# ALGORITHMIC FLOW BASED GROWTH MODEL FOR CAVD

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## Introduction

Calcific Aortic Valve Disease (CAVD) is one of the most prevalent cardiovascular diseases in the world, associated with high morbidity and mortality. It is characterized by a progressive stiffening of the valve leaflets leading to stenosis and regurgitation, which results in reduced cardiac output and finally valve failure. Despite intense research, the underlying pathophysiological mechanisms of the initiation and progression of the disease are still not fully understood. This is partly because early stenosis is compensated by left ventricular hypertrophy, causing the disease to remain asymptomatic and undiagnosed for a long time. As a result, there is a lack of reliable information about the early stages of the disease. Numerical simulations can help to better understand the mechanisms behind the onset and progression of CAVD, potentially contributing to the development of pharmacological therapies. Fluid-structure simulations (FSI) are well-suited to model the complex interactions between blood flow and the immersed heart valve. These simulations can be coupled with growth & remodelling (G&R) models that aim to replicate the spatial and temporal progression of calcification lesions by incorporating the underlying biomechanical disease mechanisms. Current growth models use maximum element stretch as a criterion for growth. However, recent studies show that the fluid wall shear stresses (WSS) on the valve surface have a strong influence on the onset and progression of calcification [1]. While high laminar WSS prevail on the ventricular side of the valve, weak, oscillatory WSS act on the mechanosensitive endothelial cells on the aortic side (Fibrosa). These stresses stimulate the interstitial cells in the tissue through paracrine signalling, promoting osteoblast-like cell differentiation and thereby initiating calcification [2].

## Methods

In this work, a novel flow-based growth model is presented, which for the first time calculates the progression of calcification as a function of WSS. The growth algorithm is implemented in MATLAB and iteratively coupled with a FSI Simulation in LS-DYNA. The algorithm consists of three steps. In a first step, the WSS acting on the surface of the Fibrosa are calculated from the global element stresses for each element and timestep. In the second step, the current flow situation at each element is quantified over a complete cardiac cycle in terms of degree of oscillation using the oscillatory shear index (OSI). In the last step, the calcification progress is modelled by adjusting the stiffness of the elements depending on their OSI values and adjusting the FSI solver input files for the next iteration. The FSI Simulation includes a volumetric model of the leaflets with physiological thickness distribution to realistically capture the hemodynamic forces and flow dynamics around the valve. Furthermore, an Arbitrary Lagrangian Eulerian approach was chosen to accurately resolve the fluid-structure interface and capture the WSS on the surface.

## Results and Discussion

For the first series of simulations, the OSI threshold was set to 0.375 lying in the middle of the pathological calcification range specified in [1]. A maximum stiffening step of 10 MPa per iteration was used to model a significant increase in rigidity compared to the physiological tissue while still allowing for gradual calcification. A physiological heart valve simulation was used as the baseline for calcification modelling, focusing on key parameters such as stroke volume, geometric orifice area (GOA) and valve opening time.

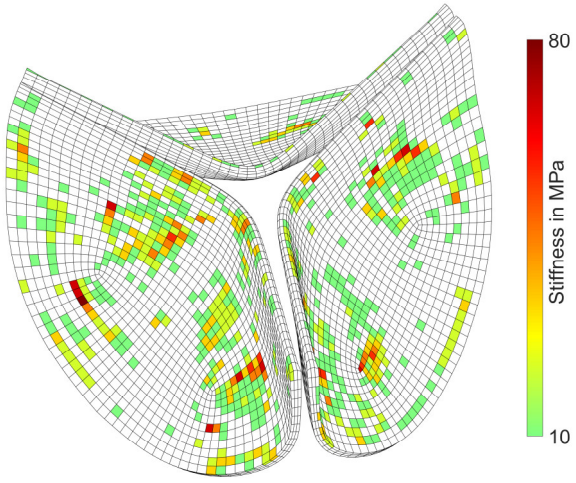


*Figure 1: Calcified elements after the first ten iterations*

The results reveal early calcification regions, with larger areas aligning with initiation points reported in literature [3]. A progressive asymmetric calcification of the Fibrosa was observed, characterized by both spatial expansion as well as gradual stiffening of already calcified regions. The progressive stiffening has visible impact on the valve dynamics and is accompanied by a reduction of GOA and stroke volume, as well as a delayed valve opening. The presented algorithm can model calcifications based on the current flow situation. The model provides a promising new approach, even though further simulation and validation are necessary.

## References

1. Hsu et al, J Bioengineering, 9(8): 393, 2022
2. Kraler et al, J European Heart, 43(7): 683-697, 2022
3. Halevi et al, J Biomech Eng, 140(10): 101008, 2018