EXTRACORPOREAL MEMBRANE OXYGENATION FOR PATIENTS WITH REFRACTORY VENTRICULAR ARRHYTHMIAS

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Extracorporeal membrane oxygenation (ECMO) provides mechanical cardiopulmonary support and has been used for children with severe respiratory failure, intractable heart failure, sepsis, pulmonary hypertension, and as a bridge to heart transplantation.¹⁻⁵ The use of ECMO has not been reported in children with structurally normal hearts and medically refractory polymorphic ventricular tachycardia (VT) and ventricular fibrillation (VF).

Clinical summaries

PATIENT 1. A 3½-year-old (15 kg) girl was well until the day of admission, when she was found pale and pulseless on the

floor. A neighbor quickly initiated cardiopulmonary resuscitation (CPR), and within 5 minutes an emergency medical team arrived. The initial rhythm strip revealed VF. She was intubated, defibrillated, and transported to Children's Hospital of Philadelphia. CPR was continued throughout the transport. On arrival in the emergency department she was asystolic and received 150 µg of epinephrine and defibrillation with 30 J. The rhythm after attempted cardioversion was polymorphic VT. She subsequently received multiple doses of lidocaine, bretylium, magnesium, calcium, bicarbonate, and asynchronous cardioversion leading to brief periods of a perfusing rhythm, only to be followed by polymorphic VT and VF (Fig 1). There were no electrolyte abnormalities. The chest radiograph showed a normal heart size and an echocardiogram during a brief period of sinus rhythm revealed good biventricular function. The initial arterial blood gas examination revealed a pH of 6.95, PaCO₂ 49 mm Hg, PaO₂ 60 mm Hg, and a base deficit of 20. Her head was packed in ice for the entire resuscitation. Because of the recalcitrant nature of the arrhythmia, the right carotid artery and right internal jugular vein were cannulated and she was supported with venoarterial ECMO with resolution of the VT/VF. The estimated time of CPR was 50 minutes. The patient was transferred to the cardiac intensive care unit while being supported by ECMO (flow: 65 mL · kg⁻¹ · min⁻¹ at 37°C) in addition to infusions of lidocaine and esmolol. An echocardiogram 2

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Fig 1. Rhythm strip (V_1) with an initial perfusing rhythm followed by polymorphic VT.

hours after the cardiac arrest revealed markedly depressed left ventricular function, consistent with myocardial stunning. Left ventricular function recovered to normal levels, and she was weaned from ECMO and decannulated 48 hours later. The electrocardiogram was consistent with long QT syndrome (QTc interval 480 ms and abnormal T waves). A magnetic resonance imaging scan showed no evidence of right ventricular dysplasia. Cardiac catheterization revealed a cardiac index of $3.5 \text{ L} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$ and angiography revealed normal coronary arteries. The patient underwent placement of a transvenous automatic internal cardioverter defibrillator (VENTAK MINI IV, Guidant, St Paul, Minn). She continued to have nonsustained VT controlled with a combination of mexiletine and nadolol. The patient had no end-organ damage and was evaluated by a pediatric neurologist who found no focal deficits. She was discharged home 26 days after her initial admission.

PATIENT 2. A 10-year-old boy (22 kg) status-post orthotopic heart transplantation was brought to the emergency department with exercise intolerance, fatigue, and dyspnea. The next day, under general anesthesia (propofol), he underwent cardiac catheterization for hemodynamic assessment and endomyocardial biopsy. There was a brief period of bradycardia during induction that responded to atropine (0.4 mg) and isoproterenol (20 μg). The remainder of the catheterization was uneventful. Seven endomyocardial biopsy specimens were taken from the right ventricular septum. The hemodynamic results were within normal limits with a cardiac index of $3.4 \text{ L} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$. Angiography revealed normal left ventricular function and coronary arteries. The arterial blood gas examination at the end of the procedure revealed a pH of 7.47, PaCO_2 36 mm Hg, PaO_2 250 mm Hg, base excess 3 mmol/L, and hematocrit value 32%. Catheters were removed and approximately 22 minutes later the patient began having sinus bradycardia with eventual asystole. Pulses were palpable. Atropine, epinephrine, and isoproterenol were given in succession with simultaneous chest compressions and prompt return of sinus rhythm. A 5F 110-cm transvenous balloon-pacing catheter (Bard USCI Division, Billerica, Mass) was placed in the right ventricle and acceptable thresholds were noted. Shortly thereafter, pacing spikes were noticed but no cardiac output was generated. CPR was reinstituted, ice was applied to the head, and additional doses of isoproterenol were given. The rhythm was VF, and asynchronous defibrillation was attempted with no change in rhythm. Multiple doses of lidocaine, bretylium, calcium, bicarbonate, and magnesium were given with no perfusion

rhythm elicited. There were no electrolyte abnormalities. Asynchronous defibrillation (3 J/kg) was attempted 3 additional times without interruption of the VF. During ongoing CPR (total time 1 hour), the right cervical vessels were cannulated for ECMO. Once ECMO was established, defibrillation was successful and sinus rhythm ensued. The patient was transferred to the cardiac intensive care unit while supported by ECMO (flow: $50 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ at 37°C) with consistent cardiac ejection noted at full flow. He became responsive within minutes of defibrillation. The biopsy specimens were consistent with grade 3A rejection (International Society for Heart and Lung Transplantation classification; mononuclear infiltrate, edema, and focal myocyte necrosis). The patient received pulsed steroids for 3 days and was weaned and decannulated from ECMO. No end-organ damage was detectable. On evaluation by a neurologist, his neurologic examination and electroencephalogram were within normal limits. An electrophysiologic study 12 days later revealed normal sinus and atrioventricular node function. There were no inducible ventricular arrhythmias with programmed electrical stimulation at 2 sites (right ventricular apex and outflow tract) both at baseline and on isoproterenol treatment. Follow-up biopsy specimens revealed grade 1A rejection. The patient made a complete neurologic recovery and was discharged to his home 20 days later.

Discussion. VT in children is rare, typically associated with congenital heart defects after reparative or palliative operations. Successful management of VT is usually achieved with antiarrhythmic drugs such as lidocaine, esmolol, bretylium, procainamide, or amiodarone, as well as correction of any electrolyte abnormality. Overdrive ventricular pacing can be used for VT at slower rates. VF is even less common and is one of the presumed terminal events causing sudden death in children and young adults.

VF results in ineffective perfusion of the myocardium and requires immediate defibrillation as well as the initiation of CPR. Despite antiarrhythmic treatment and multiple attempts at cardioversion, both of our patients continued to have intractable polymorphic VT degenerating into VF. Ongoing VF results in elevated end-diastolic pressures, limiting the ability to maintain myocardial perfusion during increased myocardial oxygen consumption. Prolonged VT/VF may lead to subendocardial ischemia and ventricular dysfunction. By providing biventricular support, venoarterial ECMO decompressed the heart, reduced myocardial work, and enabled standard medical therapy to terminate the arrhythmia. Both patients had significant myocardial dysfunction,

which resolved during venoarterial ECMO support. Venoarterial ECMO provides adequate cardiac output during the period of ventricular dysfunction without the need for inotropic agents that may be proarrhythmic.

A critical factor in the successful resuscitation of patients who have had a cardiac arrest is the prompt establishment of an adequate cardiac output. CPR and ECMO should be considered in venues other than the cardiac intensive care unit (ie, emergency department) for patients with a witnessed cardiac arrest (known “down-time”) who do not respond to medical management. Because of inherent delays in initiating ECMO (ie, time, priming the pump, and personnel), consideration for mechanical support should be given early if conventional medical strategies fail to control the arrhythmia. The rapid deployment of ECMO for medically refractory polymorphic VT and VF led to a successful outcome for these 2 patients.

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