ECMO in cardiac arrest and cardiogenic shock

Cardiogenic shock and cardiac arrest are life-threatening emergencies with a high mortality rate despite numerous efforts in diagnosis and therapy. For a long time medical therapy – at the forefront with catecholamines, vasodilators and others – and mechanical ventilation, if necessary, were the standard of care for cardiogenic shock. Oxygen supply and perfusion are critically reduced during shock and arrest, and both are physical processes that are in principle amenable to (temporary) extracorporeal mechanical support. Early pioneering work to prove this principle was performed in animals as early as 1937 [1] and in humans 20–30 years later [2, 3]. With the seminal paper by Hill and coworkers [4], extracorporeal membrane oxygenation (ECMO), which can provide blood flow support and extracorporeal gas exchange at the same time, was introduced into the clinic. Since then, technical improvements have contributed to the current worldwide use of ECMO for severe respiratory and cardiopulmonary failure refractory to medical therapy. Recently, there has been some discussion on initiating mechanical support even earlier, with the intention to avoid multiorgan failure associated with excessive catecholamine doses and/or aggressive ventilator settings. By analogy with the concept of veno-venous ECMO and lung-protective ventilation for treatment of acute respiratory distress syndrome, the goal of mechanical support in cardiogenic shock is myocardial rest while protecting end organ perfusion.

In the following, we review ECMO support in the context of cardiogenic shock and refractory cardiac arrest, with a special focus on technical aspects of veno-arterial ECMO. Of note, the following statements are primarily true for percutaneous ECMO with femoral cannulation and may not necessarily be directly transferable to central or upper-body cannulation.

### Cardiogenic shock and cardiac arrest

Cardiogenic shock is the main cause of early mortality in patients with acute myocardial infarction [5]. Other conditions leading to shock comprise acutely decompensated chronic heart failure, decompensated valvular heart disease, myocarditis, Takotsubo syndrome, acute pulmonary embolism, acute allograft failure, incessant arrhythmia, peripartum cardiomyopathy [6], and others [7]. During cardiogenic shock not only the heart itself suffers from pump failure, but even more end organs such as the brain, kidney, liver, and gut are at risk due to insufficient perfusion (multiorgan dysfunction syndrome) [8], and the rate of congestion-associate pneumonia increases. Beyond blood pressure and heart rate as classic shock markers, serum lactate, central venous oxygenation, liver enzyme levels, and urine output are surrogate markers of circulatory failure and multiorgan dysfunction [9]. Reduced coronary perfusion further decreases cardiac output, and multiorgan dysfunction/failure is further complicated by metabolic acidosis and acute coagulopathy. All of these conditions aggravate each other in a fatal vicious circle [8, 9].

Out-of-hospital cardiac arrest (OHCA) occurs with an estimated incidence of 500,000 per year in Europe [10, 11], with two thirds having a primary cardiac cause [12]. Mortality after OHCA remains high despite interventional therapy and modern intensive care. Only 10–15% of those who arrive at the normal hospital survive [13, 14], of whom about 50–80% have a favorable neurological prognosis [15, 16]. In this context, immediate bystander CPR and area-wide availability of automated external defibrillators are essential to increase survival and prognosis. The first electric shock should be applied as early as possible [17] to minimize the time of hypoperfusion, associated LV pump failure, and consecutive development of shock [18]. After return of spontaneous circulation (ROSC), the patient needs to be transferred to an experienced center, which holds all required diagnostic and therapeutic tools [19]. In clinical routine, the first 24 h after resuscitation often

---

**Abbreviations**

| Acronym | Description |
|---------|-------------|
| CPR     | Cardiopulmonary resuscitation |
| ECMO    | Extracorporeal membrane oxygenation |
| ECPR    | Extracorporeal cardiopulmonary resuscitation |
| IABP    | Intra-aortic balloon pump |
| LV      | Left ventricle |
| LVAD    | Left ventricular assist device |
| OHCA    | Out-of-hospital cardiac arrest |
| ROSC    | Return of spontaneous circulation |
**Main topic**

**Table 1** Strategies of mechanical circulatory support

| Strategy          | Indication (examples)                                                                 | Principle                                                                 | Goal            |
|-------------------|---------------------------------------------------------------------------------------|----------------------------------------------------------------------------|-----------------|
| Bridge-to-recovery| Acute heart failure (myocarditis, acute myocardial infarction)                       | Stabilize systemic circulation, ensure end organ perfusion and reduce preload until myocardial recovery | Recovery        |
| Bridge-to-transplantation | Terminal heart failure                                                                 | Stabilize systemic circulation, ensure end organ perfusion until heart transplantation | Transplantation |
| Bridge-to-destination | Terminal heart failure                                                                 | Stabilize systemic circulation, ensure end organ perfusion until LVAD implantation | LVAD            |
| Bridge-to-surgery  | Acute pulmonary embolism with shock (and contraindication for fibrinolysis)          | Reduce preload and stabilize systemic circulation until emergent embolectomy | Embolectomy     |
| Bridge-to-decision | Extracorporeal CPR                                                                    | Stabilize systemic circulation, ensure end organ perfusion until (neurological) re-evaluation and decision on therapeutic strategy | Re-evaluation   |
| Refractory cardiogenic shock | ECMO implantation at the referral center by the ECMO team and transport to the tertiary center for further therapy | Transfer                          |                 |

*Table 1: Strategies of mechanical circulatory support*

| CPR | cardiopulmonary resuscitation, ECMO extracorporeal membrane oxygenation, LVAD left ventricular assist device |

decide on the outcome, and guidelines recommend cardiac catheterization in most cases early after OHCA [20, 21]. Therefore, primary admission to a tertiary center should be preferred over admission to a regional hospital and secondary transfer to a tertiary center, when progression of shock has already occurred.

The majority of patients after OHCA develop post-cardiac arrest syndrome [22, 23] in a vicious circle: Cardiac arrest leads to ischemia of the myocardium and end organs, which results in adverse metabolism, acidosis, and vasoplegia. The hypoperfused heart is not able to respond to the circulatory needs, which in turn aggravates peripheral ischemia [24]. Therefore, restoration of systemic perfusion is essential – particularly in the immediate and early phase after ROSC – in order to limit multiorgan dysfunction [25], which can also be considered a “whole-body reperfusion syndrome.” In this context, complete cardiac revascularization is recommended [12, 26], but care of other end organs such as the brain, intestine, liver, and kidneys is equally important [23].

As outlined, cardiogenic shock and cardiac arrest share many pathophysiological features and evoke many similar responses. Thus, it was not surprising but very important to prove that the prognosis of both conditions is equally adverse: In a recent study of 250 consecutive patients from Denmark, 130 were admitted to a tertiary center with cardiogenic shock, while 118 had OHCA. Interestingly, both groups had the same dismal outcome with 60% 1-week mortality [27]. This underlines the urgent need for novel therapeutic strategies for patients with cardiogenic shock and arrest.

### Restoration of systemic circulation

For many years catecholamines have been used for stabilization of patients with cardiogenic shock. Inotropes such as dobutamine are given with the intention to increase cardiac output by their positive inotropic and chronotropic function. In contrast, vasopressors such as norepinephrine are administered for increasing blood pressure by vasoconstriction and indirect effects such as increased preload. Epinephrine shares features of both drug classes. However, inotropic drugs increase myocardial oxygen consumption, heart rate, arrhythmogenicity, and inflammation in the already diseased heart [28]. Beta1-adrenoceptor agonists have been associated with energy depletion, oxidative stress, and adverse outcome in acute heart failure [29]. Vasopressors increase myocardial afterload and potentially impair peripheral tissue perfusion. Thus, from a pathophysiological perspective, inotropes as well as vasopressors are associated with adverse effects on the heart and other end organs while these organs should recover. Consistently, current guidelines recommend catecholamines as a short-term bridge in the acute situation (only class IIb, level of evidence C), but clearly mention the disadvantages of such drugs, also in light of the paucity of clinical studies demonstrating a survival benefit [25, 30, 31]. In clinical routine, catecholamines are often “effective” in terms of increasing blood pressure, but linked to impaired microcirculation and multiorgan failure, and thus not sufficient for sustained and harmless stabilization of patients with severe cardiogenic shock and resuscitation. In this context, beta-blockers and calcium antagonists taken by the patient before arrest might further contribute to the limited efficacy of catecholamines. Therefore, it is increasingly being discussed to initiate mechanical circulatory support as a powerful tool for bridging earlier and more frequently, in order to improve the prognosis of patients with severe cardiogenic shock or refractory arrest [32]. However, this trend is based on data from many registries and retrospective/observational studies, while evidence from prospective randomized controlled studies is lacking.

### Mechanical circulatory support

Several modes and devices of mechanical support are currently available [32], of which each has its own features and advantages.

The intra-aortic balloon pump (IABP) consists of a catheter-mounted balloon that inflates during diastole and deflates during systole in the descending tho-
in the acute situation. In nonsurgical application in adults, ECMO is a modified form of cardiopulmonary bypass [36], and has undergone a dramatic technical evolution since the widely known publication by Hill and coworkers in 1972 [4]. In principle, ECMO drains venous blood through a cannula and tubing and returns it via another tubing and cannula into the body, both driven by a rotor unit. During ECMO passage the blood becomes oxygenated, decarboxylated, and warmed in an extracorporeal gas exchange unit. In nonsurgical application in adults, peripheral cannulation of the femoral and/or jugular vessels is the standard technique, usually with 21–25 French

**ECMO in cardiac arrest and cardiogenic shock**

**Abstract**

Cardiogenic shock is an acute emergency, which is classically managed by medical support with inotropes or vasoressors and frequently requires invasive ventilation. However, both catecholamines and ventilation are associated with a worse prognosis, and many patients deteriorate despite all efforts. Mechanical circulatory support is increasingly considered to allow for recovery or to bridge until making a decision or definite treatment. Of all devices, extracorporeal membrane oxygenation (ECMO) is the most widely used. Here we review features and strategetical considerations for the use of ECMO in cardiogenic shock and cardiac arrest.

**Keywords**

Cardiogenic shock · Cardiac arrest · Sudden cardiac death · Cardiopulmonary resuscitation · ECMO · Mechanical circulatory support · Microaxial pump · Extracorporeal resuscitation

In general, mechanical support can be used with different strategies (Table 1). In patients with severe cardiogenic shock from myocardial infarction or myocarditis, mechanical support is routinely employed in a bridge-to-recovery approach. In the case of acute decompensated chronic heart failure, the potential for recovery may be limited, which sometimes results in a bridge-to-destination approach. In resuscitated patients, a bridge-to-decision strategy is usually required, as further therapies such as LVAD surgery, ICD implantation etc. are postponed until awakening of the patient allows for estimating neurological recovery and eligibility.

**Technical aspects**

ECMO is a modified form of cardiopulmonary bypass [36], and has undergone a dramatic technical evolution since the widely known publication by Hill and coworkers in 1972 [4]. In principle, ECMO drains venous blood through a cannula and tubing and returns it via another tubing and cannula into the body, both driven by a rotor unit. During ECMO passage the blood becomes oxygenated, decarboxylated, and warmed in an extracorporeal gas exchange unit. In nonsurgical application in adults, peripheral cannulation of the femoral and/or jugular vessels is the standard technique, usually with 21–25 French
draining and 15–19 French returning cannulas (Table 2). Veno-venous (VV) ECMO drains from and returns to the right atrium. It is used for replacement of lung function, typically during acute respiratory distress syndrome, and is not further discussed here.

In contrast, veno-arterial (VA) ECMO drains blood from the right atrium and returns to the arterial system, typically to the iliac arteries toward the aorta (Fig. 1). By this, VA-ECMO reduces preload and increases aortic flow and end organ perfusion [36]. With arterial cannulation, placement of a dedicated sheath for antegrade perfusion of the cannulated leg (Fig. 1) is recommended to prevent leg ischemia [37], which is standard in many centers.

A great advantage of VA-ECMO is that cannulation may be performed nearly everywhere, as the system and all parts are transportable. Thus, an unstable patient can receive ECMO support in the emergency room, on the ward, in the catheterization laboratory, the operating theater, or even in the field [38, 39]. In contrast to other support systems, fluoroscopy or echocardiography guidance is – albeit helpful – not required for successful implantation. Once ECMO is running, the patient can be transferred with the whole unit, which is another advantage over other systems. Therefore ECMO is frequently used for transport of unstable patients by car, helicopter, or even by plane as an air-bridge [40].

VA-ECMO establishes a massive right-to-left shunt by draining venous blood and returning it to the iliac artery. This flow support, which can reach 7 l/min with large cannulas and contemporary rotors, results in a significant increase in blood pressure as long as there is enough vascular resistance (pressure = flow × resistance). The massive venous drainage effectively reduces preload and thus leads to venous decongestion. Arterial reinfusion to the systemic circulation strongly enhances perfusion of end organs and is therefore attractive during severe cardiorespiratory failure or resuscitation. Of note, at the same time retrograde flow support increases LV afterload (see next section).

### Contraindications and complications

Notwithstanding the fast set-up of the system and the efficient hemodynamic support, VA-ECMO has contraindications and harbors a significant risk of complications (Table 2). Most contraindications are relative owing to the lifesaving nature of ECMO support, which in turn underlines that ECMO should only be initiated when ethical aspects or the patient’s wish do not preclude mechanical support. Uncontrolled bleeding is a contraindication, as ECMO requires heparin for anticoagulation at least for longer support. In selected patients, however, this contraindication is relative, if ECMO is the only strategy to save the life of the patient. There are indeed centers that run ECMO support in high-risk patients without any anticoagulation (off-label) for a limited time (such as in severe trauma [41] or diffuse alveolar hemorrhage [42]). A nearly absolute contraindication is severe aortic regurgitation: The retrograde flow support of VA-ECMO would cause severe LV distension and pulmonary edema. VA-ECMO results in LV distension even in patients with moderate aortic regurgitation [43]. Further contraindications are listed in Table 2.

ECMO support is an invasive procedure with profound changes of body oxygenation and circulation, and inherently associated with potentially severe complications [37, 44]. Among these are vascular complications, leg ischemia, bleeding, hyperfibrinolysis, stroke, and air embolism (Table 2). These are anticipated and in most cases effectively controlled in tertiary centers. This emphasizes that initiation, maintenance, weaning, and removal of ECMO requires a strong theoretical and practical expertise and should be performed in high-volume centers only.

### Pathophysiology: watershed

The retrograde ECMO output meets the antegrade LV output at a zone called the “watershed” [36, 45, 46]. In most cases the watershed occurs somewhere between the aortic root and the di-

---

**Table 2** Technical features of VA-ECMO

| Implantation | Cannulation of femoral artery (15–19 Fr) and vein (21–15 Fr) with modified Seldinger’s technique takes about 10 min until circuit starts |
|-------------|----------------------------------------------------------------------------------------------------------------------|
| Mobility    | Inter- and intrahospital transfer, up to air-bridge (flight transfer)                                                     |
| Hemodynamic effect | Increased systemic perfusion by retrograde flow support                                                              |
|             | Preload reduction                                                                                                         |
|             | Afterload increase                                                                                                         |
| Flow rates  | Up to 7 l/min, depending on cannulas and rotor/oxygenator                                                                |
| Gas exchange | Highly efficient oxygenation and decarboxylation of reinfused blood                                                    |
| Contraindications | Ethical considerations, patient’s will                                                                 |
|             | No perspective of a bridging strategy                                                                                      |
|             | Severe peripheral artery disease (iliac)                                                                                |
|             | (Severe) aortic regurgitation                                                                                            |
|             | Aortic dissection                                                                                                         |
|             | Left ventricular thrombus (relative)                                                                                      |
|             | Uncontrolled bleeding disorder (relative)                                                                                 |
| Potential complications | Leg ischemia                                                                                                               |
|             | Bleeding                                                                                                                  |
|             | Vascular complications                                                                                                    |
|             | Two-circulation syndrome                                                                                                  |
|             | LV distension                                                                                                             |
|             | Hyperfibrinolysis                                                                                                         |
|             | Embolism                                                                                                                  |

Fr French, VA-ECMO veno-arterial extracorporeal membrane oxygenation
aphragm (Fig. 2), depending on the native output of the heart: The higher the LV output relative to ECMO output, the more distal the watershed [46]. Since the output of most ECMO devices is nonpulsatile, pulse pressure measured at the right radial artery serves as an estimate of LV output [46]. For example, a blood pressure of 80/70 mm Hg at an ECMO flow of 4.5 l/min suggests a watershed in the aortic root, whereas a blood pressure of 140/70 mm Hg at the same ECMO flow suggests a watershed in the descending thoracic aorta. Blood from the ECMO is usually well oxygenated; however, oxygenation of blood from the LV depends on the respiratory function of the lung. Therefore the position of the watershed is critical for oxygenation. Aortic root oxygenation cannot be continuously measured with standard equipment. If the watershed is located in the ascending aorta and blood from the LV has an oxygen saturation of, e.g., 56% during lung failure, then the heart itself may be perfused for hours or days with an extremely insufficient oxygen saturation from the lungs in the presence of sufficient oxygenation of all other organs from the ECMO. In this context, the extreme form of dismal circulation is the “two-circulation-syndrome” [47]: If the venous cannula is incorrectly placed in the inferior caval vein, so that only blood from the upper body is drained, blood from the upper body goes through the lungs to the ascending aorta. Then venous drainage from and the perfusion of the upper body are both disconnected from that of the lower body. This results in a “Harlequin”-like appearance of the patient, with upper-body hypoxia and lower-body hyperoxia.

As outlined, circulation and oxygenation are overall subject to profound changes during VA-ECMO. Therefore
multiple parameters have to be monitored in a patient on VA-ECMO at the same time (Table 3; [48]).

**Triple cannulation**

VA-ECMO delivers powerful circulatory and respiratory support (Table 2). Carbon dioxide elimination by the ECMO is nearly always sufficient, thus hypercapnia is nearly never a problem in patients on ECMO support – in contrast to (differential) hypoxia. As outlined earlier, the high oxygen content of ECMO output reaches only organs below the watershed. Thus, under normal conditions the lower extremities, gut, kidneys, liver etc. are well oxygenated during VA-ECMO support. An additional effect on organ oxygenation results from a higher amount of oxygen delivered to the lower body and an associated higher venous backflow oxygen: Depending on oxygenation settings, ECMO outflow pO2 usually equals at least 200–300 mm Hg, compared with 50–100 mm Hg in arterial blood oxygenated in the lungs of a standard ventilated shock patient. This results in a higher total oxygen delivery to the body, which may have an effect also on organs perfused by LV blood, yet the relevance of this effect is unclear to date.

However, in some patients on VA-ECMO support secondary lung failure develops. This is a dangerous situation: Depending on the watershed position, all organs perfused by blood from the heart are prone to severe ischemia in the presence of ECMO support, in particular the heart and brain. If lung failure is due to pulmonary edema, ultrafiltration and active LV unloading (see later) are sufficient to achieve decongestion. However, in many patients with lung failure on VA-ECMO support, the problem results from an ARDS-like condition, which cannot be or should not be effectively solved by aggressive ventilation or decongestion. In these patients an elegant and very effective treatment is upgrading the ECMO circuit to a triple-cannulated ECMO, with one venous-draining, one arterial-supplying, and one venous-supplying cannula (“VAV-ECMO”, Fig. 3; [36, 49]). In addition to the VA circuit, the additional venous cannula adds preoxygenated blood to the lungs and thereby establishes a “VV component.” This ensures sufficient oxygen content of blood ejected by the heart and allows for lung protective ventilation. Of note, VAV-ECMO requires sufficient RV function, otherwise it may be necessary to relocate the venous-supplying cannula into the pulmonary artery [49] for bypassing the RV. Retrospective studies suggest efficacy of VAV cannulation for rescue of body oxygenation and recovery of lung failure [50–52], but prospective studies are needed to confirm the observed benefit.

**Pathophysiology: afterload, decompression**

During acute heart failure, the diseased LV has impaired ability to eject, and

---

**Table 3 Monitoring of patients on VA-ECMO**

| Parameter | Reason/surrogate |
|-----------|-----------------|
| **Hemodynamics** |  |
| PA catheter: Mean PA pressure, PC wedge pressure | Efficacy of preload reduction |
| Central venous pressure | Efficacy of preload reduction |
| Right radial pulsatility | LV output |
| Right radial mean blood pressure | Perfusion pressure |
| Consider CCO catheter | LV output |
| Central venous oxygen saturation | Systemic circulation |
| Urine output | Renal perfusion and function |
| Lab: liver enzymes | Venous decongestion |
| **Respiratory support** |  |
| Right radial blood gases | Brain oxygenation, decarboxylation |
| Lactate | End organ ischemia |
| Transcutaneous continuous near-infrared spectroscopy | Tissue oxygenation (independent of pulsatility) |
| Pulse oximetry (right hand finger or ear) | Tissue oxygenation (largely dependent of pulsatility) |
| Acral perfusion (clinical) | Tissue perfusion |
| ECMO outflow blood gases | Control of oxygenator capacity |
| **Imaging** |  |
| Echocardiography | LV distension |
| | Aortic regurgitation |
| | Pericardial effusion |
| | RV function |
| | LV thrombus |
| Chest X-Ray | Pulmonary edema, pneumothorax |
| Pleural sonography | Pleural effusion |
| **Coagulation** |  |
| D-dimer, fibrinogen, platelet count | Hyperfibrinolysis |
| Free hemoglobin, LDH | Hemolysis |
| Activated clotting time (POCT) | Anticoagulation |
| Blood cell count | Anemia, thrombopenia |
| **Leg perfusion** |  |
| Clinical perfusion assessment | Ischemia of the cannulated leg |

**General critical care monitoring**

CCO continuous cardiac output, LDH lactate dehydrogenase, LV left ventricle, PA pulmonary artery, PC pulmonary capillary, POCT point of care testing

*Peripheral femoro-femoral cannulation

*Classic thermodilution is not reliable owing to right atrial drainage
stroke work and myocardial oxygen consumption are increased [30, 53]. When bridge-to-recovery is the therapeutic goal (e.g., myocarditis or myocardial infarction), stroke work and myocardial oxygen consumption have to be reduced to facilitate regeneration. However, notwithstanding the immediate massive hemodynamic and respiratory support and the reduction of preload, VA-ECMO increases LV afterload [53–57]. This may result in increased LV filling pressures, wall stress, and severe pulmonary congestion despite reduction of preload. Moreover, ECMO is often ascribed a positive effect on coronary perfusion; however, human data are lacking and data from animal studies are conflicting [58, 59]. From a pathophysiological perspective, a high LV pressure during diastole impairs coronary perfusion by reducing the transcoronary perfusion gradient. In patients with extremely low systolic LV function and in all patients with ongoing arrest, VA-ECMO support results in a functionally closed aortic valve without relevant transaortic blood flow. This potentially results in severe LV distension [54] and pulmonary congestion in the presence of sufficient systemic circulation.

Thus, LV unloading, prevention of LV distension, reduction of myocardial wall stress, and enhancement of coronary perfusion are important goals during mechanical circulatory support for bridge-to-recovery. Unloading (= “venting”) can be achieved by different methods. One way is venting through the atrial septum, either by atrioseptostomy [60, 61] or placement of an additional draining cannula through the atrial septum [62], both of which are potentially hazardous [61] particularly in the already critically ill patient. Another possibility is transvalvular unloading across the aortic valve, which has already been performed in an experimental approach with a transvalvular coronary catheter connected to the venous draining ECMO cannula [63]. However, simple draining of the LV has no direct effect on coronary perfusion and does not increase antegrade transaortic blood flow. Therefore, pumps have been developed that are percutaneously inserted, drain the LV, and eject into the ascending aorta. Their first use (Hemopump®) was published as early as in 1990 [64], but the clinical breakthrough took nearly 20 years to occur, mainly attributed to technical improvement of the device. Today, the only transvalvular microaxial pump approved in the United States and Europe is the Impella® device (Abiomed, Danvers, USA), which is the current device of choice of most centers for active LV unloading, also combined with VA-ECMO (Fig. 4).

The frequency of the combined use of ECMO and Impella® varies greatly between centers. Of note, it is unclear to date which patients have a benefit of additional Impella® support in parallel to VA-ECMO. There are two published studies reporting combined support [65, 66]. Their data point to a benefit of dual support, but further studies are unequivocally needed. Fig. 5 shows a proposal for the management of VA-ECMO and potential unloading, based on pathophysiological considerations and clinical practice in our center. In general, the lower systolic LV function is in a given patient, the sooner active LV unloading should be considered.

**VA-ECMO for cardiogenic shock**

Despite the broad use of ECMO in experienced centers, data from larger studies are limited. Most studies are retrospective series or registry studies. Some years ago, IABP was used in many countries almost routinely for patients with severe cardiogenic shock, but later on randomized studies demonstrated the noneffectiveness of routine IABP support [33]. With this in mind, the decision for or against mechanical support and the de-
VA-ECMO is the commonly used device for bridge-to-decision. By contrast, VA-ECMO may not be the ideal support form for isolated LV dysfunction with potential for recovery (acute myocardial infarction, myocarditis, Takotsubo syndrome, etc.), since afterload increases and recovery may be hampered.

Of note, these are considerations from daily clinical routine and pathophysiology, but dedicated studies are urgently needed to prospectively compare the different support forms. One such study is the prospective, open-label, multicenter, randomized, controlled “ANCHOR” trial (Assessment of ECMO in Acute Myocardial Infarction with Non-reversible Cardiogenic Shock to Halt Organ Failure and Reduce Mortality), which is currently investigating the use of ECMO in cardiogenic shock during myocardial infarction. In this context, an interesting tool that is already mentioned in current heart failure guidelines is the “SAVE” score to estimate the prognosis of patients with cardiogenic shock on VA-ECMO. Another promising score is the “ENCOURAGE” score.

Mechanical support is increasingly used in cardiogenic shock to minimize or avoid catecholamines and to facilitate regeneration of the diseased heart. Refractory cardiac arrest is an emerging indication for mechanical support, and recently more centers have developed ECPR programs. Cardiogenic shock and...
| Reference          | Origin      | Design          | Comparison                      | Etiology                                      | Patients (N) | Age                  | Implantation              | LVEF       | Outcome                           | Complications                                      |
|--------------------|-------------|-----------------|---------------------------------|-----------------------------------------------|--------------|----------------------|---------------------------|------------|------------------------------------|----------------------------------------------------|
| Sheu et al. [67]   | Taiwan      | Prospective     | ECMO+IABP vs. IABP              | 100% STEMI in both groups                     | 46 vs. 25    | 65.1 ± 10.6 years vs. | In the cathlab (probably shortly after PCI, but timepoint not exactly reported) | Data not reported | 30-d survival: 60.9% ECMO-IABP vs. 28.0% IABP | Bleeding or vascular complications: 39.1%          |
| Tsao et al. [68]   | Taiwan      | Retrospective   | ECMO+IABP vs. IABP              | ECMO+IABP: 54.5% STEMI, 45.5% NSTEMI (93.9% had IABP) | 33 vs. 25    | 74.1 ± 12.2 years vs. | In the emergency room or cathlab | ECMO+IABP: 38 ± 10% IABP: 39 ± 14% | Successful weaning: 81.8% in ECMO+IABP vs. 44.0% in IABP survival to discharge: 66.7% in ECMO+IABP vs. 32.0% in IABP 1-year survival: 63.6% in ECMO+IABP vs. 24.0% in IABP | Data not reported                                  |
| Sakamoto et al. [69]| Japan       | Retrospective   | no device comparison all had VA-ECMO | 100.0% ACS, 36.7% had cardiac arrest before ECMO 95.9% received emergency revascularization | 98           | 72 ± 12 years (mean, SD) | 44.9% implant on admission, 33.7% implant during PCI, 20.4% implant after PCI. 95.9% had additional IABP | Data not reported | Successful weaning: 55.1% survival to discharge: 32.7% | 35.7% ECMO-related complications: 23.5% cannula site complications: 4.1% retroperitoneal hemorrhage: 7.1% lower limb ischemia: 3.1% cerebral hemorrhage |
| Sattler et al. [70]| Germany     | Retrospective   | ECMO vs. IABP                   | ECMO: 66.7% STEMI, 33.3% NSTEMI with 66.7% OHCA and 16.7% IHCA IABP: 83.3% STEMI, 16.7% NSTEMI, with 41.7% OHCA and 16.7% IHCA | 12 vs. 12    | 54.8 ± 13.3 years vs. | 1 pat. before PCI 9 pat. immediately after PCI 2 pat. 24 and 48 h after PCI and IABP | ECMO: 48 ± 10% IABP: 32 ± 13% | 30-d survival: 67.0% ECMO vs. 33.0% IABP | 3/12 bleeding: 2/12 compartment syndrome: hemolysis with 21.0 ± 12.4 packed red blood cell transfusions per patient |
| Reference  | Origin | Design          | Comparison                  | Etiology                                      | Patients (N) | Age                  | Implantation | LVEF | Outcome                  | Complications                                    |
|------------|--------|-----------------|-----------------------------|-----------------------------------------------|--------------|----------------------|--------------|------|--------------------------|--------------------------------------------------|
| Aso et al. [71] | Japan  | Register no device comparison all had VA-ECMO | 42.2% Ischemic heart disease (IHD), 34.8% Heart failure (HF), 13.7% Valvular heart disease (VHD), 4% Myocarditis (MYO), 4.1% Cardiomyopathy (CMP), 0.7% Takotsubo syndrome (TS), 0.3% Infectious endocarditis (IE) Patients who had cardiac arrest: All 47.0%, IHD 25.0%, HF 15.0%, VHD 2.7%, MYO 1.4%, CMP 2.5%, TS 0.3%, IE 0.06% | 4,658 | 73.0% men              | All 64.8 ± 13.7 years (mean, SD) | Data not reported | 60.8% had IABP prior to or in parallel to VA-ECMO | Data not reported | Survival to discharge all patients 26.4%, IHD 20.9%, HF 32.2%, VHD 23.0%, MYO 43.0%, CMP 26.9%, TS 35.3%, IE 25.0% | Data not reported |
| Muller et al. [72] | France | Prospective observational no device comparison all had VA-ECMO | 100% acute myocardial infarction 13.8% received VA-ECMO during CPR and 43.5% after CPR | 138 | 79.7% men (median, IQR) | 55 (46–63) years (median, IQR) | 10.1% before and 89.9% after PCI 69.6% had IABP parallel to ECMO 2.2% had Impella and ECMO 11.6% were switched to central ECMO cannulation | Data not reported | 20 (15–25% (median, IQR) | Successful weaning 35.5% 6-months survival 41.3% | 39.1% ECMO complications: 12.3% bleeding 10.9% leg ischemia 11.6% access site infection 3.6% hemolysis 11.6% overt pulmonary edema on ECMO |

CPR cardiopulmonary resuscitation, ECMO extracorporeal membrane oxygenation, ECPR extracorporeal CPR, IABP intra-aortic balloon pump, IQR interquartile range, LVEF left ventricular ejection fraction, NSTEMI Non-ST-elevation myocardial infarction, pat patients, PCI percutaneous coronary intervention, STEMI ST-elevation myocardial infarction
| Reference | Origin | Design | IHCA/ OHCA | Etiology | Patients (N) | Age | Bystander CPR | Initial rhythm | Time-to-ECMO | Initial pH | Initial lactate | Outcome | ECMO-related complications | Predictors of mortality |
|-----------|--------|--------|------------|----------|--------------|-----|---------------|----------------|--------------|------------|-----------------|---------|---------------------------|-----------------------|
| Chen et al. [74] | Taiwan | Retrospective | 96.5%/ 3.5% | 24.6% postcardiotomy all cardiac origin, further details not reported | 57 | 59.6% men | 57.1 ± 15.6 years (mean, SD) | VF | 47.4%, VT 14.0%, PEA/asystole 38.6% | 47.6 ± 13.4 min. (mean, SD) | Data not reported | Data not reported | Weaning off ECMO 66.7% overall survival 31.6% post-cardiotomy 57.1% non-post-cardiotomy 23.3% | Massive retroperitoneal hematoma 1.8% limb amputation after ECMO cannulation 1.8% further data not reported |
| Massett et al. [75] | France | Retrospective | 87.5%/ 12.5% | 40% ACS, 10% HF, 15% Intoxication, 10% RH, 10% post-cardiotomy, 7.5% PE, 5% MYO | 40 | 57.5% men | 42 ± 15 years (mean, SD) | Data not reported | Data not reported | 105 ± 44 min. (mean, SD) | Data not reported | Data not reported | Weaning off ECMO 30% survival to discharge 20% | Vascular complications 12.5% leg ischemia 2.5% bleeding 7.5%, pulmonary hemorrhage 12.5% |
| Sung et al. [76] | South Korea | Observational | 100%/ 0% | 36.3% coronary artery disease, 36.3% after cardiac surgery, 9% HF, 9% others, 4.5% PE, 4.5% MYO | 22 | 54.5% men | 62.5 ± 14.0 years (mean, SD) | Data not reported | Data not reported | 48.5 ± 29.0 min. (mean, SD) | Data not reported | Data not reported | Weaning off ECMO 59.1% survival to discharge with good neurological outcome 40.9% | 13.6% bleeding 4.5% vascular complications |
| Chen et al. [77] | Taiwan | Prospective observational | 100%/ 0% | 62.7% ACS, 10.2% HF, 8.5% MYO, 11.9% post-cardiotomy, 1.7% PE, 5.1% others | 59 | 84.7% | 57.4 ± 12.5 years (mean, SD) | VT/VF | 49.2%, PEA 28.8%, Asystole 22.0% | 52.8 ± 37.2 min. (mean, SD) | Data not reported | Data not reported | Weaning off ECMO 49.2% survival to discharge 28.8% 1-year survival 18.6% | Data not reported | Time-to-ECMO initial rhythm other than VT/VF |
| Reference | Origin       | Design                      | IHCA/OHCA   | Etiology                                                                 | Patients (N) | Age            | Bystander CPR | Initial rhythm | Time-to-ECMO | Initial pH | Initial lactate | Outcome                                             | ECMO-related complications | Predictors of mortality |
|-----------|--------------|-----------------------------|-------------|---------------------------------------------------------------------------|--------------|----------------|---------------|----------------|--------------|-------------|----------------|------------------------------------------------------|-----------------------------|--------------------------|
| Kagawa et al. [78] | Japan       | Retrospective IHCA vs. OHCA | 49.4%/50.6% | IHCA 55% ACS, 3% HF, 5% MYO, 16% PE, 21% others OHCA 56% ACS, 5% HF, 3% MYO, 15% PE, 21% others | 38 vs. 39 56 years | VT/VF 26% | OHCA 59 (45–65) min. | IHCA 25 (21–43) min. | OHCA 7.24 (7.09–7.39) | Data not reported | Data not reported | Weaning off ECMO IHCA 61%, OHCA 36% good neurological outcome at discharge IHCA 26%, OHCA 10% 30-days survival IHCA 34%, OHCA 13% | Leg ischemia IHCA 18%, OHCA 21% Bleeding or hematoma IHCA 68%, OHCA 59% | Time-to-ECMO initial rhythm other than VF |
| Le Guen et al. [79] | France      | Prospective observational   | 0%/100%     | 86% cardiac (no further details), 6% trauma, 4% drug overdose, 2% respiratory, 2% others | 51 90% men | VF 63% | Asystole 29% min. | 120 (102–149) min. | 6.93 ± 0.17 (mean, SD) | 19.9 ± 6.7 (mean, SD) | 24 h-survival 40% 48 h-survival 12% survival with good neurological outcome at day 28 4% | 14% severe hemorrhage further data not reported | Lactate at baseline end-tidal CO2 time-to-ECMO |
| Avalli et al. [80] | Italy        | Retrospective IHCA vs. OHCA | 57.1%/42.9% | IHCA 37% ACS, 33% postcardiomy, 13% PE, 9% HF, 9% others OHCA 67% ACS, 5% HF, 11% RHY, 17% others | 24 vs. 18 67 years | IHCA 100% | OHCA 55% | IHCA 55 (40–70) min. | Data not reported | Data not reported | Weaning off ECMO IHCA 58%, OHCA 16% 28-days survival IHCA 46%, OHCA 5% | IHCA 46% vascular compl. OHCA 33% vascular compl. | Data not reported |
| Reference       | Origin     | Design              | IHCA/ OHCA (%) | Etiology                                                                 | Patients (N) | Age (mean, SD) | Bystander CPR | Initial rhythm | Time-to-ECMO | Initial pH (Mean, SD) | Initial lactate (Mean, SD) | Outcome                                      | ECMO-related complications | Predictors of mortality |
|-----------------|------------|---------------------|----------------|---------------------------------------------------------------------------|--------------|----------------|---------------|----------------|--------------|------------------------|----------------------------|-------------------------------------------|----------------------------|--------------------------|
| Chungetal. [81] | Taiwan     | Prospective         | 100%/0%        | 27.6% STEMI, 11.9% NSTEMI, 22.4% postsurgery, 10.5% HF, 19.4% MYO, 6.0% post-PCI, 2.2% others | 134          | 51.8 ± 20.5 years | 100%          | VT/VF 27.6%, further data not reported | Data not reported | Data not reported | Data not reported | Weaning off ECMO 50.7% survival to discharge 42.5% survival 30 days 54.5% | Overall 21.6% peripheral limb ischemia 3.0% further data not reported | APA-CHE-II-Score ≥22 unsuccessful weaning off ECMO |
| Haneya etal. [82] | Germany | Retrospective     | 69.4%/30.6% | 30.6% ACS, 15.3% HF, 17.6% post-PCI/TAVI, 16.5% PE, 2.4% HYPO, 5.9% TRA, 11.6% others. Post-cardiomy patients were excluded | 85           | 59 ± 16 years | Data not reported | VT/VF 29.4%, PEA 42.4%, Asystole 28.2% | 51 ± 35 min. (mean, SD) | All 7.01 ± 0.22 IHCA 7.09 ± 0.18 OHCA 6.85 ± 0.24 (mean, SD) | All 11 ± 6.9 IHCA 7.2 ± 5.6 OHCA 14.7 ± 9.1 (mean, SD) | Weaning off ECMO 47.1% (IHCA 57.6%, OHCA 23.1%) survival to discharge 34.1% (IHCA 42.4%, OHCA 15.4%) 93.1% without severe neurological deficit among discharged patients | Overall 32.9% leg ischemia 16.5% bleeding 3.5% cannulation complications 12.9% | pH, CPR duration |
| Fagnou etal. [83] | Belgium | Prospective         | 41.7%/58.3% | 29.2% ACS, 20.8% RHY, 12.5% PE, 8.3% TRA, 8.3% Intoxication, 12.5% HYPO, 8.3% others | 24           | 48 (38–55) years (median, IQR) | VT/VF 41.7%, PEA, Asystole 58.3% | Survivors 7.22 ± 0.23 min. (mean, SD) | Survivors 9.8 ± 5.3 non-survivors 14.9 ± 4.85 (mean, SD) | Weaning off ECMO 29.2% survival to ICU discharge 25.0% | Major bleeding on ECMO site 29.2% diffuse bleeding 41.7% | Time-to-ECMO (non-significant trend) |
| Reference | Origin | Design      | IHCA/ OHCA | Etiology                                                                 | Patients (N) | Age (mean, SD) | Bystander CPR | Initial rhythm | Time-to-ECMO | Initial pH | Initial lactate | Outcome | ECMO-related complications | Predictors of mortality |
|-----------|--------|-------------|------------|---------------------------------------------------------------------------|--------------|----------------|---------------|---------------|--------------|------------|----------------|---------|---------------------------|------------------------|
| Leick et al. [84] | Germany | Retrospective | 0%/100%     | 53.6% ACS, 21.4% HF, 23.1% septic shock, 7.1% Takotsubo syndrome, 3.6% PE, 3.6% MYO | 28 53.6% men | 53.9 ± 15.9 years (non-survivors) 60.3 ± 9.6 years (survivors) (mean, SD) | Data not reported | VF 28.6%, Asystole 21.4%, PEA 39.3%, 10.7% not reported | 44.0 (31.0–45.0) min. (survivors) 53.0 (40.0–61.3) min. (non-survivors) (median, IQR) | Survivors 7.2 (7.0–7.3) (median, IQR) | 30-day survival 39.3% | Leg ischemia 3.6% bleeding 32.1% | Time-to-ECMO |
| Stub et al. [85] | Australia | Prospective observational | 57.7%/42.3% | 53.8% ACS, 7.7% HF, 11.5% Arrhythmia, 7.7% PE, 7.7% respiratory, 11.5% others | 26 77% men | 52 (38–60) years (median, IQR) | Data not reported | VF 73.1%, PEA 15.4%, Asystole 11.5% | 56 (40–85) min. (median, IQR) | all 6.9 (6.7–7.1) survivors 7.0 (6.8–7.1) non-survivors 6.8 (6.7–7.0) (median, IQR) | all 10 (7–14) survivors 8 (6–12) non-survivors 13 (9–14) (median, IQR) | Weaning off ECMO 54.1% survival to discharge 53.8% | Bleeding 69.2% peripheral vascular issues 38.5% vascular surgery 41.7% | Time-to-ECMO, pH, troponin |
| Jung et al. [86] | Germany | Retrospective | 70.9%/29.1% | 23.1% VT/VF in HF, 40.2% VT/VF in ACS, 28.1% post-surgery/-intervention, 9.4% others | 117 68.4% men | 61 (51–74) years (median, IQR) | Data not reported | VT/VF 63.2%, further data not reported | Data not reported | Data not reported | Data not reported | Waning off ECMO 52.1% 30-days survival 23.1% good neurological outcome 14.5% | Data not reported | Lactate, hemoglobin |

**ACS** acute coronary syndrome, **CPR** cardiopulmonary resuscitation, **ECMO** extracorporeal membrane oxygenation, **HF** heart failure, **HYPO** accidental hypothermia, **IHCA** in-hospital cardiac arrest, **IQR** interquartile range, **MYO** myocarditis, **NSTEMI** non-ST-elevation myocardial infarction, **OHCA** out-of-hospital cardiac arrest, **PE** pulmonary embolism, **PEA** pulseless electrical activity, **RHY** arrhythmia, **SD** standard deviation, **STEMI** ST-elevation myocardial infarction, **TRA** trauma, **VF** ventricular fibrillation, **VT** ventricular tachycardia

*No overlapping patients*
arrest share many pathophysiological features, and in this context VA-ECMO is a powerful extracorporeal life support system, as long as it is initiated early. VA-ECMO use requires a dedicated bridging strategy, such as bridge-to-recovery, bridge-to-decision, or bridge-to-destination, and complications need to be anticipated. Retrograde flow support increases LV afterload and may result in LV distension, which can be prevented and resolved by LV venting or active LV unloading. Prospective controlled studies are needed to develop specific protocols for defined clinical conditions, in order to find the optimal mechanical support strategy in a given situation.

**Table 6**  Proposed criteria for extracorporeal CPR (ECPR)

| Inclusion criteria (all need to be met) |
|----------------------------------------|
| Witnessed circulatory arrest |
| Bystander CPR |
| Age < 75 years* |
| No ROSC after 10 min of professional CPR* |

| Exclusion criteria (one criterion is sufficient) |
|-----------------------------------------------|
| Severe comorbidity (cancer, end-stage liver cirrhosis, etc.) |
| Preexisting cognitive impairment/brain damage |

| Preclinical CPR > 1h* |
|-----------------------|

| Optional exclusion criteria |
|-----------------------------|
| pH at baseline < 6.8 |
| Lactate at baseline > 15 mmol/l |

| Exceptions for criteria above |
|-----------------------------|
| Accidental hypothermia |

| CPR | Cardiopulmonary resuscitation, ECMO extracorporeal membrane oxygenation |
|-----|-----------------------------------------------------------------------|

*Age limit depends on comorbidities and biological age
*Excellent CPR until ECMO is an essential prerequisite for success
*May be extended in single cases, when very young patients need time for transfer and have optimal CPR

**References**

1. Gibbon JH Jr. (1937) Artificial maintenance of circulation during experimental occlusion of pulmonary artery. Arch Surg 34:1105–1131
2. Kennedy JH (1966) The role of assisted circulation in cardiac resuscitation. JAMA 197:615–618
3. Gibbon JH Jr. (1954) Application of a mechanical heart and lung apparatus to cardiac surgery. Minn Med 37:171–185
4. Hill JD, O’Brien TG, Murray JJ et al (1972) Prolonged extracorporeal oxygenation for acute post-traumatic respiratory failure (shock-lung syndrome). Use of the Bramson membrane lung. N Engl J Med 262:510–513
5. Goldberg RJ, Spencer FA, Gore JM et al (2009) Early defibrillation: an advisory statement from the Advanced Life Support Working Group of the International Liaison Committee on Resuscitation. Circulation 119:879–887
6. Hilfiker-Kleiner D, Nonhoff J, Bauer-Jung J et al (2015) Peripartum cardiomyopathy: current management and future perspectives. Eur Heart J 36:1090–1097
7. Reynolds HR, Hochman JS (2008) Cardiogenic shock: current concepts and improving outcomes. Circulation 117:686–697
8. Ponzini B, Werdan K, Buerke M (2004) Cardiogenic shock: pathophysiology, clinics, therapeutic options and perspectives. Internist (Berlin) 45:284–295
9. Cooper HA, Panza JA (2013) Cardiogenic shock. Cardiol Clin 31:367–380
10. de Vree L, Swagemakers JG, Groegers JP, Dubois-Arbouw WI et al (1997) Out-of-hospital cardiac arrest in the 1990s: a population-based study in the Maastricht area on incidence, characteristics and survival. J Am Coll Cardiol 30:1500–1505
11. Atwood C, Eisenberg MS, Herlitz J, Rea TD (2005) Incidence of EMS-treated out-of-hospital cardiac arrest in Europe. Resuscitation 67:75–80
12. Dumas F, Cariou A, Manzo-Silberman S et al (2010) Immediate percutaneous coronary intervention is associated with better survival after out-of-hospital cardiac arrest: insights from the PROCAT (Parisian Region Out of Hospital Cardiac Arrest) registry. Circ Cardiovasc Interv 3:200–207
13. Berdowski J, Berg RA, Tijssen JG, Koster RW (2010) Global incidences of out-of-hospital cardiac arrest and survival rates: Systematic review of 67 prospective studies. Resuscitation 81:1479–1487
14. Wong MK, Morrison LJ, Qiu F et al (2014) Trends in short- and long-term survival among out-of-hospital cardiac arrest patients alive at hospital arrival. Circulation 130:1883–1890
15. Smith K, Andrew E, Lijovic M et al (2015) Quality of life and functional outcomes 12 months after out-of-hospital cardiac arrest. Circulation 131:174–181
16. Goldberger ZD, Chan PS, Berg RA et al (2012) Duration of resuscitation efforts and survival after in-hospital cardiac arrest: an observational study. Lancet 380:1473–1481
17. Klock W, Cummins RO, Chamberlain D et al (1997) Early defibrillation: an advisory statement from the Advanced Life Support Working Group of the International Liaison Committee on Resuscitation. Circulation 95:2183–2184
18. Braunwald E, Kloner RA (1982) The stunned myocardium: prolonged, postischemic ventricular dysfunction. Circulation 66:1146–1149
19. Stewart GC, Stevenson LW (2011) Keeping left ventricular assist device acceleration on track. Circulation 123:1559–1568
20. Pollack JM, McPherson JA, Mooney MR et al (2014) Early cardiac catheterization is associated with improved survival in comatose survivors of cardiac arrest without STEMI. Resuscitation 85:88–95
21. Nolan JP, Soar J, Carluo A et al (2015) European Resuscitation Council and European Society of Intensive Care Medicine Guidelines for Post-resuscitation Care 2015: Section 5 of the European Resuscitation Council Guidelines for Resuscitation 2015. Resuscitation 95:202–222
22. Adrie C, Laurent L, Monchi M et al (2004) Postresuscitation disease after cardiac arrest: a sepsis-like syndrome? Curr Opin Crit Care 10:208–212
23. Neumar RW, Nolan JP, Adrie C et al (2008) Post-cardiac arrest syndrome: epidemiology, pathophysiology, treatment, and prognostication. A consensus statement from the International Liaison Committee on Resuscitation (American Heart Association, Australian and New Zealand Council on Resuscitation, European Resuscitation Council, Heart and Stroke Foundation of Canada,

**Corresponding address**

L. C. Napp, MD
Cardiac Arrest Center, Acute and Advanced Heart Failure Unit, Department of Cardiology and Angiology, Hannover Medical School, Carl-Neuberg-Str. 1, 30625 Hannover, Germany
napp.christian@mh-hannover.de

**Compliance with ethical guidelines**

**Conflict of interest**  L. C. Napp received travel support outside this work from Abbott, Abiomed, Bayer, Biotronik, Boston Scientific, Cordis, Lilly, Medtronic, Pfizer, Servier and Volcano, and lecture honoraria from Maquet. C. Kuhn received lecture honoraria from Maquet and J. Bauersachs received lecture honoraria from Abiomed.

This article does not contain any studies with human participants or animals performed by any of the authors.

**Open Access** This article is distributed under the terms of the Creative Commons Attribution 4.0 International License (http://creativecommons.org/licenses/by/4.0/), which permits unrestricted use, distribution, and reproduction in any medium, provided you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons license, and indicate if changes were made.

41 | Herz 1 · 2017
71. Aso S, Matsui H, Fushimi K, Yasunaga H (2016) In-hospital mortality and successful weaning from venoarterial extracorporeal membrane oxygenation: analysis of 5,263 patients using a national inpatient database in Japan. Crit Care 20:80
72. Muller G, Flecher E, Lebrotton G et al (2016) The ENCOURAGE mortality risk score and analysis of long-term outcomes after VA-ECMO for acute myocardial infarction with cardiogenic shock. Intensive Care Med 42:370–378
73. Schmidt M, Burrell A, Roberts L et al (2015) Predicting survival after ECMO for refractory cardiogenic shock: the survival after veno-arterial-ECMO (SAVE)-score. Eur Heart J 36:2246–2256
74. Chen YS, Chao A, Yu HY et al (2003) Analysis and results of prolonged resuscitation in cardiac arrest patients rescued by extracorporeal membrane oxygenation. J Am Coll Cardiol 41:197–203
75. Massetti M, Tasle M, Le Page O et al (2005) Back from irreversibility: extracorporeal life support for prolonged cardiac arrest. Ann Thorac Surg 79:178–184
76. Sung K, Lee YT, Park PW et al (2006) Improved survival after cardiac arrest using emergent autoprimeing percutaneous cardiopulmonary support. Ann Thorac Surg 82:651–656
77. Chen YS, Lin JW, Yu HY et al (2008) 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 372:554–561
78. Kagawa E, Inoue I, Kawagoe T et al (2010) Assessment of outcomes and differences between in- and out-of-hospital cardiac arrest patients treated with cardiopulmonary resuscitation using extracorporeal life support. Resuscitation 81:968–973
79. Le Guen M, Nicolas-Robin A, Carreira S et al (2011) Extracorporeal life support following out-of-hospital refractory cardiac arrest. Crit Care 15:829
80. Avalli L, Maggioni E, Formica F et al (2012) Favourable survival of in-hospital compared to out-of-hospital refractory cardiac arrest patients treated with extracorporeal membrane oxygenation: an Italian tertiary care centre experience. Resuscitation 83:579–583
81. Chung SY, Sheu JJ, Lin YJ et al (2012) Outcome of patients with profound cardiogenic shock after cardiopulmonary resuscitation and prompt extracorporeal membrane oxygenation support. A single-center observational study. Circ J 76:1385–1392
82. Haneya A, Philipp A, Diez C et al (2012) A 5-year experience with cardiopulmonary resuscitation using extracorporeal life support in non-postcardiotomy patients with cardiac arrest. Resuscitation 83:1331–1337
83. Fagnoul D, Taccone FS, Belhaj A et al (2013) Extracorporeal life support associated with hypothermia and normoxemia in refractory cardiac arrest. Resuscitation 84:1519–1524
84. Leick J, Liebetrut C, Szardien S et al (2013) Door-to-implantation time of extracorporeal life support systems predicts mortality in patients without out-of-hospital cardiac arrest. Clin Res Cardiol 102:661–669
85. Stub D, Bernard S, Pellegrino V et al (2015) Refractory cardiac arrest treated with mechanical CPR, hypothermia, ECMO and early reperfusion (the CHEER trial). Resuscitation 86:88–94
86. Jung C, Janssen K, Kaluza M et al (2016) Outcome predictors in cardiopulmonary resuscitation facilitated by extracorporeal membrane oxygenation. Clin Res Cardiol 105:196–205
87. Fagnoul D, Combes A, De Backer D (2014) Extracorporeal cardiopulmonary resuscitation. Curr Opin Crit Care 20:259–265
88. Belohlavek J, Kucera K, Jarkovsky J et al (2012) Hyperinvasive approach to out-of-hospital cardiac arrest using mechanical chest compression device, prehospital intraaast cooling, extracorporeal life support and early invasive assessment compared to standard of care. A randomized parallel groups comparative study proposal. "Prague OHCA study". J Transl Med 10:163

Fachnachrichten

Steigerung auf hohem Niveau
Deutscher Herzbericht 2016

Der Herzbericht stellt die deutschen Herz-Medizin ein gutes Zeugnis aus. Zwar zeigen die Statistiken, dass Herzkrankungen weiter zu den häufigsten Gründen für eine Krankenhausaufnahme zählen, jedoch überleben immer mehr Betroffene.

„Noch 1990 starben 324,8 von 100.000 Einwohnern an den häufigsten Herzkrankungen, 2014 waren es 256,1“, erklärt der Präsident der Deutschen Gesellschaft für Kardiologie, Prof. Dr. Hugo Katus (Uniklinikum Heidelberg). „Dieser Rückgang um 21,15 % dokumentiert auf eindrucksvolle Weise den Stellenwert und die Fortschritte der deutschen Herz-Medizin.“

Angeführt wird die Statistik von Krankheiten, die auf angeborene Fehlbildungen zurückgehen. Im Vergleich zu 1990 ging die Zahl der dadurch bedingten Todesfälle pro 100.000 Einwohner (Sterbeziffer) um 66,67 % zurück. Es folgen die beiden häufigsten Herzkrankungen: An einer Herzinsuffizienz starben 2014 um 33,05 % weniger Patienten als 1990, bei Patienten mit koronaren Herzkrankungen (Angina Pectoris, Herzinfarkt) um 31,02 %. „Wegen der Erkrankungshäufigkeit haben die Entwicklungen bei diesen beiden Krankheitsbildern wesentlich zur reduzierten Gesamt-Sterblichkeit bei Herzerkrankungen beigetragen“, so Prof. Katus.

„Besonders erfreulich ist, dass selbst auf hohem Niveau noch Verbesserungen erzielt werden konnten“, zieht Prof. Katus Bilanz. So zeigt sich, dass die Sterbeziffer der häufigsten Herzkrankheiten 2014 um 4,76 % unter dem Wert von 2013 liegt – ein Trend, der sich bei nahezu allen Erkrankungsformen zeigt: Bei Fehlbildungen sank die Sterbeziffer von 2013 auf 2014 um 16,67 %, bei den koronaren Herzkrankungen um 6,46 %, bei Herzinsuffizienz um 3,17 % und bei den Rhythmusstörungen um 2,16 %. Lediglich bei den Herzklappen-Krankheiten blieb die Sterbeziffer mit 19,7 bzw. 19,8 praktisch konstant.

Quelle: Deutscher Herzbericht / Deutsche Gesellschaft für Kardiologie
Weitere Infos: www.dgk.org
Berlin/Düsseldorf, 25.1.2017
Hier steht eine Anzeige.

(Springer)