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Direct numerical simulation of transitional flow in a patient-specific intracranial aneurysm

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ABSTRACT

In experiments turbulence has previously been shown to occur in intracranial aneurysms. The effects of turbulence induced oscillatory wall stresses could be of great importance in understanding aneurysm rupture. To investigate the effects of turbulence on blood flow in an intracranial aneurysm, we performed a high resolution computational fluid dynamics (CFD) simulation in a patient specific middle cerebral artery (MCA) aneurysm using a realistic, pulsatile inflow velocity. The flow showed transition to turbulence just after peak systole, before relaminarization occurred during diastole. The turbulent structures greatly affected both the frequency of change of wall shear stress (WSS) direction and WSS magnitude, which reached a maximum value of 41.5 Pa. The recorded frequencies were predominantly in the range of 1–500 Hz. The current study confirms, through properly resolved CFD simulations that turbulence can occur in intracranial aneurysms.

1. Introduction

Stroke is a leading cause of death in the Western world (Feigin, 2005). One reason for stroke is the rupture of aneurysms usually found in or near the Circle of Willis, an arterial network located at the base of the brain. It is estimated that 1–6% of the population will harbor aneurysms during their lifetime (Schievink, 1997), and that the average risk of rupture is 1–2% annually (Rinkel et al., 1998). The initiation, growth, and rupture of intracranial aneurysms is a complex and multi-factorial process. Population studies have shown that aneurysm rupture is influenced by, e.g., age, gender, smoking, alcohol consumption, hormonal factors, and a mother's age when the first child is born, cf. (Eden et al., 2008; Kongable et al., 1996; Mhurchu et al., 2001; Brisman et al., 2006; Weir, 2002; Schievink, 1997; Longstreth et al., 1994; Humphrey, 2001). The precise mechanism however is still not known. It is well-known that arteries remodel themselves according to flow conditions (Chien, 2007). For example, blood vessels thicken if blood pressure rises, is lengthened with axial loads, or increase internal diameter with high values of WSS. In addition, if the WSS exceeds a limit of 40 Pa, the endothelial cells are believed to be damaged, and an aneurysm might form (Davies et al., 1986; Fry, 1968).

Blood flow in cerebral arteries is a complex phenomenon. Establishing adequate computational flow models that are of clinical relevance has been an active area of research the last decades. Many studies focus on the relevance of nonlinear rheology (Johnston et al., 2006; Fisher and Rossmann, 2009; Kim et al., 2008; Gijsen et al., 1999; Lee and Steinman, 2007; Galdi et al., 2008), and perhaps the most active area is interaction between blood flow and elastic vessel walls (Gerbeau et al., 2005; Heil, 2004; Bazilevs et al., 2010). There has been remarkably little focus on the presence of turbulence in cerebral arteries, and the assumption of laminar flow is commonly accepted. The clinical relevance of determining whether a flow is turbulent is revealed when examining the effects of turbulent flows. A turbulent flow may produce highly increased magnitude of WSS, increased frequency at which the WSS changes direction and local pressure fluctuations. The resulting cell remodeling under such flow conditions is currently unknown.

Turbulence has previously been shown to occur both in the aorta (Khanafer et al., 2007) and in a stenosed carotid artery (Lee et al., 2008). Audible sound, which implies turbulence, has successfully been recorded from saccular aneurysms in dogs (Sekhar et al., 1990) and humans (Ferguson, 1970; Kurokawa et al., 1994). The lowest recorded Reynolds number where turbulence occurred, Re_c, in glass models of human intracranial

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