

# ICU Physiology in 1,000 Words: Veno-Arterial Extra-Corporeal Membrane Oxygenation (VA-ECMO)

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### Veno-Arterial Extra-Corporeal Membrane Oxygenation (VA-ECMO)

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Perhaps the most memorable patient of both my pulmonary and critical care fellowships was that of a very young woman who suffered from propofol-related infusion syndrome [PRIS]. As a consequence of PRIS, she endured multiple cardiac arrests and was placed on Veno-Arterial Extra-Corporeal Membrane Oxygenation [VA-ECMO] during cardiopulmonary resuscitation. Because of the rapid resuscitation she received from my colleagues, she survived to discharge – neuro-cognitively intact – despite spending an entire evening without cardiac activity. The details of her case may be reviewed here [1]. The aforementioned use of VA-ECMO highlights its life-preserving capability, and underscores the importance of its hemodynamic workings.

#### *Veno-Arterial versus Veno-Venous Extra-Corporeal Membrane Oxygenation*

VA-ECMO is hemodynamically distinct from veno-venous extra-corporeal membrane oxygenation [VV-ECMO]. The latter is often used as a salvage therapy for severe ARDS in patients with preserved cardiac function as reviewed here [2]. VV-ECMO removes venous blood, performs gas exchange through an external circuit and then returns the blood back to the venous system. By contrast, VA-ECMO removes venous blood, performs gas exchange through an external circuit, and then returns the ‘revitalized’ blood to the arterial tree. VA-ECMO, therefore, bypasses the heart and the lungs completely and may be used in advanced cardiac-arrest algorithms as described in the case above. Importantly, should VA-ECMO be employed in advanced cardiac failure, the hemodynamic repercussions must be anticipated; this may be accomplished using the paradigm given to us by Arthur Guyton.

#### *A Guytonian Primer*

A fantastic model – borne from the principles Arthur Guyton – was formed by Sunagawa

et al [3]; very recently, this model was further developed by Sakamoto and colleagues as a framework for understanding VA-ECMO [4]. Before turning to these advanced concepts, a basic review of venous return and cardiac function is warranted.

The circulatory system is described by the simultaneous workings of both venous return and cardiac function. With respect to the right heart, venous return is determined by a driving pressure – the mean systemic pressure [Pms]. This crucially important pressure is determined by the ‘volume status’ of the patients’ venous beds and the fraction of said volume that is ‘stressed’ [which is largely dependent upon adrenergic tone].

Additionally, venous return is determined by the resistance to venous return; many exceptional reviews exist on this topic [5-8].

Venous return, however, is only half of the cardiovascular story as valvular competence, heart rate and rhythm, contractility, diastolic properties and afterload also mediate circulatory homeostasis. All of these properties together are described by the summative cardiac function curve which is also known as the Frank-Starling curve or cardiac response curve [9-11]. The intersection of cardiac function and venous return defines the central venous pressure [12] which is one determinant of IVC volume [13].

The left heart faces an identical physiology with the pressure head for venous return originating within the pulmonary circuit [14]; pulmonary venous return is then mediated by the cardiac function of the left heart and left atrial pressure is determined at the physiological intersection thereof. As above, the slope of the cardiac function curve is determined not just by contractility, but by multiple variables one of which is afterload [10]. Thus an increase in left heart afterload will impair – shift down – the slope of the cardiac function curve.

### *Guyton and VA-ECMO*

As elaborated by the model put forth by Sakamoto et al., VA-ECMO short-circuits the normal hemodynamic path [4]. When VA-ECMO is initiated, blood is removed from the venous bed which attenuates mean systemic pressure and diminishes venous return to the right heart. Exogenous or endogenous adrenergic tone would tend to oppose this effect, but the flow at which venous blood is removed by VA-ECMO is a major determinant of the fall in venous return. Attenuated blood flow to the right heart would

mitigate each of the following: right atrial pressure, IVC volume and pulmonary blood flow. These effects are important if a pulmonary artery catheter is employed; further, change in pulmonary blood flow may alter gas exchange within the native circuit [15]. After blood passes through the external VA-ECMO circuit, it is returned to the arterial tree; the increase in arterial blood volume raises the afterload of the left ventricle [LV]. As a consequence, there is a shift of the LV cardiac function curve downwards and left atrial pressure, for any given degree of pulmonary venous return, is raised [4]. As VA-ECMO is often initiated in patients with poor, or absent LV function, this effect may be profound. However, as above, because pulmonary blood flow and pulmonary venous return are diminished, the rise in left atrial pressure is blunted. Nevertheless, acute pulmonary edema upon initiation of VA-ECMO is described [16].

Interestingly, an important prediction of the Sakamoto model is that left atrial pressure – and therefore the risk of pulmonary edema – will remain high in states of right ventricular [RV] failure, even when LV function is supra-normal [4]. As they offer no mechanistic explanation, it is tempting to speculate. While VA-ECMO lowers right atrial pressure, in RV failure it is possible that RV volume and pressure remain high despite blood being shunted to the arterial tree. Potentially, through ventricular interdependence, LV diastolic function is impaired and compounded by the rise in LV afterload imposed by VA-ECMO physiology. Thus, augmenting VA-ECMO flow will decongest the failing RV, but at the cost of raising the afterload of the LV. In such patients, then, it may be beneficial to consider a ventricular assist device [VAD] rather than ECMO [4].

The aforementioned physiology is also important when weaning a patient from VA-ECMO [17]. If RV or biventricular failure is present, it is imperative that the clinician recognize that increased venous return may have disastrous hemodynamic consequences. Such patients should have their RV function optimized with careful consideration for each of the following: volume status, inotropic support, pulmonary vasodilators, and pulmonary mechanics including the mode of and settings of mechanical ventilation [18]; consultation with a specialist in the management of pulmonary hypertension may even be beneficial.

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