INTRODUCTION

Intracranial aneurysms appear as sac-like outpouchings of the cerebral vasculature wall; inflated by the pressure of the blood that fills them. They are relatively common and affect up to 5% of the adult population. Fortunately, most remain asymptomatic. However, there is a small but inherent risk of rupture: 0.1% to 1% of detected aneurysms rupture every year. If rupture does occur there is a 30% to 50% chance of fatality. Consequently, if an aneurysm is detected, clinical intervention may be deemed appropriate. Therapy is currently aimed at pre-rupture detection and preventative treatment. However, interventional procedures are not without risk to the patient. The improvement and optimization of interventional techniques is an important concern for patient welfare and is necessary for rationalisation of healthcare priorities. Hence there is a need to develop methodologies to assist in identifying those ICAs most at risk of rupture. We focus on the mathematical modelling and computational simulation of ICA evolution. Models must take into consideration: (i) the biomechanics of the arterial wall; (ii) the biology of the arterial wall and (iii) the complex interplay between (i) and (ii), i.e. the mechanobiology of the arterial wall. The ultimate ambition of such models is to aid clinical diagnosis on a patient-specific basis. However, due to the significant biological complexity coupled with limited histological information such models are still in their relative infancy. Current research focuses on simulating the evolution of an ICA with an aim to yield insight into the growth and remodelling (G&R) processes that give rise to inception, enlargement, stabilisation and rupture. We present a novel Fluid-Structure-Growth computational framework for modelling aneurysm evolution.

PATIENT-SPECIFIC MODELLING OF INTRACRANIAL ANEURYSM EVOLUTION

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METHODS

Figure 1: Clinical MRI data is automatically segmented using @neufuse software. A small sidewall saccular aneurysm developing on the right internal carotid artery is identified. (b) Boundary conditions for the haemodynamic computational domain.

A selection of sidewall saccular aneurysms are identified from clinical imaging data (see Fig. 1(a)). The data is automatically segmented and centrelines are created through the vasculature. The section of vasculature that contains the aneurysm is removed and replaced with a cylindrical section (see Fig. 1b), hereon referred to as aneurysmal section, to represent an idealised section of healthy artery on which the aneurysm develops. The aneurysmal section utilises a realistic constitutive model of the arterial wall that accounts for the structural arrangement of collagen fibres in the medial and adventitial layers, the natural reference configurations in which the collagen fibres are recruited to load bearing and the mass of the elastin and collagenous constituents [1]. It is reconnected to the upstream and downstream sections of the vasculature using an automated algorithmic method [2]: boundary curves propagate along a centreline to smoothly morph the surface sections together.
Figure 2 illustrates the computational methodology. The computational modelling cycle begins with a structural analysis of the aneurysmal section to solve the equilibrium deformation field for given pressure and boundary conditions. The structural analysis quantifies the stress and stretch, and the cyclic deformation, of the ECM components and the cells (each of which may have different natural reference configurations). The (systolic) geometry of the aneurysmal section is exported to be prepared for haemodynamic analysis. To achieve physiologically realistic flow in the region where the aneurysm develops, the aneurysm geometry is integrated into a physiological geometrical domain, which is then automatically meshed with ANSYS ICEM (ANSYS Inc, Canonsburg, PA); physiological flow rate and pressure boundary conditions are applied. The flow is solved with ANSYS CFX assuming rigid boundaries for the haemodynamic domain. The haemodynamic quantities of interest, for example, WSS, WSSG are then exported and interpolated onto the nodes of the structural mesh: each node of the structural mesh contains information regarding the mechanical stimuli obtained from the haemodynamic and structural analyses. G&R algorithms simulate cells responding to the mechanical stimuli and adapting the tissue: the constitutive model of the aneurysmal tissue is updated. The structural analysis is executed to calculate the new equilibrium deformation fields. The updated geometry is exported for haemodynamic analysis. The cycle continues and as the tissue adapts an ICA evolves.

RESULTS
Initially, the degradation of elastin is prescribed to create a small outpouching of the computational domain to perturb the haemodynamic environment [3]. Subsequent elastin degradation is then linked to low WSS in this localised region of the computational domain [4], whilst the collagen fabric adapts (throughout the arterial domain) to restore its strain to the homeostatic value. Figure 2a illustrates the evolving systolic and diastolic geometries and Fig. 2b the geometry of the actual aneurysm that was removed. Interestingly, we observe that using the hypothesis that low WSS degrades elastin for patient specific haemodynamics yields qualitatively similar aneurysm geometries. Figure 3 illustrates the WSS, WSSG and pressure distributions following the evolvement of a model of an ICA on patient-specific vasculature.