Extracorporeal life support for refractory ventricular tachycardia

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Abstract: Extracorporeal life support (ECLS) is a very effective bridging therapy in patients with refractory ventricular tachycardia (VT) associated with cardiogenic shock. A moribund patient in extremis, is not amenable to optimization by standard ACC/AHA guidelines. New approaches and novel salvage techniques are necessary to improve outcomes in patients with refractory clinical settings such as malignant ventricular arrhythmias, cardiac arrest, cardiogenic shock and/or pulmonary failure until further management options are explored. Data base searches were done using key words such as ECLS, VT, cardiac arrest, VT ablation, venoarterial extra-corporeal membrane oxygenation (VA-ECMO). The use of ECLS has been described in a few case reports to facilitate VT ablation for incessant VT refractory to medical therapy. For patients with, out-of- hospital ventricular fibrillation (VF) and VT, Minnesota Resuscitation Consortium has implemented emergent advanced perfusion and reperfusion strategy, followed by coronary angiography and primary coronary intervention to improve outcome. The major indications for ECLS are cardiogenic shock related to acute myocardial infarction, myocarditis, post embolic acute cor pulmonale, drug intoxication and post cardiac arrest syndrome with the threat of multi-organ failure. ECLS permits the use of negative inotropic antiarrhythmic drug therapy, facilitates the weaning of catecholamine administration, thereby ending the vicious cycle of catecholamine driven electric storm. ECLS provides hemodynamic support during ablation procedure, while mapping and induction of VT is undertaken. ECLS provides early access to cardiac catheterization laboratory in patients with cardiac arrest due to shockable rhythm. The current evidence from literature, supports the use of ECLS to ensure adequate vital organ perfusion in patients with refractory VT. ECLS is a safe, feasible and effective therapeutic option when conventional therapies are insufficient to support cardiopulmonary function. A highly driven multidisciplinary team approach is essential to accomplish this task.

Keywords: Ventricular tachycardia (VT); venoarterial extra-corporeal membrane oxygenation (VA-ECMO); extracorporeal life support (ECLS)

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Introduction

Refractory ventricular tachycardia (VT) is a life threatening condition. VT and ventricular fibrillation (VF) are responsible for most of the sudden cardiac deaths in the United States, approximately 300,000 deaths per year (1). Frequent episodes of unstable VT could lead to hemodynamic compromise and/or deteriorate into VF; the patient may present with chest pain, anxiety, loss of consciousness, palpitations, or a complaint of

being light-headed. Further physical examination may reveal hypotension, tachypnea, change in consciousness, diaphoresis, pallor, increases in jugular venous pressure, and cannon a waves (2). The electrocardiogram is the standard method of diagnosis. A moribund patient, in an emergency state, is not amenable to optimization by standard ACC/AHA guidelines where medication, implantable devices, and ablation are usually indicated (3). When the conventional therapy fails to convert refractory VT leading to cardiogenic shock, venoarterial extra-corporeal membrane oxygenation/extracorporeal life support (VA ECMO/ECLS) can be instituted as a rescue technique to maintain hemodynamic stability and end organ perfusion until further decision or recovery (4,5).

VT classification and definitions

Different classifications and definitions have been well described in the setting of VT ablation (6,7). VT is defined as 3 or more consecutive QRS complexes at a rate 100 beats per minute or more. Sustained VT refers to VT lasting longer than 30 seconds in duration and/or requiring termination due to hemodynamic compromise in less than 30 seconds. VT storm (electrical storm, arrhythmogenic storm) is defined as 3 or more episodes of sustained VT within 24 hours each requiring intervention.

Etiology/types of VT

VT may occur in patients with or without structural heart disease. The most common cause of VT is ischemic heart disease (8). VT in patients without structural heart disease is also known as idiopathic VT; it usually originates in cardiac outflow tracts e.g., right ventricular outflow tract, pulmonary artery, left ventricular outflow tract, mitral annulus or tricuspid annulus. VT may also occur from non-outflow tract pathologies such as papillary muscles and reentrant type from fascicles (9), which are usually benign.

Malignant forms of VT are a common cause of cardiac death in patients with structural heart disease. Prior myocardial infarction is the most common cause of VT from a scar related macro reentry phenomenon. Non ischemic etiologies of VT encompass a wide variety of genetic and non-genetic disorders. Genetic syndromes with polymorphic VT (PMVT) include long QT syndrome, catecholaminergic PMVT, Brugada syndrome and short QT syndrome (9). Other causes for VT are dilated cardiomyopathy, hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy, hypertensive heart disease, congenital heart disease, noncompaction of ventricular myocardium, systemic sclerosis, Chagas disease, myotonic dystrophy type 1, electrolyte disorders, legal and illegal sympathomimetic agents, digitalis, and systemic diseases that affect the heart, or cause cardiac scarring such as systemic lupus, amyloidosis, sarcoidosis, rheumatoid arthritis, and hemochromatosis (2,8).

Therapeutic options for VT

Management options for VT include antiarrhythmic drugs, implantable cardioverter defibrillators (ICDs), catheter ablation, and antiarrhythmic surgery. In a clinically stable VT, antiarrhythmic agents are the mainstay therapy. They have a narrow therapeutic window and carry a significant risk of detrimental adverse effects including pro-arrhythmia. The arrhythmogenic risk increases with rising serum levels and careful attention must be paid to drug pharmacokinetics. Drug therapy aimed to reduce arrhythmogenic death after myocardial infarction was abandoned due to increased mortality (10). Drug therapy consisting of beta-blockers and calcium channel blockers is frequently used in the management of VT. However, these agents have limited effectiveness in the suppression of arrhythmias. Antiarrhythmic drugs such as flecainide, propafenone and amiodarone have better outcomes but also have associated side effects. Caution needs to be exercised with the use of antiarrhythmic agents especially in infants with refractory VT. Incessant VT can lead to a low cardiac output state, and antiarrhythmic drug therapy may precipitate hemodynamic collapse, thereby necessitating ECMO (11).

ICDs represent suppressive therapy, and entail recognition and termination of VT, but do not prevent recurrences of arrhythmia. Recurrent shocks may affect quality of life and survival (12) and inappropriate shocks may contribute to a new clinical problem.

Catheter guided ablation is a valuable option in abolishing VT (13,14). Radiofrequency ablation should be considered in patients with a history of syncope, very fast VT (ventricular rate >230 beats per minute), and extremely frequent ectopy (>20,000 extrasystoles per day), which may lead to premature ventricular contraction induced cardiomyopathy (5,15,16). Catheter ablation may not abolish VT in a single procedure; however, an ablation procedure may reduce VT burden, reduce ICD shocks, and may allow reduction

or discontinuation of antiarrhythmic medication (14). A wide range of mapping and ablation techniques have been described. Various mapping and ablation techniques include activation mapping/entrainment, substrate modification guided by pacemapping, late potential and abnormal electrogram ablation, scar de-channeling, and core isolation. Hemodynamic collapse may occur during VT ablation procedure. In order to accomplish successful mapping and ablation, various support devices may be needed. Percutaneous left ventricular assist devices (17), and ECMO (11) have been successfully used in the management of VT ablation procedure.

In the 1970's the surgical approach became established for the control of sustained ventricular arrhythmias. The technique involves sub-endocardial resection with or without aneurysmectomy. Surgical resection is now reserved for cases that are refractory to catheter ablation.

The use of ECMO has been described in neonates, infants and adults in the setting of incessant arrhythmias leading to progressive severe cardiogenic shock (18-21). VA ECMO provides vital organ perfusion and permits medical treatment in the setting of cardiogenic shock from refractory VT (22,23).

VA ECMO/ECLS has been used since the mid twentieth century for cardiac surgery and there have been multiple case series demonstrating its use in successful resuscitation in pulmonary, cardiac, and toxicity (including cardiac) scenarios (24-30). More recently Johnson *et al.* have studied ECLS (n=26) as a strategy for cardiac arrest in the out of hospital setting and the emergency department (31); 42% of patients had VF or pulseless VT. The investigators were able to cannulate their patients in 77±51 minutes; four survived to discharge and three (12%) were neurologically intact at six months. While the cost/benefit ratio could be questioned, it provides a benchmark for future work.

A trial of medications such as calcium channel blockers e.g., intravenous verapamil (with its known hypotensive and negative inotropic effects) can be achieved with ECMO. ECLS is especially useful in weaning catecholamine infusions and helping terminate catecholamine driven electrical storm while restoring systemic circulation. Institution of ECMO should be considered early in the management of hemodynamically unstable arrhythmias when conventional therapy fails (32). The AHA in its 2015 recommendations on adult advanced cardiovascular life support (part 7) relied on the 2015 systematic review done by the International Liaison Committee on Resuscitation (ILCOR). They compared ECLS techniques for adults who had in hospital and out of hospital cardiac arrest that received conventional CPR techniques (either mechanical or manual) in regard to return of spontaneous circulation, neurological recovery and survival. The ILCOR assessment did not directly address VT, but AHA stated that in the context of cardiac arrest, there is insufficient evidence at this time to recommend the routine use of ECLS (33). However, when it can be rapidly applied to those patients who may have experienced a reversible event, a limited period of mechanical cardiorespiratory support may be useful (33).

It must be noted that there is good evidence that early access to cardiac catheterization laboratories and the use of refractory VF/VT protocols have led to higher survival rates. In such settings ECLS could well be a successful adjunctive therapy for those whose predicted survival may be marginal (34,35). To address this subject, more studies involving large numbers of patients in multi-institutional settings must be pursued relative to the use of VA ECMO/ECLS in a life-saving context in VT scenarios.

Conclusions

Refractory VT is a medical emergency and early deployment of ECMO helps prevent persistent low cardiac output state and multi organ failure. In addition, ECMO allows administration of antiarrhythmic agents with significant negative inotropic and hypotensive effects and prevents dilation of the left ventricle. ECLS is useful in the setting of refractory VT with hemodynamic instability until palliative or corrective intervention can be carried out and its use is suggested before secondary organ damage occurs.

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

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