PCI or CABG surgery.

Disclosures

None.

References

1. Mahajan VS, Jarolim P. How to interpret elevated cardiac troponin levels. *Circulation*. 2011;124:2350–2354.
2. O'Gara PT, Kushner FG, Ascheim DD, Casey DE Jr, Chung MK, de Lemos JA, Ettinger SM, Fang JC, Fesmire FM, Franklin BA, Granger CB, Krumholz HM, Linderbaum JA, Morrow DA, Newby LK, Ornato JP, Ou N, Radford MJ, Tamis-Holland JE, Tommaso CL, Tracy CM, Woo YJ, Zhao DX, Anderson JL, Jacobs AK, Halperin JL, Albert NM, Brindis RG, Creager MA, DeMets D, Guyton RA, Hochman JS, Kovacs RJ, Kushner FG, Ohman EM, Stevenson WG, Yancy CW; American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2013;127:e362–e425.
3. Volpi A, Cavalli A, Santoro E, Tognoni G. Incidence and prognosis of secondary ventricular fibrillation in acute myocardial infarction. Evidence for a protective effect of thrombolytic therapy. GISSI Investigators. *Circulation*. 1990;82:1279–1288.
4. Mehta RH, Starr AZ, Lopes RD, Hochman JS, Widimsky P, Pieper KS, Armstrong PW, Granger CB; APEX AMI Investigators. Incidence of and outcomes associated with ventricular tachycardia or fibrillation in patients undergoing primary percutaneous coronary intervention. *JAMA*. 2009;301:1779–1789.
5. Ng VG, Lansky AJ, Meller S, Witzenbichler B, Guagliumi G, Peruga JZ, Brodie B, Shah R, Mehran R, Stone GW. The prognostic importance of left ventricular function in patients with ST-segment elevation myocardial infarction: the HORIZONS-AMI trial. *Eur Heart J Acute Cardiovasc Care*. 2014;3:67–77.
6. Bossaert L, O'Connor RE, Arntz HR, Brooks SC, Diercks D, Feitosa-Filho G, Nolan JP, Hoek TL, Walters DL, Wong A, Welsford M, Woolfrey K; Acute Coronary Syndrome Chapter Collaborators. Part 9: Acute coronary syndromes: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. *Resuscitation*. 2010;81 Suppl 1:e175–e212.
7. Hazinski MF, Nolan JP, Billi JE, Böttiger BW, Bossaert L, de Caen AR, Deakin CD, Drajer S, Eigel B, Hickey RW, Jacobs I, Kleinman ME, Kloeck W, Koster RW, Lim SH, Mancini ME, Montgomery WH, Morley PT, Morrison LJ, Nadkarni VM, O'Connor RE, Okada K, Perlman JM, Sayre MR, Shuster M, Soar J, Sunde K, Travers AH, Wyllie J, Zideman D. Part 1: Executive summary: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Circulation*. 2010;122(16 Suppl 2):S250–S275.
8. Abella BS, Sandbo N, Vassilatos P, Alvarado JP, O'Hearn N, Wigder HN, Hoffman P, Tynus K, Vanden Hoek TL, Becker LB. Chest compression rates during cardiopulmonary resuscitation are suboptimal: a prospective study during in-hospital cardiac arrest. *Circulation*. 2005;111:428–434.
9. Yang Z, Li H, Yu T, Chen C, Xu J, Chu Y, Zhou T, Jiang L, Huang Z. Quality of chest compressions during compression-only CPR: a comparative analysis following the 2005 and 2010 American Heart Association guidelines. *Am J Emerg Med*. 2014;32:50–54.
10. Parnia S, Nasir A, Ahn A, Malik H, Yang J, Zhu J, Dorazi F, Richman P. A feasibility study of cerebral oximetry during in-hospital mechanical and manual cardiopulmonary resuscitation*. *Crit Care Med*. 2014;42:930–933.
11. Larsen AI, Hjørnevik AS, Ellingsen CL, Nilsen DW. Cardiac arrest with continuous mechanical chest compression during percutaneous coronary intervention. A report on the use of the LUCAS device. *Resuscitation*. 2007;75:454–459.
12. Bonnemeier H, Simonis G, Olivecrona G, Weidtmann B, Götberg M, Weitz G, Gerling I, Strasser R, Frey N. Continuous mechanical chest compression during in-hospital cardiopulmonary resuscitation of patients with pulseless electrical activity. *Resuscitation*. 2011;82:155–159.
13. Rubertsson S, Karlsten R. Increased cortical cerebral blood flow with LUCAS; a new device for mechanical chest compressions compared to standard external compressions during experimental cardiopulmonary resuscitation. *Resuscitation*. 2005;65:357–363.
14. Brooks SC, Bigham BL, Morrison LJ. Mechanical versus manual chest compressions for cardiac arrest. *Cochrane Database Syst Rev*. 2011:CD007260.
15. Cave DM, Gazmuri RJ, Otto CW, Nadkarni VM, Cheng A, Brooks SC, Daya M, Sutton RM, Branson R, Hazinski MF. Part 7: CPR techniques and devices: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2010;122(18 Suppl 3):S720–S728.
16. Brady WJ, Gurka KK, Mehring B, Peberdy MA, O'Connor RE; American Heart Association's Get with the Guidelines (formerly, NRCPR)

Investigators. In-hospital cardiac arrest: impact of monitoring and witnessed event on patient survival and neurologic status at hospital discharge. *Resuscitation*. 2011;82:845–852.
17. Cardarelli MG, Young AJ, Griffith B. Use of extracorporeal membrane oxygenation for adults in cardiac arrest (E-CPR): a meta-analysis of observational studies. *ASAIO J*. 2009;55:581–586.
18. Wong JK, Smith TN, Pitcher HT, Hirose H, Cavarocchi NC. Cerebral and lower limb near-infrared spectroscopy in adults on extracorporeal membrane oxygenation. *Artif Organs*. 2012;36:659–667.
19. Sheu JJ, Tsai TH, Lee FY, Fang HY, Sun CK, Leu S, Yang CH, Chen SM, Hang CL, Hsieh YK, Chen CJ, Wu CJ, Yip HK. Early extracorporeal membrane oxygenator-assisted primary percutaneous coronary intervention improved 30-day clinical outcomes in patients with ST-segment elevation myocardial infarction complicated with profound cardiogenic shock. *Crit Care Med*. 2010;38:1810–1817.
20. del Nido PJ, Armitage JM, Fricker FJ, Shaver M, Cipriani L, Dayal G, Park SC, Siewers RD. Extracorporeal membrane oxygenation support as a bridge to pediatric heart transplantation. *Circulation*. 1994;90(5 Pt 2):II66–II69.
21. Levine GN, Bates ER, Blankenship JC, Bailey SR, Bittl JA, Cercek B, Chambers CE, Ellis SG, Guyton RA, Hollenberg SM, Khot UN, Lange RA, Mauri L, Mehran R, Moussa ID, Mukherjee D, Nallamothu BK, Ting HH. 2011 ACCF/AHA/SCAI Guideline for Percutaneous Coronary Intervention: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines and the Society for Cardiovascular Angiography and Interventions. *Circulation*. 2011;124:e574–e651.
22. Thielmann M, Leyh R, Massoudy P, Neuhäuser M, Aleksic I, Kamler M, Herold U, Piotrowski J, Jakob H. Prognostic significance of multiple previous percutaneous coronary interventions in patients undergoing elective coronary artery bypass surgery. *Circulation*. 2006;114(1 Suppl):I441–I447.
23. Deb S, Wijeysundera HC, Ko DT, Tsubota H, Hill S, Fremes SE. Coronary artery bypass graft surgery vs percutaneous interventions in coronary revascularization: a systematic review. *JAMA*. 2013;310:2086–2095.
24. Serruys PW, Morice MC, Kappetein AP, Colombo A, Holmes DR, Mack MJ, Ståhle E, Feldman TE, van den Brand M, Bass EJ, Van Dyck N, Leadley K, Dawkins KD, Mohr FW; SYNTAX Investigators. Percutaneous coronary intervention versus coronary-artery bypass grafting for severe coronary artery disease. *N Engl J Med*. 2009;360:961–972.
25. Levine GN, Bates ER, Blankenship JC, Bailey SR, Bittl JA, Cercek B, Chambers CE, Ellis SG, Guyton RA, Hollenberg SM, Khot UN, Lange RA, Mauri L, Mehran R, Moussa ID, Mukherjee D, Nallamothu BK, Ting HH. 2011 ACCF/AHA/SCAI Guideline for Percutaneous Coronary Intervention: executive summary: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines and the Society for Cardiovascular Angiography and Interventions. *Circulation*. 2011;124:2574–2609.
26. Muller DW, Topol EJ, Ellis SG, Sigmon KN, Lee K, Califf RM. Multivessel coronary artery disease: a key predictor of short-term prognosis after reperfusion therapy for acute myocardial infarction. Thrombolysis and Angioplasty in Myocardial Infarction (TAMI) Study Group. *Am Heart J*. 1991;121(4 Pt 1):1042–1049.
27. Kornowski R, Mehran R, Dangas G, Nikolsky E, Assali A, Claessen BE, Gersh BJ, Wong SC, Witzenbichler B, Guagliumi G, Dudek D, Fahy M, Lansky AJ, Stone GW; HORIZONS-AMI Trial Investigators. Prognostic impact of staged versus "one-time" multivessel percutaneous intervention in acute myocardial infarction: analysis from the HORIZONS-AMI (harmonizing outcomes with revascularization and stents in acute myocardial infarction) trial. *J Am Coll Cardiol*. 2011;58:704–711.
28. Toma M, Buller CE, Westerhout CM, Fu Y, O'Neill WW, Holmes DR Jr, Hamm CW, Granger CB, Armstrong PW; APEX-AMI Investigators. Nonculprit coronary artery percutaneous coronary intervention during acute ST-segment elevation myocardial infarction: insights from the APEX-AMI trial. *Eur Heart J*. 2010;31:1701–1707.
29. Hannan EL, Samadashvili Z, Walford G, Holmes DR Jr, Jacobs AK, Stamato NJ, Venditti FJ, Sharma S, King SB 3rd. Culprit vessel percutaneous coronary intervention versus multivessel and staged percutaneous coronary intervention for ST-segment elevation myocardial infarction patients with multivessel disease. *J Am Coll Cardiol. Cardiovasc Interv*. 2010;3:22–31.
30. Wald DS, Morris JK, Wald NJ, Chase AJ, Edwards RJ, Hughes LO, Berry C, Oldroyd KG; PRAMI Investigators. Randomized trial of preventive angioplasty in myocardial infarction. *N Engl J Med*. 2013;369:1115–1123.
31. Yellon DM, Hausenloy DJ. Myocardial reperfusion injury. *N Engl J Med*. 2007;357:1121–1135.
32. Mooney MR, Unger BT, Boland LL, Burke MN, Kebed KY, Graham KJ, Henry TD, Katsiyiannis WT, Satterlee PA, Sendelbach S, Hodges JS, Parham WM. Therapeutic hypothermia after out-of-hospital cardiac arrest: evaluation of a regional system to increase access to cooling. *Circulation*. 2011;124:206–214.
33. Deakin CD, Nolan JP, Soar J, Sunde K, Koster RW, Smith GB, Perkins GD. European Resuscitation Council Guidelines for Resuscitation 2010 Section 4. Adult advanced life support. *Resuscitation*. 2010;81:1305–1352.
34. Mild therapeutic hypothermia to improve the neurologic outcome after cardiac arrest. *N Engl J Med*. 2002;346:549–556.
35. Bernard SA, Gray TW, Buist MD, Jones BM, Silvester W, Gutteridge G, Smith K. Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. *N Engl J Med*. 2002;346:557–563.
36. Nielsen N, Wetterslev J, Cronberg T, Erlinge D, Gasche Y, Hassager C, Horn J, Hovdenes J, Kjaergaard J, Kuiper M, Pellis T, Stammet P, Wanscher M, Wise MP, Åneman A, Al-Subaie N, Boesgaard S, Bro-Jeppesen J, Brunetti I, Bugge JF, Hingston CD, Juffermans NP, Koopmans M, Køber L, Langørgen J, Lilja G, Møller JE, Rundgren M, Rylander C, Smid O, Werer C, Winkel P, Friberg H; TTM Trial Investigators. Targeted temperature management at 33°C versus 36°C after cardiac arrest. *N Engl J Med*. 2013;369:2197–2206.
37. Scirica BM. Therapeutic hypothermia after cardiac arrest. *Circulation*. 2013;127:244–250.
38. Holmes DR Jr, Bates ER, Kleiman NS, Sadowski Z, Horgan JH, Morris DC, Califf RM, Berger PB, Topol EJ. Contemporary reperfusion therapy for cardiogenic shock: the GUSTO-I trial experience. The GUSTO-I Investigators. Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries. *J Am Coll Cardiol*. 1995;26:668–674.
39. Hochman JS, Sleeper LA, Webb JG, Dzavik V, Buller CE, Aylward P, Col J, White HD; SHOCK Investigators. Early revascularization and long-term survival in cardiogenic shock complicating acute myocardial infarction. *JAMA*. 2006;295:2511–2515.
40. Thiele H, Zeymer U, Neumann FJ, Ferenc M, Olbrich HG, Hausleiter J, Richardt G, Hennersdorf M, Empen K, Fuernau G, Desch S, Eitel I, Hambrecht R, Fuhrmann J, Böhm M, Ebelt H, Schneider S, Schuler G, Werdan K; IABP-SHOCK II Trial Investigators. Intraaortic balloon support for myocardial infarction with cardiogenic shock. *N Engl J Med*. 2012;367:1287–1296.
41. Myerburg RJ, Halperin H, Egan DA, Boineau R, Chugh SS, Gillis AM, Goldhaber JI, Lathrop DA, Liu P, Niemann JT, Ornato JP, Sopko G, Van Eyk JE, Walcott GP, Weisfeldt ML, Wright JD, Zipes DP. Pulseless electric activity: definition, causes, mechanisms, management, and research priorities for the next decade: report from a National Heart, Lung, and Blood Institute workshop. *Circulation*. 2013;128:2532–2541.
42. Andreka P, Frenneaux MP. Haemodynamics of cardiac arrest and resuscitation. *Curr Opin Crit Care*. 2006;12:198–203.
43. Wagner H, Terkelsen CJ, Friberg H, Harnek J, Kern K, Lassen JF, Olivecrona GK. Cardiac arrest in the catheterisation laboratory: a 5-year experience of using mechanical chest compressions to facilitate PCI during prolonged resuscitation efforts. *Resuscitation*. 2010;81:383–387.
44. Rubertsson S, Lindgren E, Smekal D, Östlund O, Silfverstolpe J, Lichtveld RA, Boomars R, Ahlstedt B, Skoog G, Kastberg R, Halliwell D, Box M, Herlitz J, Karlsten R. Mechanical chest compressions and simultaneous defibrillation vs conventional cardiopulmonary resuscitation in out-of-hospital cardiac arrest: the LINC randomized trial. *JAMA*. 2014;311:53–61.
45. Trivedi K, Borovnik-Lesjak V, Gazmuri RJ. LUCAS 2 device, compression depth, and the 2010 cardiopulmonary resuscitation guidelines. *Am J Emerg Med*. 2013;31:1154 e1151-1152.
46. Girotra S, Nallamothu BK, Spertus JA, Li Y, Krumholz HM, Chan PS; American Heart Association Get with the Guidelines–Resuscitation Investigators. Trends in survival after in-hospital cardiac arrest. *N Engl J Med*. 2012;367:1912–1920.
47. Chen YS, Lin JW, Yu HY, Ko WJ, Jerng JS, Chang WT, Chen WJ, Huang SC, Chi NH, Wang CH, Chen LC, Tsai PR, Wang SS, Hwang JJ, Lin FY. Cardiopulmonary resuscitation with assisted extracorporeal life-support versus conventional cardiopulmonary resuscitation in adults with in-hospital cardiac arrest: an observational study and propensity analysis. *Lancet*. 2008;372:554–561.

48. Shin TG, Choi JH, Jo IJ, Sim MS, Song HG, Jeong YK, Song YB, Hahn JY, Choi SH, Gwon HC, Jeon ES, Sung K, Kim WS, Lee YT. Extracorporeal cardiopulmonary resuscitation in patients with inhospital cardiac arrest: A comparison with conventional cardiopulmonary resuscitation. *Crit Care Med*. 2011;39:1–7.
49. Shen I, Levy FH, Vocelka CR, O'Rourke PP, Duncan BW, Thomas R, Verrier ED. Effect of extracorporeal membrane oxygenation on left ventricular function of swine. *Ann Thorac Surg*. 2001;71:862–867.
50. Aiyagari RM, Rocchini AP, Remenapp RT, Graziano JN. Decompression of the left atrium during extracorporeal membrane oxygenation using a transseptal cannula incorporated into the circuit. *Crit Care Med*. 2006;34:2603–2606.
51. Cheng A, Swartz MF, Massey HT. Impella to unload the left ventricle during peripheral extracorporeal membrane oxygenation. *ASAIO J*. 2013;59:533–536.
52. Koeckert MS, Jorde UP, Naka Y, Moses JW, Takayama H. Impella LP 2.5 for left ventricular unloading during venoarterial extracorporeal membrane oxygenation support. *J Card Surg*. 2011;26:666–668.
53. Vlasselaers D, Desmet M, Desmet L, Meyns B, Dens J. Ventricular unloading with a miniature axial flow pump in combination with extracorporeal membrane oxygenation. *Intensive Care Med*. 2006;32:329–333.
54. Avalli L, Maggioni E, Sangalli F, Favini G, Formica F, Fumagalli R. Percutaneous left-heart decompression during extracorporeal membrane oxygenation: an alternative to surgical and transeptal venting in adult patients. *ASAIO J*. 2011;57:38–40.
55. Dixon SR, Henriques JP, Mauri L, Sjauw K, Civitello A, Kar B, Loyalka P, Resnic FS, Teirstein P, Makkar R, Palacios IF, Collins M, Moses J, Benali K, O'Neill WW. A prospective feasibility trial investigating the use of the Impella 2.5 system in patients undergoing high-risk percutaneous coronary intervention (The PROTECT I Trial): initial U.S. experience. *J Am Coll Cardiol. Cardiovasc Interv*. 2009;2:91–96.
56. O'Neill WW, Kleiman NS, Moses J, Henriques JP, Dixon S, Massaro J, Palacios I, Maini B, Mulukutla S, Dzavík V, Popma J, Douglas PS, Ohman M. A prospective, randomized clinical trial of hemodynamic support with Impella 2.5 versus intra-aortic balloon pump in patients undergoing high-risk percutaneous coronary intervention: the PROTECT II study. *Circulation*. 2012;126:1717–1727.
57. Thiele H, Zeymer U, Neumann FJ, Ferenc M, Olbrich HG, Hausleiter J, de Waha A, Richardt G, Hennersdorf M, Empen K, Fuernau G, Desch S, Eitel I, Hambrecht R, Lauer B, Böhm M, Ebelt H, Schneider S, Werdan K, Schuler G; Intraaortic Balloon Pump in cardiogenic shock II (IABP-SHOCK II) trial investigators. Intra-aortic balloon counterpulsation in acute myocardial infarction complicated by cardiogenic shock (IABP-SHOCK II): final 12 month results of a randomised, open-label trial. *Lancet*. 2013;382:1638–1645.
58. Madershahian N, Wippermann J, Liakopoulos O, Wittwer T, Kuhn E, Er F, Hoppe U, Wahlers T. The acute effect of IABP-induced pulsatility on coronary vascular resistance and graft flow in critical ill patients during ECMO. *J Cardiovasc Surg (Torino)*. 2011;52:411–418.
59. Burkhoff D, Cohen H, Brunckhorst C, O'Neill WW; TandemHeart Investigators Group. A randomized multicenter clinical study to evaluate the safety and efficacy of the TandemHeart percutaneous ventricular assist device versus conventional therapy with intraaortic balloon pumping for treatment of cardiogenic shock. *Am Heart J*. 2006;152:469.e1–469.e8.

KEY WORDS: cardiopulmonary resuscitation ■ extracorporeal circulation ■ myocardial infarction ■ shock, cardiogenic

Assisted Beating of the Ischemic Heart: How to Manage the Pulseless ST—Segment-Elevation Myocardial Infarction Patient

Christopher Lotz, Oliver Ritter and Ralf Michael Muellenbach

Circulation. 2014;130:1095-1104
doi: 10.1161/CIRCULATIONAHA.114.009270
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Print ISSN: 0009-7322. Online ISSN: 1524-4539