Extracorporeal membranous oxygenation support for acute fulminant myocarditis: analysis of a single center’s experience

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Abstract

Objectives: Acute fulminant myocarditis (AFM) is a disease category that is easily neglected. Circulatory mechanical support is sometimes required for this devastating condition. We analyzed our experience in managing AFM with mechanical circulatory support.

Methods: We applied extracorporeal membrane oxygenation (ECMO) as a first-line rescue for AFM. The diagnosis was mainly derived from clinical results and biopsy.

Results: Seventy-five patients were enrolled in the age range of 29.6 ± 18.6 years and the pediatric group (<18 years) comprised 32% (n = 24) of our patient group. Thirty-five patients (47%) underwent cardiopulmonary resuscitation (CPR) before ECMO. The indication for ECMO included high inotropic support 69% (n = 54) and continuous CPR at ECMO setup 31% (n = 23). The ECMO duration was 171 ± 121 h. Survival to discharge was 64% (n = 48), 61% in adult group, and 70.8% in pediatric group. Six patients were later bridged to ventricular assist device use (5 left ventricular assist device (LVAD) and 1 bi-ventricular assist device (BVAD)) but three died of multiple-organ failure. Three patients (4%) underwent heart transplantation and all of them survived to discharge. Resuscitation did not have a significant factor for survival. Only two patients (3%) developed late mortality due to a cardiac event.

Conclusions: AFM still carries high mortality rates in spite of advanced mechanical support. Most of the survivors did not require transplantation and could return to good lifestyle. Due to its simplicity and effectiveness, ECMO can be a first-line tool to rescue this group of patients.

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Keywords: Extracorporeal membranous oxygenator; Acute myocarditis; Fulminant myocarditis

1. Introduction

With intensive studies and increased recognition of this entity of disease, acute myocarditis remains a challenge for all clinicians because of its wide range of symptoms, unpredictable clinical course, and high mortality if not diagnosed in time [1,2]. When conventional therapy failed to support the patients and the failing myocardium to recovery, cardiovascular collapse or even death ensues. Mechanical circulatory support sometimes is required under these critical circumstances. Due to the limited availability and high cost of the implantable ventricular assist device (VAD), we applied extracorporeal membrane oxygenation (ECMO) as a first-line mechanical support when conventional therapy failed. The present study was designed to evaluate the survival outcomes and complications of the group of patients with acute myocarditis rescued with ECMO and to try to identify the risk.

2. Material and methods

2.1. Patient population

In a retrospective chart review from 1994 to 2009, 78 consecutive patients initially diagnosed with acute/fulminant myocarditis required mechanical circulatory support for their critical condition. All patients had presented with acute cardiac failure or arrhythmia and a recent history of flu-like illness without previous history of heart disease. Three of them were finally diagnosed as pheochromocytoma after detailed investigation [3]. They were excluded from the study because of a different etiology. Finally, 75 patients were enrolled in the present study. They received maximal medical therapy, including ventilator support, diuretics, vasodilators, high-dose catecholamine, and even intraaortic balloon pump (IABP). The present study was approved by the Institutional Review Board.

2.2. Indication and timing of ECMO

As mentioned in the previous studies [4,5] patients who experienced profound, rapidly progressive ventricular
dysfunction with persistent low blood pressure (systolic blood pressure <80 mmHg for adults, <60 mmHg for children, and <50 mmHg for infants), as measured by arterial lines, oliguria, (<0.5 ml kg⁻¹ h⁻¹), and frequent ventricular premature beats for at least 4 h under inotropic equivalents (IE, µg kg⁻¹ min⁻¹ = dopamine + dobutamine + 100 × epinephrine-epinephrine + 100 × norepinephrine + 100 × isoproterenol + 15 × milrinone), >40 µg kg⁻¹ min⁻¹ support was considered as the criterion for ECMO implantation. Patients requiring continuous cardiopulmonary resuscitation (CPR) with external cardiac massage were definitely considered for ECMO support.

### 2.3. Device and management

The ECMO circuit consisted of a centrifugal pump, a hollow fiber membrane oxygenator, and two thin-walled cannula (Medtronic Inc., Anaheim, CA, USA), all of which were heparin-bonded bioactive surfaces. Peripheral femoral cannulation by open Selderinger method was preferred for those >30 kg. Neck cannulation was reserved for those <30 kg. Central cannulation through median sternotomy was only preferred if there was difficulty in peripheral approach or required left-side decompression.

Low-dose heparin was infused to maintain activated clotting time (ACT) at 180–200 s. The pump flow started around 50–75 ml kg⁻¹ min⁻¹ and was adjusted according to the hemodynamic status and the status of myocardial recoverability. Inotropes did not taper immediately after ECMO because an abrupt decrease of the inotropes might lead to myocardial stunning and result in lung edema. Left-side heart decompensation might be indicated through left atrium (LA) or left ventricle (LV) if there is persistent lung edema, disappearance of arterial pulsatility, or non-opening of aortic valve for more than 48–72 h. The bridging tube between arterial and venous circuit was not applied during the ECMO support, and was reconnected to the circuit only when the possibility of weaning was controversial. A distal reperfusion catheter sometimes might be required to prevent distal limb ischemia.

Weaning was usually not attempted in the initial 48 h. The dose of inotropic agents was slowly tapered off according to the tissue perfusion and hemodynamic status. The ECMO was weaned by decreasing the pump flow when left ventricular ejection fraction (LVEF) improved more than 30% and IE was <20 µg kg⁻¹ min⁻¹ with acceptable hemodynamic conditions. Heparin dose should be increased to maintain higher ACT. Decannulation was considered if the hemodynamic condition could be maintained under ECMO flow around 500 ml min⁻¹ with IE <20 µg kg⁻¹ min⁻¹. In case of controversy in weaning and decannulation, the bridge tube was connected to the circuit and the weaning duration would extend to several hours to ensure the status of myocardial recovery.

### 2.4. Data collection and statistical analysis

All demographic data (including age, gender, and body surface area) and clinical variables (pre-ECMO clinical status, laboratory chemistry, pathology, and echocardiography) were compared between survivors and non-survivors. The values are presented as the mean ± standard deviation (SD). Laboratory findings were compared between the two groups using the Student’s t-test for normally distributed variables or the Mann—Whitney U-test for other variables. To compare the proportions of patients, Fisher’s exact test was performed. Comparisons of data using all these statistical tests were performed using Sigma Stat version 11.0 (SPSS, Chicago, IL, USA). All statistical tests were two-sided and significance was defined as $p < 0.05$. The receiver operating characteristic (ROC) curve was used to confirm the predicting power of the chosen variables.

### 3. Results

#### 3.1. Patient demographic data and pathological diagnosis

The basic data are shown in Table 1. The diagnosis was mainly clinically oriented according to the definition from Leiberman et al. [6]. The mean age of our patients is 29.6 ± 18.6 years with 29 of them being males (39%). There were 24 (32%) pediatric patients (18 years old) in the present study.

Endomyocardial biopsy to prove the diagnosis was performed in 50 patients (67%). Since the symptom of acute myocarditis developed dramatically, only six patients received biopsy before ECMO support, and biopsy was performed during ECMO support in 40 patients and after ECMO removal in four patients. Endomyocardial biopsy was more frequently performed (24/28, 86%) in patients with transthoracic approach (n = 28). However, pathological findings confirmed the myocarditis diagnosis only in 43 patients (86% of patients with biopsy).

Endomyocardial biopsy was performed less in the pediatric group except in patients who required transthoracic ECMO implantation (4/24, 17%). There was no increased morbidity or mortality when the patient underwent biopsy during the acute phase of myocarditis.

#### 3.2. Pre-ECMO status

Due to hemodynamic instability, 35 patients had experienced CPR before ECMO set up (47%), of which 23 were set up

<table>
<thead>
<tr>
<th>Variable</th>
<th>Total (n = 75)</th>
<th>Survivors (n = 48)</th>
<th>Nonsurvivors (n = 27)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yrs</td>
<td>29.7 ± 18.7</td>
<td>27.5 ± 17.7</td>
<td>33.5 ± 20.0</td>
<td>0.18</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td>0.32</td>
</tr>
<tr>
<td>Female</td>
<td>46</td>
<td>27</td>
<td>19</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>29</td>
<td>21</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>BSA (kg/m²)</td>
<td>1.4 ± 0.4</td>
<td>1.4 ± 0.3</td>
<td>1.43 ± 0.4</td>
<td>0.92</td>
</tr>
</tbody>
</table>

BSA: body surface area; CAD: coronary artery disease; CKD: chronic kidney disease; COPD: chronic obstructive pulmonary disease; CVA: cerebral vascular accident; DM: diabetes mellitus; ECMO: extracorporeal membrane oxygenation.
ECMO in status of continuous CPR (E-CPR, n = 23, 31% of study group). IABP was inserted in 23 patients (31% of AFM group, all adults) for hemodynamic support before ECMO rescue. All patients required ventilator support before ECMO support. The mean systolic blood pressure was low (75.5 ± 26.7 mmHg) with high central venous pressure (18.7 ± 5.6 mmHg) under high IE (164.1 ± 126.5 μg kg⁻¹ min⁻¹) support before ECMO. The available LVEF by transthoracic echocardiography was 34 ± 13%. No one had renal or hepatic dysfunction before AFM episodes, but 31 patients (41%) needed hemodialysis for fluid overload or anuria after ECMO set up.

The pre-ECMO data including cardiac enzyme, hepatic biochemistry, hematology, and lactate and renal indicators are demonstrated in Table 2. The data revealed elevated lactate, hepatic enzyme, and creatine kinase levels, indicating acute hemodynamic collapse with hypoperfusion. For cardiac enzyme, troponin T (TnT) was used in the first five patients, 21.3 ± 6.8 ng ml⁻¹, and troponin-I (Tn-I) in the remaining patients, 40.75 ± 30.45 ng ml⁻¹. A significant elevation of the myocardial markers (Table 2) demonstrated the severity of myocardial damage before ECMO.

Due to the rapid deterioration of hemodynamics in most patients, only six adult patients underwent right heart catheterization and myocardial biopsy before ECMO in the early years. The white count and platelet were found to have significant differences between the survivors and non-survivors (Table 2). It might be due to the sampling or statistical bias or the more severe condition in the non-survivors. However, it did not reflect similar phenomena in the lactate data (Table 2).

3.3. ECMO status

After ECMO implantation, according to the recommendation for acute myocarditis by McNamara et al. [7] most patients (n = 69, 91%) received intravenous immunoglobulin 2 g kg⁻¹. Eight patients received IABP after ECMO implantation to create the arterial pulsatility, increase the coronary blood flow, and to increase the cardiac output. Since some patients lost the arterial pulsatility and even developed lung edema, several invasive procedures were performed to decrease the left ventricular distension (Fig. 1). The situation usually represented more depressed left ventricular function. Totally, 23 patients were required to undergo decompression procedure for left heart distension or lung edema, seven with LV drainage, one with atrial septal defect (ASD) creation, and 15 with LA drainage. For patients with LV drain (n = 7), two patients survived to bridge to LVAD and then received transplantation. None of this group of patients (with LV drainage) was able to wean with native heart recovery. For the patient with ASD creation, he was successfully weaned and survived with a later atrial septal defect occluder implantation.

For patients with LA drain delivery (n = 15), eight patients survived with one patient receiving transplantation during the LA setting (Fig. 1), in which five patients recovered from acute myocarditis, weaned from ECMO, and survived to discharge. Four of them were bridged to VAD (3 LVAD and 1 BVAD), but two patients expired due to multiple-organ failure.
and two successfully recovered and survived to discharge. In the view of recoverability without death/transplantation, patients with ECMO without left-side drain had better recoverability of myocardium rate than those with left-side drain (37/52 vs 8/23, \( p = 0.005 \)).

Seventeen patients received another drainage cannula either to the right atrium, left atrium, or to both. Thirteen patients shifted the arterial cannula from peripheral to ascending aorta for better perfusion. There were 11 patients surviving to discharge in this subgroup and requiring additional drainage. Two patients received transthoracic ECMO implantation initially and all survived to hospital discharge.

3.4. Renal complications

Thirty-one patients (41.3%) received hemodialysis after ECMO support even though all of them did not have abnormal renal function before the AFM episode. After weaning off ECMO, six patients needed continuous dialysis for 2–3 weeks. One patient received heart and renal transplantation because of prolonged resuscitation. Interestingly, her native kidney recovered function 5 months after the transplantation even though her donor kidney lost its function. None of our survivors developed elevated renal marker (creatinine >2.0 mg dl\(^{-1}\)) or required long-term dialysis. When comparing the hemodialysis and non-dialysis groups, we found the need for hemodialysis after ECMO set up as a risk factor for mortality (\( p = 0.0069 \)).

3.5. Neurological complications

Sixteen patients (21%) had neurological complications, and seven (9%) were considered to be related to ECMO. The manifestation was either cerebral infarction (\( n = 4 \)) or hemorrhage (\( n = 3 \)). Embolus from the hypokinetic left ventricle was suspected to be the cause of infarction. Cerebral hemorrhage might be due to prolonged ACT. Among these patients with neurological complication, two patients survived to be discharged with mild sequel and leading independent normal lifestyle.

Regarding the other nine patients whose complications were considered not to be related to ECMO, most of them might have been due to prolonged CPR before ECMO implantation (>20 min) and one was a case of enterovirus -71 infection with neurological involvement.

3.6. Respiratory complications

Severe respiratory insufficiency did not develop in survivors except for the infant with enterovirus infection. The need for long-term ventilation of the baby was due to the neurological complications of the enterovirus infection but weaning was successful 6 months later. The mean intubation period in survivors, excluding the baby with enterovirus encephalitis was 14.5 ± 10.6 days.

3.7. Hemostatic complications

There were 21 (28%) bleeding complications. In peripheral approach group, six patients were found bleeding from the cannulation sites. All of them could be controlled by either re-exploration of the cut site, by wound compression, or by skin wound sutures.

ECMO shifting from peripheral to transthoracic mode was performed in 28 patients and 15 of them (53%) needed re-exploration for hemostasis. Nine of these 21 patients died of multiple-organ failure.

3.8. Weaning and survival rate

Fifty patients (66%) were weaned off mechanical support successfully but only 48 survived to be discharged with the survival rate of 64%. Three patients (4%) in the study group underwent heart transplantation and both of them survived to discharge. For adult patients, the survival rate was 60.8%; for pediatric patients, the survival rate was 70.8%. The difference between adult and pediatric group was not statistically significant (\( p = 0.45 \)).

3.9. Follow-up

Forty-eight patients survived to discharge (64%). The LVEF showed significant improvement than the initial echocardiography except in one patient. His myocardial function did not have significant improvement and remained around 35%. A year later, he had a sudden death at home, which was believed to be of cardiac origin. Another girl expired during exercise 4 years later, which was also considered to be of cardiac origin. She had an uneventful recovery course during ECMO with borderline cardiac function being 40–45% of LVEF.

The mean LVEF was about 57 ± 6% for the survivors at 1-year follow-up when compared with the LVEF (34 ± 13%) under very high doses of inotropic support before ECMO implantation. They all remained in functional class I without or with minimal medication during the clinical follow-up period of 66.8 ± 45.7 months.

3.10. Factors predicting the recoverability of myocardium and survival (Table 3)

Several factors in the study group demonstrated difference between survivors and non-survivors, including location of ECMO set up, dialysis, and Tn levels (Table 3). Serial serum biochemical marker data showed gradual reduction in levels, but they did not have any correlation with survival except in that of Tn. Initial Tn levels were elevated. The absolute level of Tn could not differentiate patients who would survive or not, since any resuscitation may dramatically increase the level. However, when we looked at the trend of the Tn levels, we found that there is significant increased mortality when the Tn-I level did not reach its peak within 48 h (\( p = 0.042 \)). In other words, when the Tn levels did not decrease in 48 h after the ECMO implantation, the chance of survival is reduced. As shown in Fig. 2A and B, the ROC curve showed a fair, good predictive value on the timing of the peak of the Tn-I level.

This finding may suggest that initially high Tn values do not necessarily indicate irreversible damage of the myocardium, and the reduction of Tn release in 48 h with ECMO rescue is a good indicator for survival. The persistent elevation of Tn certainly was considered as a poor outcome predictor.
4. Discussion

Acute myocarditis is still a challenging disease entity, especially for those requiring mechanical circulatory support. The manifestation of the group of patients may deteriorate rapidly and unexpectedly in several hours. To our knowledge, this is the largest single center experience to report the result of ECMO support on patients with acute myocarditis. For acute/fulminant myocarditis, ECMO could offer acceptable survival rates. The alertness and awareness of deterioration of AFM before resuscitation and early mechanical support may be helpful for better outcome. Transplantation is only reserved for three cases (4%).

Our result was comparable with those of the previous reports with the survival-to-hospital discharge rate of 64% (n = 48) [8—12]. Survival was related to several patient and ECMO factors. Tn peaks beyond 48 h after ECMO implantation, renal failure requiring hemodialysis under ECMO support, and thrombolic event during ECMO support will all increase the incidence of mortality. Interestingly, CPR is not the factor related to high mortality.

When the patient with fulminant myocarditis is refractory to maximal medical therapy, immediate mechanical circulatory support should be available. Because of the easy and rapid set up of ECMO, it should be the first choice for a patient suffering from hemodynamic collapse when compared with the more complicated and time-consuming VAD, especially in the scenario of CPR. ECMO also acts as a convenient tool and gives the flexibility to perform its implantation in other hospitals and then transfer the patient back to our unit. ECMO can also provide right ventricular support because most of the patients suffering from acute myocarditis needed BVAD for circulatory support due to biventricular failure. ECMO can also serve as a screening tool to select the more suitable candidate for long-term VAD use when physicians cannot determine if the patient had severe neurological deficit after prolonged CPR. Reports from Leprince et al. [11] also support our idea that femoral ECMO is as efficient as BVAD in supporting adult patient with fulminant myocarditis and is the best first-line treatment in these patients.

In our series, the inotropics dosage (IE), pre-ECMO CPR, or CPR during ECMO set up, did not influence the survival rate. There is no difference in the survival rate between the CPR group or non-CPR group (59% and 60%). This is contradictory to our original expectation. This leads us to believe that the initial damage of the myocardium may not be detrimental of the final recovery if proper and aggressive treatment is intervened.

As reported in the previous literature, we also found that the appearance of renal failure requiring hemodialysis and neurological complication will increase mortality [12—15]. Renal function is the result of impaired end-organ perfusion due to a failing heart. When patients require hemodialysis, the survival rate is only 45% when compared to those who do not need hemodialysis (77%). Intensive efforts to increase the end-organ perfusion with avoidance of renal toxic substance may preserve the renal function and decrease mortality.

Neurological deficit is also an important factor that increases the mortality. Except for ischemic encephalopathy

![Table 3. Variables during ECMO in patients with myocarditis.](https://academic.oup.com/ejcts/article-abstract/40/3/682/479231)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Total (n = 75)</th>
<th>Survivors (n = 48)</th>
<th>Nonsurvivors (n = 27)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>E-CPR</td>
<td>23</td>
<td>14</td>
<td>9</td>
<td>0.80</td>
</tr>
<tr>
<td>ECMO durations (hr)</td>
<td>171.5 ± 121</td>
<td>158.6 ± 96.2</td>
<td>194.2 ± 152.4</td>
<td>0.22</td>
</tr>
<tr>
<td>ECMO mode</td>
<td></td>
<td></td>
<td></td>
<td>0.21</td>
</tr>
<tr>
<td>Non-transthoracic</td>
<td>47</td>
<td>33</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>Transthoracic</td>
<td>28</td>
<td>15</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>ECMO set up in other centers</td>
<td>18</td>
<td>7</td>
<td>11</td>
<td>0.02</td>
</tr>
<tr>
<td>IE 1 day after ECMO setup (µg/kg/min)</td>
<td>28.16 ± 27.87</td>
<td>29.23 ± 28.56</td>
<td>32.20 ± 27.13</td>
<td>0.75</td>
</tr>
<tr>
<td>Dialysis</td>
<td>31</td>
<td>14</td>
<td>17</td>
<td>0.007</td>
</tr>
<tr>
<td>Tn-I level peak within 48 h after ECMO s</td>
<td>45/65</td>
<td>33/40</td>
<td>12/25</td>
<td>0.002</td>
</tr>
</tbody>
</table>

E-CPR: ECMO done under continuous cardiopulmonary; IE: inotropic equivalent; resuscitation; Tn-I: troponin-I.

![A](https://academic.oup.com/ejcts/article-abstract/40/3/682/479231)

Fig. 2. (A) The relationship between survive-to-discharge and the peak day of troponin-I level. (B) Receiver operating characteristic (ROC) curves for the peak day of troponin-I predicting the possibility of survive-to-discharge. The area under curve (AUC) is 0.73 (fair-to-good ROC predictive power).

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Neurological deficit is also an important factor that increases the mortality. Except for ischemic encephalopathy
resulting from prolonged CPR, the thromboembolic event composed largely of this complication category. Although ECMO-related thrombus formation occurs due to the contact of blood with the synthetic material, poor left ventricular function still plays a cardinal role. When left ventricular ejection fraction is severely reduced, the blood in the chamber is more static than usual. Even under continuous heparin infusion, thrombus from the hypokinetic left ventricle is highly suspected as the origin of infarction. The current mechanical support device will all increase the load on the left ventricle and impede the opening of the aortic valve, thereby increasing the incidence of thrombus formation in the left ventricle. Therefore, LV decompression procedure, through either LA or LA drainage, or RA drainage with ASD creation should be attempted when distended non-contractile LV without arterial pulsatility is developed. This procedure not only decreases the incidence of overextension of the left ventricle, but may also reduce the incidence of infarction.

The mode of ECMO, either transthoracic or peripheral (femoral) did not influence the outcome of our patients. This is the same as that reported by Rajagopal et al. [9] in 2010. In patients with fulminant myocarditis, it is the low cardiac output leading to end-organ hypoperfusion, ventricular distention, and thus thrombus formation due to blood stasis that caused the late mortality after ECMO resuscitation. No matter which mode we choose, the purpose is to increase the cardiac output and resume better organ perfusion. There are several methods of transthoracic shifting of ECMO, and no optimal method has been recommended. However, there is a significant higher mortality (75%) and lower recovery rate when patients receive LV drainage. This may be due to the ventricularotomy for tube placement and may lead to future development of arrhythmias or ventricular dysfunction.

According to our experience, it seems that there may be two subgroups of clinical courses in AFM with ECMO: one requires left drain and the other does not (Fig. 1). The subgroup of requiring drain might mean a more depressed left ventricular function, and the additional ventricular drain could achieve a similar survival rate. Whether VAD should or be converted better and which route should be drained still needs further investigation and evidence.

In the previous study of Chen et al. [5], the data suggested that the trend of Tn levels might have the potential to act as a predictor of the recovery of the stunned myocardium in FM. The initial cardiac enzyme level did not correlate with the myocardial recovery. This is the same for the Tn levels, though the levels of Tn might be thought to indicate more severe myocardial damage. In our study, we find the trend of the timing of peak Tn level will be an important predictor for myocardial recovery. If the Tn level did not reached its peak in 48 h, in other words, if the Tn-I level did not decrease 72 h after ECMO implantation, there is a statistically significant increase in mortality (p = 0.0037). The change in CK-MB is less remarkable and the level usually fluctuates resulting in poor clinical correlation when compared with Tn-I level.

5. Conclusion

AFM with hemodynamic collapse is still associated with high mortality even under maximal medical treatment. Most of the survivors did not require transplantation and could return to a good lifestyle. Because of its rapidity and simplicity to re-establish the circulatory system, ECMO is the good treatment of choice for these patients. Under the support of ECMO, patients get the chance for myocardium recovery or the potential to receive VAD implantation or even heart transplantation. The duration for the Tn-I to reach its peak can be used as a prognostic factor for myocardium recovery.

References


Appendix A. Conference discussion

Dr D. Loaisance (Paris, France): You have a huge experience, probably one of the largest in the world.

One comment: you are doing better with ECMO than was previously published with VADs. Nevertheless there is still a significant mortality. Acute fulminant myocarditis is a very heterogeneous syndrome. In this group, there is giant cell myocarditis, the treatment of which is very difficult. My question is, are the
patients who died on ECMO, patients with giant cell acute myocarditis? Can you elaborate on the histological findings? Did you perform any myocardial biopsies?

Dr Hsu: We have carried out endomyocardial biopsy in 50 patients, most of whom showed lymphocytic infiltration of the myocardium. There is only one patient in our series who showed suspicious findings of giant cell myocarditis, which was in the early period of our series. The patient was put on mechanical support.

Because of the hemodynamic instability of our patients, not every patient received endomyocardial biopsy during ECMO support, especially the pediatric group. There was only one suspicious case of giant cell myocarditis in a patient who subsequently died. After our experience with this series, we think that giant cell myocarditis needs a prolonged period of mechanical circulatory support.

Dr F. Beyersdorf (Freiburg, Germany): Interestingly, you have also shown that there is a difference if you have left ventricular venting via the left atrium or the left ventricle. Could you elaborate on that?

It is easy to understand that it’s better when you vent the left ventricle, but there you had ASD, LA drainage and LV drainage.

Dr Hsu: First, I have to mention that the site of drainage is chosen by surgeon’s preference. There is no strict guideline in our hospital for where or when left-side drainage will be done. The indication is shown in the previous slide.

And this is the data that we reviewed from our experience. We think that if the patient needs left-side drainage, it means they have more depressed ventricular function and more extensive myocardial injury. When we look further into this patient group, the LV drain seems to have a bad impact but we don’t know why. We may say that these patients might have the most extensive myocardial injury and the most extensive myocardial necrosis of all our patients with acute myocarditis.

But recently we had two patients where a different treatment was applied. These patients with acute myocarditis had frothy sputum from the endotracheal tube, loss of arterial pulsatility, and almost no opening of the aortic valve. The surgeon then considered left ventricular drainage. Of course, we don’t know whether the left drain would have had such a bad result.

However, we opted for a different treatment. We put another venous cannula through the right internal jugular vein to the right ventricle. We wanted to provide more drainage of the right side and to decrease the flow to the lung. This is our speculation, to decrease the flow to the lung and then to drain the left side.

And both of these patients were finally weaned successfully from ECMO. The x-ray, the frothy sputum and the aortic valve opening all improved after this right-sided drainage.

Dr F. Formica (Monza, Italy): Just one question. You report that about 46% of your patients experienced cardiopulmonary resuscitation. But I think you didn’t mention the mean time of cardiopulmonary resuscitation. Can you comment on this? Also, do you have any contraindication according to the time of the cardiopulmonary resuscitation?

Dr Hsu: Usually in our hospital we have a group of patients, as well as technicians and surgeons, on stand by for ECMO insertion. When there is cardiopulmonary resuscitation, and they experience prolonged resuscitation, insertion of ECMO may be considered.

The duration of the resuscitation was not a contraindication. If the resuscitation is effected in the ICU setting, for example, there is an arterial line and every cardiothoracic compression could produce a very good arterial line waveform. When you compress the heart you can produce arterial pressure on the right-hand side of about 80/60.

We had one patient resuscitated for more than one hour and then we inserted the ECMO and the patient was awake the next day. So I think the problem is not in the duration of resuscitation, but in its effectiveness.

Of course, in the emergency department situation, monitoring is not as good and we don’t know whether the resuscitation is effective or not. In our experience, when the patient was collapsed outside the hospital for more than 5 minutes and the rescuer is not experienced in resuscitation, we would think this is a contraindication for ECMO support.