Left Atrial Appendage Morphology Impacts Thrombus Formation Risks in Multi-Physics Atrial Models

Ahmed Qureshi*, Maximilian Balmus, Dmitry Nechipurenko, Fazoil Ataullakhanov, Steven Williams, Gregory Lip, David Nordsletten, Oleg Aslanidi, Adelaide de Vecchi

King’s College London, St Thomas’ Hospital, London, UK.

**Introduction:** Atrial fibrillation (AF) significantly increases the likelihood of thrombus formation (TF) depending on the morphology of the left atrial appendage (LAA): chicken wing (CW), broccoli (BR), windsock (WS) and cactus (CA). TF occurs by accumulation of fibrin, a key clotting protein, and is enhanced by blood stasis, hypercoaguability and endothelial injury, but the underlying mechanisms remain unclear. We propose a novel multi-physics approach combining computational fluid dynamics (CFD) and fibrin kinetics to characterise the relationship between fibrin gelification, left atrial (LA) flow and LAA anatomy during sinus rhythm (SR) and AF.

**Methods:** 2D finite-element LA meshes with four LAA morphologies were generated for CFD simulations. The interactions between coagulation proteins (thrombin, fibrinogen and fibrin), blood flow and viscosity (representing the gelification of the fibrin clot) were modelled during 1 minute of pulsatile LA flow. Endothelial injury was simulated by release of thrombin concentration in the LAA which catalysed the conversion of fibrinogen to fibrin. SR and AF flow patterns were simulated by varying pulmonary vein inflow velocities and prescribing mesh deformation for regular and impaired LAA contraction.

**Results:** CW and CA showed fibrin washout in SR, while WS and BR had residual fibrin concentration after 1 minute. CA had the highest mean LAA velocity in SR yet CW washed out fibrin 50% faster (Fig. 1a), demonstrating its effectiveness at preventing TF. TF was fastest in the BR morphology due to slow flow velocities in AF (Fig. 1b), and vortical structures prevented fibrin leakage during SR indicating BR had the highest risk of TF.

**Conclusion:** We determined that a combination of factors influence TF risk: 1) LAA flow velocity, 2) LAA shape and 3) flow topology i.e. vortex formation. This novel study lays the foundation for 3D patient-specific modelling of TF mechanisms which are essential for improving stroke risk assessment.

*Figure 1: Fibrin washout in CW during SR (a) and TF in BR during AF (b).*