Flow and transport phenomena in the left atrium: Assessing the risk of thrombogenesis

M. Guerrero-Hurtado[*], M. Garcia-Villalba[†], Y. Stoecker [†], E. Durán[‡], B. Maidu[§], C. Bargellini[§], A. Gonzalo[§], P. Martinez Legazpi[¶], C. Augustin[‖], P. Boyle[§], N. Akoum[§], E. McVeigh[**], J. Bermejo[*], A. M. Kahn[**], O. Flores [††], J. C. del Alamo[§]

The left atrium (LA) exhibits complex flow patterns due to its counter-flowing inlets, a valve-controlled outlet, and its appendage (LAA), a lateral protrusion of intricate, highly variable shape. Large scale flow phenomena associated with atrial filling and emptying coexist with smaller scale processes within the LAA. Non-Newtonian rheology caused by red blood cell aggregation, activation of the coagulation cascade, and thrombosis are more common in the LAA than in other cardiovascular structures. LA fluid mechanics is particularly relevant to clinical medicine in the context of atrial fibrillation (AFib), a common arrhythmia where LA cyclic contraction is replaced by an erratic weak trembling. Patients with AFib have increased risk of embolic events. Anticoagulants lower embolic risk but they also have secondary effects such as bleeding. Clinical tools used for risk stratification have modest accuracy and do not consider each patient's atrial morphology or blood flow. Computer models are emerging as potential tools to study thrombosis mechanisms and improve the clinical management of AFib patients.

In the presentation we will discuss the complex flow patterns observed in patient-specific LAs, derived from numerical simulations of blood flow in the LA of a number of patients. These simulations are either based on medical imaging alone [1], or based on a combination of medical imaging and multi-physics models for biomechanics and electrophysiology[2]. We will evaluate the effect of non-Newtonian effects in the LAA[3] and pulmonary vein inflow [4]. We will analyze the risk of thrombogenesis in the LA by using relatively simple metrics, such as the blood residence time, and more complex metrics that take into account the coagulation cascade[5]. We will also introduce a methodology (Fig. 1) to analyze the potential effects of novel anticoagulation therapies[6]

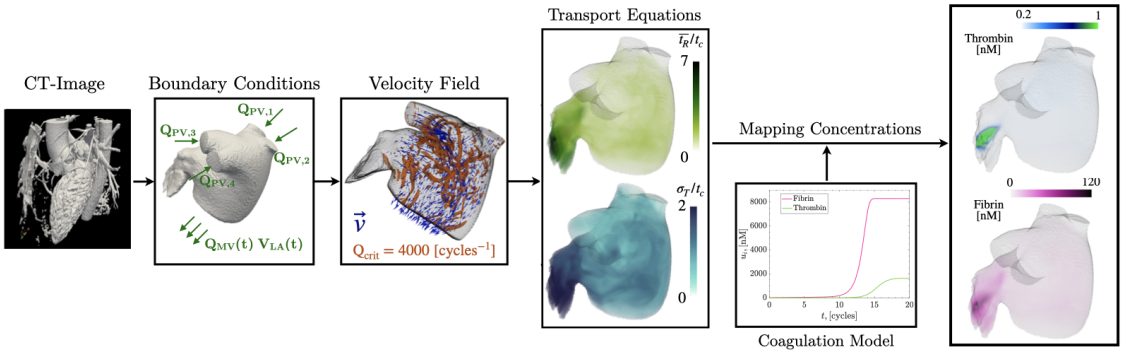

Figure 1: Workflow of methodology to assess the risk of thrombogenesis in the LA

[*]Hospital Gregorio Marañon, Madrid, Spain
[†]TU Wien, Vienna, Austria
[‡]Universidad de Málaga, Málaga, Spain
[§]University of Washington, Seattle, USA
[¶]UNED, Madrid, Spain
[‖]Medical University of Graz, Graz, Austria
[**]University of California San Diego, San Diego, USA
[††]Universidad Carlos III de Madrid, Madrid, Spain

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