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A computational framework for adjusting flow during peripheral extracorporeal membrane oxygenation to reduce differential hypoxia

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ABSTRACT

Peripheral veno-arterial extra corporeal membrane oxygenation (VA-ECMO) is an established technique for short-to-medium support of patients with severe cardiac failure. However, in patients with concomitant respiratory failure, the residual native circulation will provide deoxygenated blood to the upper body, and may cause differential hypoxemia of the heart and brain. In this paper, we present a general computational framework for the identification of differential hypoxemia risk in VA-ECMO patients. A range of different VA-ECMO patient scenarios for a patient-specific geometry and vascular resistance were simulated using transient computational fluid dynamics simulations, representing a clinically relevant range of values of stroke volume and ECMO flow. For this patient, regardless of ECMO flow rate, left ventricular stroke volumes greater than 28 mL resulted in all aortic arch branch vessels being perfused by poorly-oxygenated systemic blood sourced from the lungs. The brachiocephalic artery perfusion was almost entirely derived from blood from the left ventricle in all scenarios except for those with stroke volumes less than 5 mL. Our model therefore predicted a strong risk of differential hypoxemia in nearly all situations with some residual cardiac function for this combination of patient geometry and vascular resistance. This simulation highlights the potential value of modelling for optimising ECMO design and procedures, and for the practical utility for personalised approaches in the clinical use of ECMO.

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1. Introduction

Veno-arterial extra-corporeal membrane oxygenation (VA-ECMO) can provide short-to-medium term support of patients with severe cardiac failure, both with and without concomitant respiratory failure. The major clinical applications of VA-ECMO include patients presenting with refractory cardiogenic shock due to a severe but reversible heart condition; patients that cannot be weaned from cardio-pulmonary bypass; and as a bridge-to-decision for long-term mechanical circulatory support (Kohler et al., 2013; Martinez and Vuylsteke, 2012). VA-ECMO has also

successfully been used to treat cardiac arrest (Terri et al., 2018). As a technology whose usage has grown significantly over the last decade, where incremental improvements have been driven by experiential learning, there is considerable room for the development of theoretical modelling approaches to improve outcomes.

There are two access methods for VA-ECMO. Peripheral VA-ECMO involves cannulating the right atrium via the femoral vein (for the drain line) and the femoral artery (for the return line). It is preferred over central VA-ECMO, in which the cannulation occurs proximal to the heart via sternotomy (or the right subclavian artery) because it is faster and easier to perform, and has fewer complications (Biasi et al., 2015; Fraser et al., 2012). However, in peripheral VA-ECMO circulation, arterial blood moves retrogradely up the aorta to perfuse the upper body. In patients with residual heart function and poor lung function, this may lead to differential hypoxemia, whereby the brain and upper body are

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deoxygenated relative to the lower body (Alwardt et al., 2013). In this scenario, the left ventricle pumps poorly-oxygenated blood into the aortic arch, mixing with well-perfused blood from the ECMO return line at some point along the aorta. Aortic branch vessels upstream of this mixing zone (MZ) are therefore not as well perfused as vessels downstream.

Some investigators hypothesise that the location of this mixing zone (MZ) is dependent on the ratio of ECMO support to total circulatory blood flow (Alwardt et al., 2013; Wong et al., 2012). This was confirmed by our research team using a computational fluid dynamics model (Stevens et al., in press). This model simulated the blood flow in the aorta (from ascending aorta to the femoral bifurcation), and varied the level of ECMO support (as a function of total blood flow in the body) from 10 to 95%. It clearly showed a relationship between ECMO support level and mixing zone location. That study was a preliminary examination into this phenomenon of differential hypoxemia during VA-ECMO.

Easy identification of the MZ location, and therefore likelihood of differential hypoxemia, in individual VA-ECMO patients would aid with management. However, it is difficult to identify this location without direct measurement using angiography (Biasi et al., 2015), or aortography (Napp et al., 2015), which are both time consuming and expensive. In this paper, we propose a simulation framework, with the aim of identifying which combinations of ECMO flow rate and stroke volume are ideal to prevent differential hypoxemia in a specific patient. These personalised simulations could be used by clinicians to better identify the likelihood of differential hypoxemia in the absence of imaging technology or measures of tissue oxygenation.

2. Methods

This is a numerical study utilising computational fluid dynamics (CFD) to simulate the flow distribution and predicted oxygen perfusion of the aortic arch vessels under a range of ECMO flow/LV stroke volume conditions.

2.1. Model geometry and discretisation

The model developed in (Stevens et al., in press) was utilised for this study. Briefly, 4D-flow MRI scans were performed on a healthy subject (42 year old male). Informed consent was obtained and there was institutional ethical approval for the project. The aorta, from the aortic valve to the femoral bifurcation, along with the aortic arch branches, coeliac trunk and renal arteries, were segmented manually using ITK-SNAP (Penn Image Computing and Science Laboratory, University of Pennsylvania, PA) (Yushkevich et al., 2006). Flow extensions were added to all vessels, lengths of which were 5 times the diameter of the vessel. Then, smoothing of the geometry was performed using Meshlab (Institute of Information Science and Technologies, Pisa, Italy). The smoothed geometry was imported into ICEM CFD (ANSYS, Canonsburg, PA), and a tetrahedral based volumetric mesh consisting of 868 500 elements was generated. Increasing the number of elements to 1 024 027 resulted in a change of centreline velocity of less than 0.005 m/s, thus proving mesh independence (Stevens et al., in press). An image of the geometry is shown in Fig. 1, with the 3D geometry accessible from the online version of this article.

2.2. Simulation protocol

CFD simulations were performed using ANSYS CFX (ANSYS, Canonsburg, PA). Both ECMO and LV fluid flows were modelled as a Newtonian fluids (density 1050 kg m^{-3} , dynamic viscosity $3.5 \times 10^{-3} \text{ Pa s}$) in a fluid–fluid mixture model, with an interface

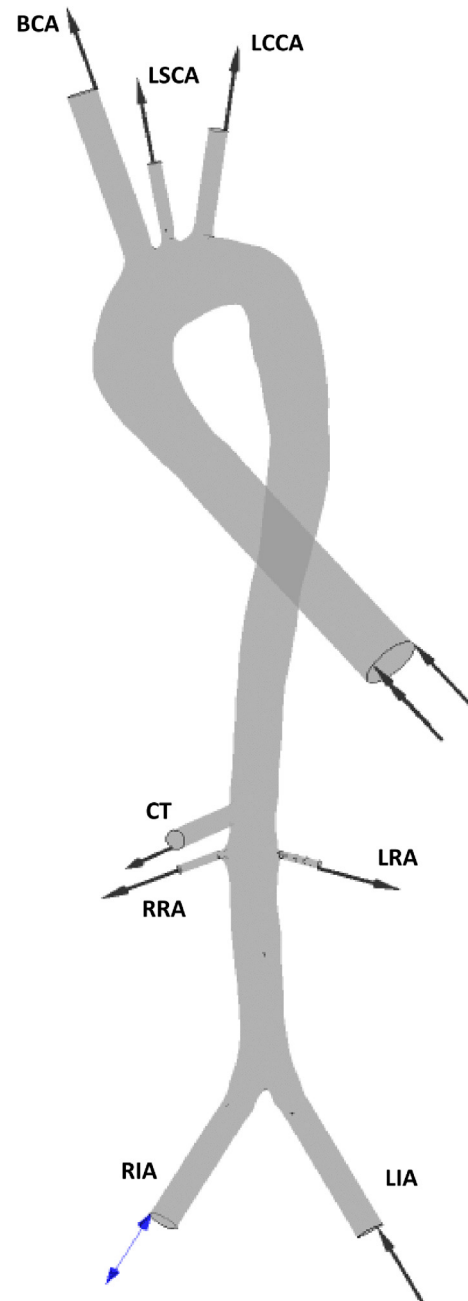


Fig. 1. Aortic geometry utilised in the simulation framework. BCA: brachiocephalic artery, LCCA: left common carotid artery, LSCA: left subclavian artery, CT: coeliac trunk, ROL: renal ostium (left), ROR: renal ostium (right), LRA: left renal artery, RRA: right renal artery.

length scale of 1 mm and a drag coefficient of 0.44 (Su et al., 2014). Flow was set as turbulent with high-resolution advection schemes and a specified blend factor of 1 ensuring that 100% of all mesh elements were solved second order accurate. The turbulence model was a first order shear-stress transport model. The phasic continuity equations for volume fractions were solved separately from the velocity and pressure.

Transient simulations using adaptive time stepping (minimum 0.0001 s, maximum 0.05 and initial timestep 0.01 s) were performed and each model was simulated for 20 cardiac cycles, ensuring periodic temporal convergence of vessel volume fractions, pressures and flow rates. The criterion for convergence was a maximum of 10^{-4} for the root mean squared residuals of the mass and momentum equations.

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