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Particle image velocimetry study of the celiac trunk hemodynamic induced by continuous-flow left ventricular assist device

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ABSTRACT

Whereas left ventricular assist device (LVAD) is the gold-standard therapy for patients with heart failure, gastrointestinal bleeding is one of the most common complications. LVAD implantation may remarkably impact aortic hemodynamics so that experimental and computational flow analyses can be used to study the disease mechanisms. Here we present an experimentally-calibrated computational model of the celiac trunk hemodynamic of a LVAD-supported patient who experienced bleeding after device implantation. Specifically, both particle image velocimetry (PIV) and echocardiography were used to measure and compare flow distributions in each branch of a phantom model of the patient abdominal aorta. Then, the distribution of wall shear stress (WSS) was estimated by computational flow analysis. At a cardiac output of 5 L/min, the highest flow division was found in the mesenteric artery (13.6% for PIV and 14.6% for echocardiography), while the left renal artery exhibited the lowest amount in the celiac trunk model (2.6% for PIV and 2.4% for echocardiography). Bland–Altman analysis demonstrated a high agreement between echocardiographic and PIV-related flow measurements, while computational flow analysis revealed that WSS was high in the LVAD graft anastomosis site and just after the ostia of both the celiac trunk and mesenteric artery. This altered shear stress distribution in the celiac trunk may lead to a flow-mediated mechanism of adverse remodeling of the von Willebrand factor and ultimately to gastrointestinal bleeding as seen clinically in this patient.

1. Introduction

Left ventricular assist device (LVAD) is the gold-standard treatment for end-stage heart failure in patients, who are not eligible for heart transplantation [1,2]. In patients with refractory heart failure, LVAD has evolved into a permanent or destination therapy [3,4]. Although outcomes after LVAD implantation have significantly improved in the last year [5], the understanding of hemodynamic alterations induced by the LVAD into the aorta, especially if support is provided by a continuous flow pump system, has to be elucidated. There are indeed significant complications and risks involved in LVAD-supported patients such as renal dysfunction [6], neurological complications and infection [7]. Nonsurgical gastrointestinal (GI) bleeding is one of the major adverse events associated with the use of a continuous-flow LVAD and presents with a range of 11–65% within the first year of LVAD placement [8,9]. During mechanical circulatory support, the development of GI is caused by an impairment of von Willebrand factor, which predisposes patients to bleeding *via* a hemodynamic-mediated mechanism [10–13]. The wall shear stress (WSS), which is the frictional force exerted by the flowing blood on the intimal surface of vessel wall, can play an important role on the mechanical demolition of von Willebrand factor. Indeed, WSS promotes arterial disease when its value falls above or below the normal, physiologic range. Acquired von Willebrand syndrome and associated development of GI was observed in both continuous-flow and pulsatile-flow LVADs [11]. Assessment of LVAD-induced shear stress by means of *in-silico* or *in-vitro* analyses may provide explanations on the pathogenetic mechanism underlying the development of bleeding, and can be used to guide prophylactic intervention in those patients at greater risk of GI.

Computational flow analysis can be adopted to characterize potentially adverse hemodynamic conditions and derive quantitative parameters such as the WSS. In LVAD-supported patients, this approach is a key tool because the mechanical support is not compatible with cardiac magnetic resonance imaging with integrated flow

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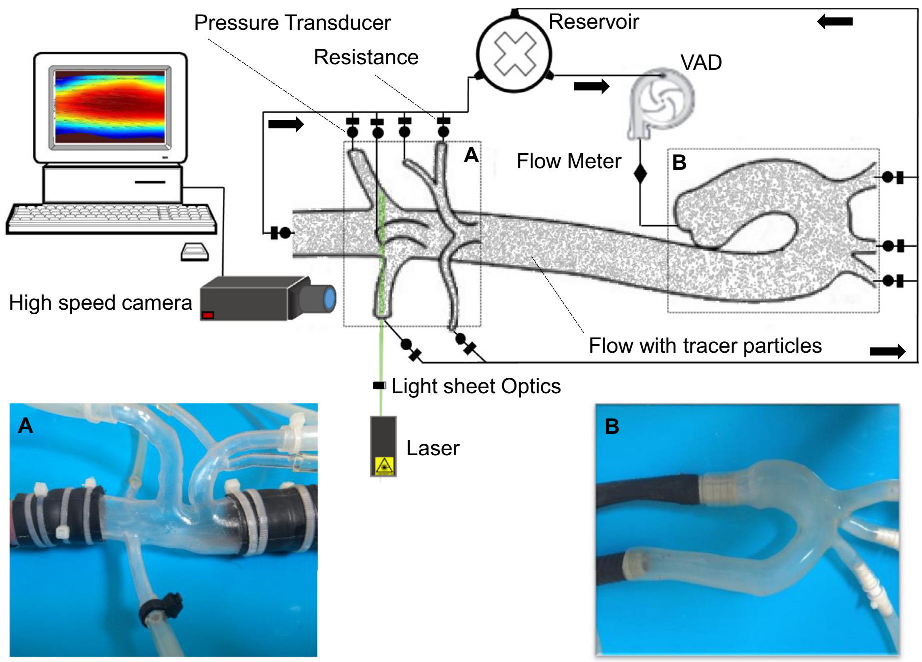

Fig. 1. Schematic of continuous-flow circuit and PIV setup showing photos of the aortic and celiac trunk models.

analysis. Computational flow analysis has previously been successful in revealing distinct features of hemodynamic disturbances in cardiovascular diseases such as aortic aneurysms and dissections [14–17]. However, modeling techniques rely on theoretical assumptions such as inflow and outflow boundary conditions, which in turn impact the WSS prediction. *In-vitro* characterization of the fluid dynamics in an aortic phantom can be achieved by experimental techniques. Particle image velocimetry (PIV) is a well-established technique to achieve high spatial resolution of the investigated fluid flow. In a different way, Doppler echocardiographic imaging has high temporal resolution but is point-based measurement method. However, experimental techniques are not reliable to estimate shear stress near the phantom wall where the velocity field tends to zero.

The purpose of our work is to develop an experimentally-calibrated computational model of the celiac trunk hemodynamic in a 3D printed model of a LVAD-supported patient complicated by GI. We believe that altered WSS magnitude in the celiac trunk wall may trigger biochemical impairments causing the development of GI. Specifically, both PIV and Doppler echocardiography were adopted to quantify and compare the outflow conditions from each branch of the celiac trunk under continuous flow LVAD setting. Then, experimentally-derived flow conditions were used as input for a computational fluid-dynamic analysis to assess the resulting WSS distribution in the abdominal aorta of the LVAD-supported patient. The difference between experimental techniques and the role of WSS distribution on the risk of GI were discussed.

2. Material and methods

To characterize potentially adverse hemodynamic conditions in the celiac trunk, the case of a 55 years old gentleman with bleeding following LVAD implantation for an ischemic dilatative cardiomyopathy was investigated. Once a phantom model of the celiac trunk anatomy was obtained, particle image velocimetry was adopted to estimate flow patterns in each branch of the abdominal aorta, and then results were compared to those obtained by echocardiographic imaging under different LVAD flow setting. Experimental flow divisions were therefore used to calibrate the computational fluid dynamic model of the LVAD-supported patient. The study was approved by the local research ethics committee, and the patient signed informed consent to the collection and use of data for research purposes.

2.1. 3D printed model and flow circuit

For the imaging of the aorta, contrast-enhanced computed-tomography angiography (CTA) following LVAD implantation was performed using a 64-slice scanner (VCT64; GE Healthcare, Milwaukee, WI, USA) with retrospective ECG gating (80% of R–R cycle). From the acquired volumetric data, the CTA scans were retro-reconstructed using a multisegment reconstruction algorithm and thin sections of 0.49–0.62 mm. To obtain the patient-specific anatomy, the aorta from the ascending to distal celiac trunk, including the splenic, gastric and hepatic vessels, through the mesenteric artery ending at the left and right renal arteries was segmented using the vascular modeling toolkit ITK (v.0.9.0; http://www.vmtk.org). The celiac trunk geometry was extrapolated from that of the entire aorta and then manufactured by 3D printing technology (Materialise NV; Leuven, Belgium) to obtain a rigid, transparent flow channel of the abdominal aorta (Fig. 1). Specifically, the TUSKXC2700T material was used as it is appropriate for water flow analysis. Surface polishing was performed to reduce inner layer roughness. Then, the 3D printed model of the celiac trunk (scale 1:1) was connected to a compliant and transparent silicone phantom model of the thoracic aorta described previously [18] (Fig. 1).

The flow circuit consisted of (i) the continuous-flow LVAD device (HeartWare Inc., Framingham, Mass, USA), (ii) the phantom model of entire aorta with the celiac trunk and (iii) the fluid reserve, all connected by silicone tubes and plastic connectors (Fig. 1). The inlet flow was imposed changing the pump rotation speed of LVAD device to investigate three different hemodynamic scenarios with cardiac output (CO) of 4, 5 and 6 L/min. In the experimental model, peripheral resistances were manually varied using adjustable valves placed distal to each outlet in order to obtain a mean pressure of 80 mmHg in the model. This pressure value was chosen in order to simulate the mean arterial pressure commonly