

# Chapter 28

## Computational Study of the Hemodynamics of Cerebral Aneurysm Initiation

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### ABSTRACT

*This chapter aims to present the authors' recent findings from studies on the computational biomechanics of blood flow in human arteries and its application to the hemodynamics of cerebral aneurysm initiation. They first briefly outline the techniques of computational fluid dynamics used in blood flow simulations of anatomically realistic artery models reconstructed from medical images acquired with CT or MRI. Then, the time course of the blood flow velocity field in the medical image-based model of a human internal carotid artery (ICA) is shown as a result of a pulsatile blood flow simulation with CFD techniques. Finally, the chapter presents an overview of the concept of a novel hemodynamic indicator for cerebral aneurysm initiation, the gradient oscillatory number (GON). The distribution of the GON for the medical image-based ICA model is also demonstrated.*

### INTRODUCTION

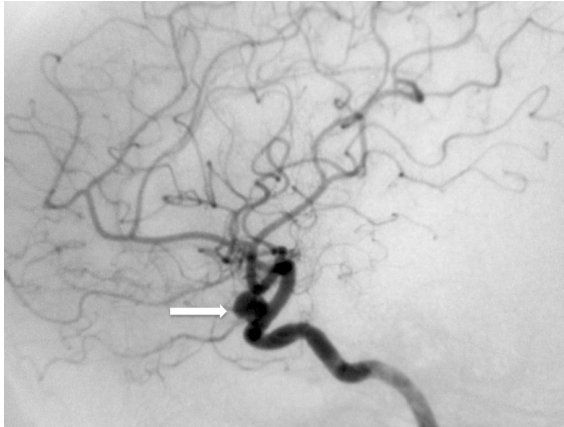
#### Background

A cerebral aneurysm is characterized by an abnormal expansion of the cerebral arterial wall, as shown in Figure 1. It is a serious pathological condition because the rupture of an aneurysm leads

to subarachnoid hemorrhage (Krex, Schackert, & Schackert, 2001; van Gijn & Rinkel, 2001). Once the cerebral aneurysm ruptures, it results in excessive bleeding into the subarachnoid space, which is the area between the arachnoid membrane and the pia mater surrounding the brain. Subarachnoid hemorrhage has a very high mortality rate between 32% and 67% (Huang & van Gelder, 2002; Schievink, 1997). Despite the risk of potentially serious consequences of cerebral aneurysm, the

DOI: 10.4018/978-1-4666-2196-1.ch028

Figure 1. Angiogram of a human internal carotid artery with an aneurysm



mechanism of its pathogenesis including initiation, growth, and rupture is still unclear.

According to a previous large-scale survey (Rinkel, Djibuti, Algra, & van Gijn, 1998), between 3.6% and 6% of the population have unruptured cerebral aneurysms. Recent developments in medical imaging techniques, such as computed tomography (CT) and magnetic resonance imaging (MRI), have enabled doctors to increasingly detect cerebral aneurysms before they rupture. Although there are some treatment options that are currently in widespread clinical use, such treatments have been known to have a non-negligible potential for complications (Raaymakers, Rinkel, Limburg, & Algra, 1998). Despite these factors, the risk of cerebral aneurysm rupture has been reported to be only approximately 1.9% annually (Rinkel et al., 1998). For these reasons, it is quite difficult for patients and doctors to make a decision on whether surgery should be performed after an aneurysm is detected. To address this problem, it is necessary to understand how cerebral aneurysms initiate and grow. Thus far, it has been widely accepted that blood flow-induced mechanical forces acting on the vessel wall, i.e., hemodynamics, play a vital role in the pathogenesis of cerebral aneurysms.

## Past Studies on the Initiation of Cerebral Aneurysm

To date, many animal models have been developed to investigate the initiation of cerebral aneurysms (AAssar, Fujiwara, Marx, Matsumoto, & Kallmes, 2003; Espinosa, Weir, & Noseworthy, 1984; Hashimoto, Handa, Nagata, & Hazama, 1984; Kamphorst, Yong-Zhong, & van Alphen, 1991; Kondo et al., 1997; Krings et al., 2003). These experimental studies reported that the initiation of cerebral aneurysms was a result of the interaction between hemodynamics and the structural degeneration of the arterial wall. At the cellular level, degeneration of endothelial cells and the medial layer was found at the initial stage of cerebral aneurysm formation (Kim, Cervós-Navarro, Kikuchi, Hashimoto, & Hazama, 1992; Miskolczi, Guterman, Flaherty, & Hopkins, 1998). Kondo et al. (1998) reported that there was an association between apoptosis of smooth muscle cells in the media and the early stage of cerebral aneurysm formation. At the molecular level, it was reported that nitric oxide (NO) and matrix metalloproteinase (MMP) activation may be important factors in the induction of arterial degenerative changes leading to aneurysm initiation (Fukuda et al., 2000; Johannning et al., 2002; Krex et al., 2001).

A number of specific hemodynamic factors, such as wall shear stress (WSS), pressure, impingement force and residence time, have been proposed as candidates responsible for the pathogenesis of cerebral aneurysms (Burlison & Turitto, 1996). Among these hemodynamic factors, WSS is the one that has received special attention in aneurysm studies (Hoi et al., 2004; Kondo et al., 1997; Rossitti, 1998), similar to hemodynamic studies on arteriosclerosis from the late 1960s (Caro, Fitzgerald, & Schroter, 1971; Fry, 1968). In animal studies by Krings et al. (2003), it was reported that there was an association between the site of aneurysm formation and areas of high WSS. They also reported the loss of the internal elastic lamina in areas of high WSS. Tateshima et al. (2003)

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