Medical Engineering and Physics 000 (2018) 1–12

Contents lists available at ScienceDirect

Medical Engineering and Physics

journal homepage: www.elsevier.com/locate/medengphy

An experimental and computational study of the inferior vena cava hemodynamics under respiratory-induced collapse of the infrarenal IVC

Elisabetta Tedaldi[a,b], Chiara Montanari[a,b], Kenneth I. Aycock[a], Francesco Sturla[c], Alberto Redaelli[b], Keefe B. Manning[a,d,*]

[a] Department of Biomedical Engineering, The Pennsylvania State University, USA
[b] Department of Electronics, Information and Bioengineering, Politecnico di Milano, Milano, Italy
[c] 3D and Computer Simulation Laboratory, IRCCS Policlinico San Donato, San Donato Milanese, Italy
[d] Department of Surgery, Penn State Hershey Medical Center, USA

ARTICLE INFO

Article history:
Received 21 June 2017
Revised 30 December 2017
Accepted 11 February 2018
Available online xxx

Keywords:
Inferior vena cava
Computational fluid dynamics
Particle image velocimetry
Respiratory
Compliant vessel
Fluid dynamics
Filter

ABSTRACT

Inferior vena cava (IVC) filters have been used for over five decades as an alternative to anticoagulation therapy in the treatment of venous thromboembolic disease. However, complications associated with IVC filters remain common. Though many studies have investigated blood flow in the IVC, the effects of respiration-induced IVC collapse have not been evaluated. Our hypothesis is that IVC collapse may have an influence on IVC filter performance. Therefore, we herein investigate the hemodynamics in uncollapsed and collapsed IVC configurations using *in vitro* flow experiments and computational simulations.

Particle image velocimetry (PIV) is used to measure the hemodynamics in an idealized, compliant model of the human IVC made of silicone rubber. Flow is studied under uncollapsed and collapsed scenarios, with the minor diameter of the IVC reduced by 30% in the collapsed state. Both rest and exercise flow conditions are investigated, corresponding to suprarenal flow rates of 2 lpm and 5.5 lpm, respectively. Finite element analysis simulations are carried out in a computational model of the undeformed, idealized IVC to reproduce the 30% collapse configuration and an additional 50% collapse configuration. Computational fluid dynamics (CFD) simulations are then performed to predict the flow in the uncollapsed and collapsed scenarios, and CFD results are compared to the experimental data.

The results show that the collapsed states generate a higher velocity jet at the iliac junction that propagates farther into the lumen of the vena cava in comparison to the jet generated in the uncollapsed state. Moreover, 50% collapse of the IVC causes a shift of the jet away from the IVC wall and towards the center of the vena cava lumen. The area of maximum wall shear stress occurs where the jet impacts the wall and is larger in the collapsed scenarios. Secondary flow is also more complex in the collapsed scenarios.

Interestingly, this study demonstrates that a small variation in the flow rate distribution between the right and left iliac veins induces significant variations in the flow characteristics. We speculate that asymmetries in the flow may generate unbalanced forces on the IVC wall and on placed IVC filters that could promote filter tilting and migration, although this requires further investigation. If unbalanced forces are present *in vivo*, the forces should be considered when determining the optimal placement positions and geometric features for IVC filters. Therefore, these findings motivate further investigation of the *in vivo* hemodynamics in the infrarenal IVC.

1. Introduction

Pulmonary embolism (PE) is the third most common cause of death from cardiovascular disease, after heart attack and stroke [1]. Usually associated with deep vein thrombosis (DVT), pulmonary embolism occurs when a venous thrombus embolizes and passes through the inferior vena cava (IVC) to the pulmonary arteries. Emboli occluding blood flow to one or both lungs can lead to impaired oxygenation, heart strain, and death [2].

IVC filters have been used for over five decades as an alternative to anticoagulation therapy to prevent PE. These devices are designed to intercept and trap large emboli before they reach the

* Corresponding author at: Department of Biomedical Engineering, The Pennsylvania State University, 205 Hallowell Bldg. University Park, PA 16802, USA.
E-mail address: kbm10@psu.edu (K.B. Manning).

https://doi.org/10.1016/j.medengphy.2018.02.003
1350-4533/

lungs while allowing blood to bypass freely. However, complications associated with IVC filters remain common, including failure in embolus capture, wall perforation, filter tilt or migration, and filter fracture [3].

As demonstrated by several *in vitro* [4–6] and computational [7–13] studies, an in-depth understanding of the IVC hemodynamics is crucial to assess both potential risks and benefits of IVC filters. On the one hand, *in vitro* studies [4–6] have investigated the fluid dynamics in IVCs partially occluded by a placed IVC filter and captured emboli. These studies revealed stagnant flow and recirculation regions downstream of the partially occluded IVC, which may promote further thrombus formation. On the other hand, computational studies have simulated the hemodynamics in unoccluded and partially occluded IVCs [7–12], embolus transport and capture [8], filter positioning in idealized [13] and patient-specific IVC geometries [10,13]. Two studies [14,15] combined both *in vitro* experiments and computational fluid dynamics (CFD) to deepen the understanding of the hemodynamic performance of different IVC filters. Notably, Stewart and colleagues [14] were the first to use a compliant vena cava model for particle image velocimetry (PIV) flow visualization. They compared flow velocities between PIV and CFD, using the latter also to quantify IVC wall shear stresses (WSS).

Nonetheless, the hemodynamics during respiratory-induced partial collapse of the IVC that occurs during normal breathing and Valsalva maneuver have not yet been evaluated. We hypothesize that partial collapse of the IVC may have an important influence on the IVC hemodynamics. Indeed, during inspiration, positive pressure in the abdomen causes the infrarenal IVC to diminish in size, while during expiration a negative pressure causes the infrarenal IVC to expand [16]. Valsalva has an effect similar to that of inspiration, with even greater positive pressure and, thereby, greater collapse of the IVC [16].

The IVC collapsibility index (IVC-CI) quantifies the effect of normal breathing and Valsalva on the IVC cross-sectional shape and is defined as the difference between the end-expiratory (D_{exp}) and end-inspiratory (D_{insp}) minor diameter of the IVC, divided by the D_{exp} [16–19]. Clinically, the IVC-CI is used to evaluate a patient's intravascular volume status and can range from zero (no collapse during respiration, a possible indication of hypervolemia) to unity (full collapse during breathing, a possible indication of hypovolemia) [20]. Reported healthy (i.e., asymptomatic) IVC-CI values are approximately 0.3 to 0.4 during normal breathing (e.g., see control groups in [21,22]) and $0.50 +/- 0.04$ during Valsalva [16].

We investigate the impact of different levels of IVC collapse under both resting and exercise flow conditions by performing PIV measurements on a compliant model of the human IVC accompanied by numerical CFD simulations. In all experiments and simulations, the complex dynamics of IVC collapse are simplified by approximating the vessel walls as rigid and the flow as quasi-steady. The combination of *in vitro* and computational approaches allows us to partially validate our numerical CFD results, thus providing greater confidence in the additional fields (e.g wall shear stress [WSS]) that are relatively easily extracted from CFD results, but are difficult to obtain directly through PIV.

2. Materials and methods

2.1. IVC model

In vitro experiments and CFD simulations are performed using an idealized model of the human IVC (Fig. 1(a)). The IVC model was created using SolidWorks (Dassault Systèmes, Vélizy-Villacoublay, France) and includes both iliac and renal veins (left and right, respectively), an infrarenal segment, and a suprarenal segment (Table 1). In the absence of collapse, all vessel cross sections are circular. Vessel diameters are representative of mean measurements found in the literature (Table 1). The left and right renal vein diameters are equal, but one iliac vein was made smaller than the other to emulate iliac vein compression (a common asymptomatic anatomical condition; see, e.g., [20]). Each inlet vessel length is ten times the corresponding diameter.

Table 1
Vessel diameters and nominal resting and exercise flow rates in the branches of IVC. The flow rates are representative of those reported in the literature (e.g., see [28]).

vessel	Diameter (mm)	Flow rate (LPM)	
		Rest	Exercise
Suprarenal vena cava		2.0	5.5
Left renal (LR) [43]	12.0	0.4	0.75
Right renal (RR) [43]	12.0	0.4	0.75
Infrarenal vena cava [44]	25.4	1.2	4.0
Left iliac (LI) [45]	9.3	0.6	2.0
Right iliac (RI) [45]	12.7	0.6	2.0

The model is also asymmetric to represent physiological human IVC anatomy: the right kidney is generally located more inferiorly than the left due to the larger size of the right lobe of the liver [21]. Thus, the right renal vein usually travels inferiorly moving away from the IVC while the left renal vein is approximately perpendicular to the IVC (Fig. 1(a)).

A silicone phantom of the IVC model was manufactured (Advanced Vascular Models, Seaside, CA) based on the CAD design. Because the silicone model did not perfectly match the original CAD drawing, a computed tomography (CT) scan of the silicone model was acquired by filling the silicone IVC with contrast agent. The CT data was then segmented in Avizo (Visualization Sciences Group, Burlington, MA) to extract a computational surface mesh corresponding to the silicone IVC phantom (Fig. 1(b)).

2.2. Experimental methods

A mock flow loop (Fig. 1(c)) was assembled to mimic the blood flow through the human IVC. The mock loop consists of the following components: a centrifugal pump (Cole Palmer, IL), the silicone IVC model, an acrylic chamber to contain the fluid surrounding the IVC, and a fluid reservoir (Fig. 1(c)). A bubble catcher was also placed downstream of the pump to reduce the presence of bubbles that diffuse light and interrupt the laser sheet during PIV imaging. The flow rate through the iliac and renal veins was measured using ultrasonic flow probes (Transonic System Inc., Ithaca, NY). Two clamps were used to provide resistance and were adjusted to obtain the proper flow rate through each vessel. The elevation of the fluid reservoir was adjusted to achieve a pressure of 10 mmHg at the outlet of the IVC. A Gemini PIV 15 system was used with a Nd:YAG laser, a CCD camera (4MP-HS, Powerview^{TM}, TSI, Inc) and a laser pulse synchronizer (TSI Inc., Shoreview, MN) which has been used previously to study the fluid dynamics in ventricular assist devices [22–24] and artificial heart valves [25].

Approximating blood as a Newtonian fluid [8], a Newtonian blood analog was prepared to mimic the viscosity of blood and the refractive index of the silicone model (IOR = 1.45). The fluid was composed of 46.6% water, 48% glycerin, and 5.7% sodium iodide (by weight). At 22 °C (i.e., room temperature) the kinematic viscosity of the fluid was 4.4 cSt. The flow rates imposed in the mock loop under resting and exercise conditions (Table 1) are representative of clinical measurements performed in the literature (e.g., [10]). The Reynolds numbers (Re) for the infrarenal IVC were 236 and 790 for the resting and exercise conditions, respectively (Table 1), and fall within the range of Re values investigated in previous studies (e.g., 229 [13], 600 [4–7], 360 [11], 320 [14] and 1000 [8]).

Please cite this article as: E. Tedaldi et al., An experimental and computational study of the inferior vena cava hemodynamics under respiratory-induced collapse of the infrarenal IVC, Medical Engineering and Physics (2018), https://doi.org/10.1016/j.medengphy.2018.02.003