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The role of boundary conditions and viscosity models in thrombosis modelling

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In the last two decades, blood flow modelling has experienced enormous growth not only due to the considerable increase in computational resources, but also due to its close cooperation with clinicians as the main providers of valuable medical data. Being the driving force in the development of various non-invasive diagnostic techniques, such as the multiscale one [5], the biomechanical research has now shifted its focus to underlaying mechanisms of acute vascular syndromes (e.g., acute cerebral and cardiac ischemic events), which are often of blood clotting nature, i.e., caused by blood clots (thrombi) blocking vessels. To better understand the role of various factors leading to thrombotic complications, the next most logical step is to perform appropriate computer simulations relevant to the studied problem. In this sense and given the various and often very complex biological and biochemical processes taking place in blood vessels prior to a thrombotic occlusion, model simplifications are often an essential part of the modelling process. On the other hand, even for a "simple" thrombosis model, one can discover very fast that the choice of the blood flow model and the accompanying boundary conditions have the power to change the course of a simulation and consequently its outcome.

To demonstrate the aforementioned simulation pitfalls, the present in-silico study is undertaken with the objective to assess the role of three keyfactors: injury extent, outlet pressure, and blood viscosity. For this purpose, the study employs an extended version of the thrombosis model previously introduced in [1] and inspired by the fundamental works [2–4]. The main parts of the model are shown in Fig. 1 and compared to the previous model [1], modified by emphasising the central role of platelets in the formation of the primary haemostatic plug. The thrombosis simulations are carried out in representative vascular geometries, including examples of venous (portal) as well as arterial (carotid) flows, i.e., each with varying degree of pulsatility and velocity magnitude. Aside from the factor of injury extent, which is approached by changing the trigger level and the size of the injury site, the effect of outlet boundary condi-

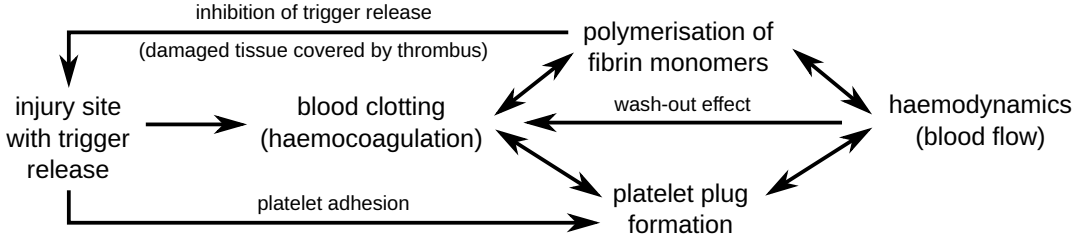

Fig. 1. Schema of the thrombosis model and its interactions with the intravascular environment

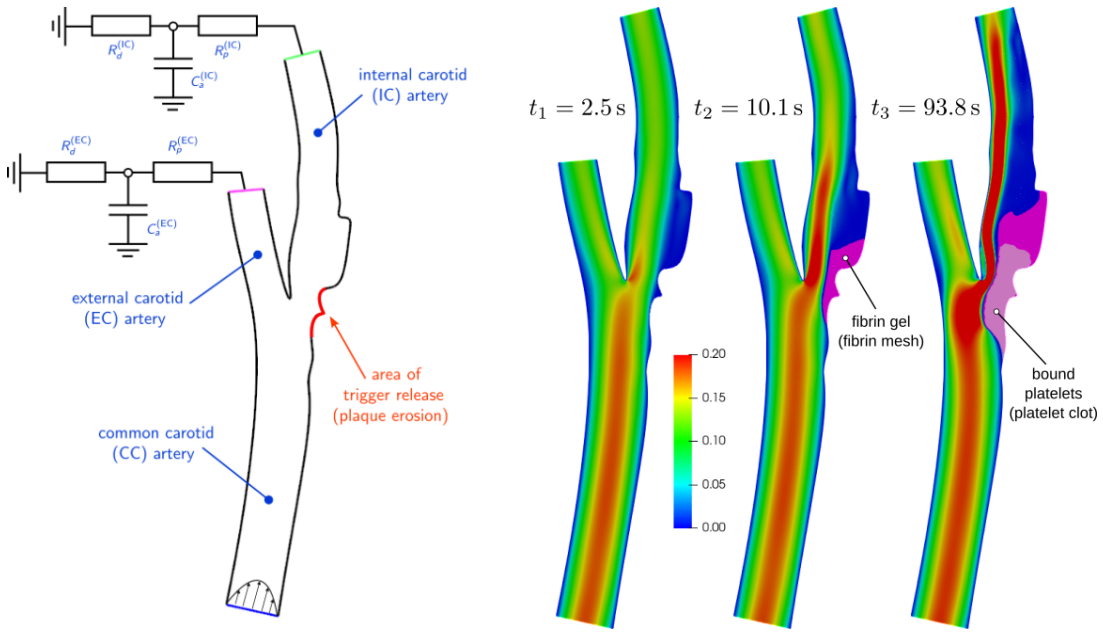

Fig. 2. (Left) Stenosed carotid bifurcation coupled with two outflow Windkessel models. (Right) Evolution of the systolic velocity field (in [m/s]) after trigger release with increasing IC blood blockage

tions is demonstrated on vascular bifurcations by assuming either a constant outlet pressure or an outflow model in the form of a Windkessel model, see Fig. 1 (left). Lastly, given the complex blood flow dynamics often seen during thrombotic events, Fig. 1 (right), the non-Newtonian effects are assessed as another keyfactor influencing the intensity of blood clotting. In this case, the Newtonian and Carreau-Yasuda models are applied with the possibility of a more advanced rheological model.

Acknowledgements

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