A Comparative Study of Transient Flow through Cerebral Aneurysms using CFD

S.M. Abdul Khader, Md. Zubair, Raghuvir Pai. B, V.R.K. Rao, S. Ganesh Kamath

Abstract— The recent advances in computational fluid dynamics (CFD) can be useful in observing the detailed hemodynamics in cerebral aneurysms for understanding not only their formation and rupture but also for clinical evaluation and treatment. However, important hemodynamic quantities are difficult to measure in vivo. In the present study, an approximate model of normal middle cerebral artery (MCA) along with two cases consisting broad and narrow saccular aneurysms are analyzed. The models are generated in ANSYS WORKBENCH and transient analysis is performed in ANSYS-CFX. The results obtained are compared for three cases and agree well with the available literature.

Keywords—Aneurysms, ANSYS – CFX, CFD, Pulsatile flow.

I. INTRODUCTION

COMPUTATIONAL haemodynamics has become a powerful and desirable tool in investigation of cardiovascular diseases such as atherosclerosis and aneurysm, which are influenced by hemodynamic factors [1]. Aneurysms are local dilatations or balooning of the vessel wall and most commonly occur in the base of the brain (cerebral aneurysm), or near the aorta (aortic aneurysm). These aneurysms most often are caused by arteriosclerotic diseases, infections, head injuries, alcohol and smoke abuse and due to many other reasons [2]. In the present study, main emphasis is on cerebral aneurysms especially in MCA and in circle of Willis. The most serious complications occur when aneurysm ruptures due to intra cranial bruise, subsequent recurrent bleeding, hydrocephaly and spasms in brain vessels. Aneurysms are most frequently diagnosed using visualization modalities such as CT scanning and MR imaging. Upon detection, the risk of rupture versus the risk of treatment needs to be evaluated. The main criterion in deciding whether an aneurysm needs to be

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treated is the aneurysm size [3]. Treatment options are endovascular coil embolization, surgical clipping and more recently stenting. Coil embolization consists of filling the aneurysm with a platinum coil to decrease blood circulation and promote thrombus formation in the aneurysm [4]. This procedure may be preceded by stenting of the parent artery, especially in wide-necked aneurysms. Clipping consists of placing a clip around the aneurysm neck, preventing blood from flowing into the aneurysm. The choice of treatment depends on the individual risk assessment of each aneurysm, including factors like the patient's condition and characteristics of the aneurysm like location, size and shape.

Aneurysm hemodynamics is contingent on the aneurysm geometry and its relation to the parent vessel, its volume and aspect ratio (depth/neck width) [5]. The geometry of the aneurysm can be described by several characteristic dimensions like the orifice size (l), the neck width (n), the dome width (w), the dome semi-axis height(s) and the dome height (h) as shown in the fig.1. The characterization of the shape of an aneurysm can be done by determining ratios like w/n, h/n, w/h and h/s.
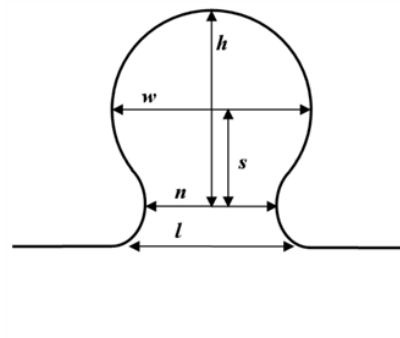

Fig. 1 Saccular aneurysm dimensions

The pathogenesis and causes for expansion and eventual rupture of saccular aneurysms are not clear. It is generally accepted that unique structural features of the cerebral vasculature contribute to the genesis of these aneurysms. A typical saccular aneurysm has a very thin tunica media and the internal elastic lamina is absent in most cases. Thus, the arterial wall is composed of only intima and adventitia. Consequently, hemodynamics factors such as blood velocity, wall shear stress, pressure, particle residence time and flow impingement, play important roles in the pathogenesis of aneurysms and thrombosis. These factors play an important

role in the outcome of endovascular and surgical treatments as measuring these hemodynamic quantities in vivo is difficult and various modeling approaches using CFD have been considered in the past.

The systematic CFD studies provide the thorough understanding of the influence of variations in the boundary conditions on the biologically important haemodynamics parameters [6]. Also such a study would help vascular surgeons to understand flow patterns associated with vascular pathology as well as to predict the hemodynamic changes induced by surgical procedures.

II. METHODOLOGY AND MODEL DESCRIPTION

A. Governing equation of CFD

The blood flow in cerebral arteries is assumed to be Newtonian, laminar and it is governed by the incompressible flow equations consisting of continuity and Navier–Stokes equations [1,2,3].

$$\nabla . u = 0 \tag{1}$$

$$\rho \left(\frac{\partial u}{\partial t} + u . \nabla u \right) = -\nabla p + \mu \nabla^2 u \tag{2}$$

Where ρ is the density, p is the pressure, μ is the viscosity and u is the velocity.

B. Model Description

The length of the MCA widely ranges from 12 to 28 mm with a mean of 20 mm and the diameter at its origin ranges from 2.5 to 4 mm with a mean of 3.35 mm [7]. In the present study, the length and diameter of the normal MCA model is considered to be 18mm and 3mm respectively as shown in fig.2. The broad neck MCA aneurysm shown in fig.3 has ratios w/n=2.5. The ratio w/n =1.5 is chosen for narrow neck MCA aneurysm as shown in fig.4. The 3D models are generated in ANSYS WORKBENCH-11.0 and meshed models are later transferred to ANSYS-CFX for further transient analysis [9].
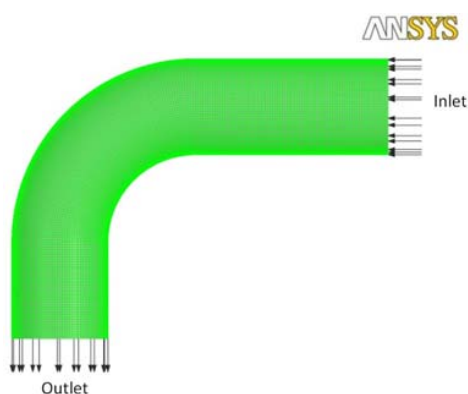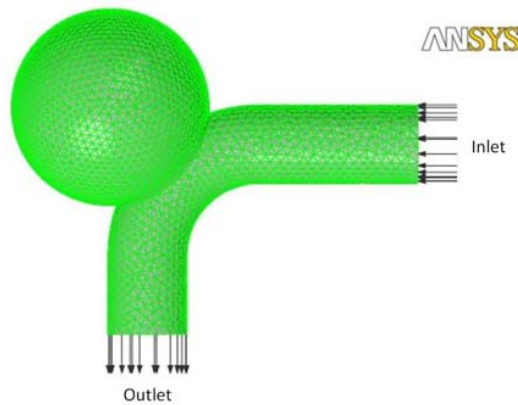

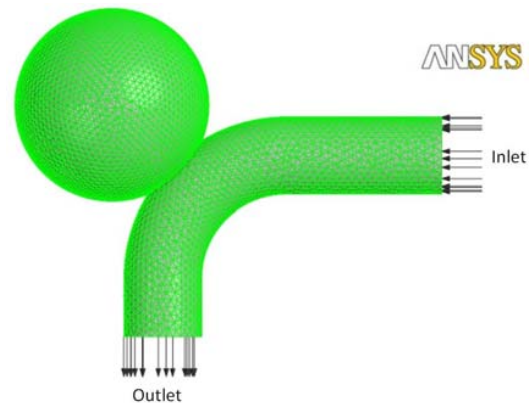Fig. 2 Normal MCA

Fig. 3 Broad neck MCA aneurysm

Fig. 4 Narrow neck MCA aneurysm

III. NUMERICAL ANALYSIS

In the present analysis, the flow is considered to be laminar, incompressible and Newtonian [1, 2]. The boundary conditions applied are: (i) a time dependent uniform velocity at inlet, (ii) a zero normal traction at outlet and (iii) no slip boundary conditions at the vessel walls [1,8,10]. The normal MCA is discritized into 300000 hexahedral elements, while narrow and broad neck MCA aneurysm are discritized into 137000 and 104000 tetrahedral elements [9].
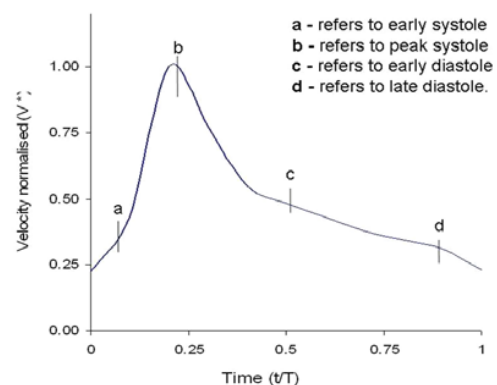

Fig. 5 Inlet velocity waveform

The arterial wall is assumed to be rigid and transient analysis is performed for four pulse cycles and each pulse cycle is divided into fifty time steps. The blood density and viscosity are specified as 1060 kg/m^3 and 0.004 Ns/m^2 respectively [8, 10]. The normalized inlet velocity waveform shown in fig.5 and other prescribed boundary conditions remains same for all the three cases.

IV. RESULTS AND DISCUSSION

The computed results are compared for three cases viz. normal MCA model, Broad Neck Aneurysm (BNA) model and Narrow Neck Aneurysms (NNA) model considering velocity profile, pressure contour and wall shear stress. The flow contour is compared at specified instants in a pulse cycle as highlighted in fig.5 and it is found to be maximum during the peak systole when inflow in maximum. It is observed that the flow is almost stagnant in sac for both BNA and NNA models for entire pulse cycle especially at the center [2,8]. Because of the bend, the flow in downstream is more turbulent leading to the formation of eddies along inner side wall for entire pulse cycle. The velocity contours at peak systole is shown in fig.6, fig.7 and fig.8 and it is observed that the formation of aneurysm has deviated the normal flow path. The extent of sac filling is more in BNA than in NNA because of the large neck size as shown in fig.7 and fig.8.
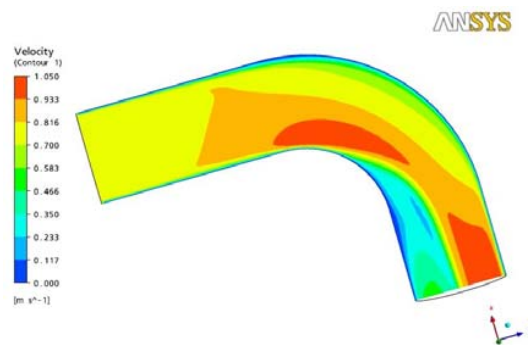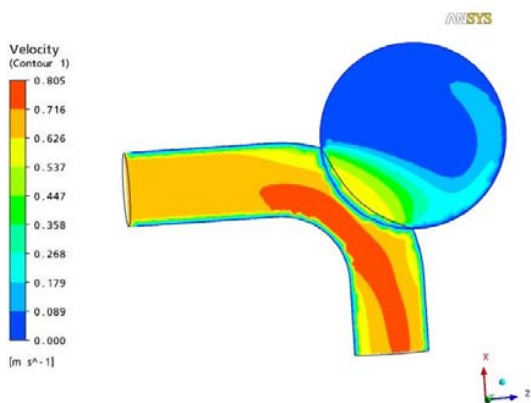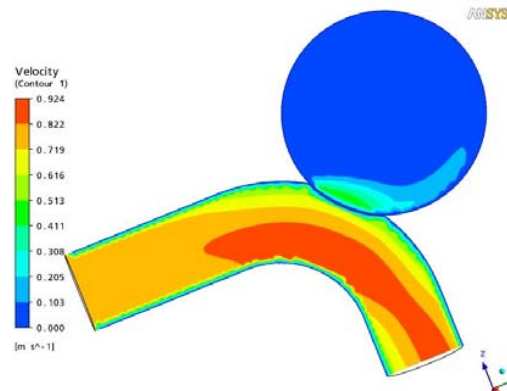

Fig. 8 Velocity contour in NNA model (Peak systole)

The pressure contours are compared which is maximum at peak systole for BNA and NNA model as shown in the fig. 9 and fig.10 respectively [11]. It is observed that the difference in sac pressure is exhibited to be more in NNA than in BNA. The effect is more localized in later and spread across the whole dome in former, because of the broad neck, the stagnation of the flow in sac is less and the neck region is more pressurized.
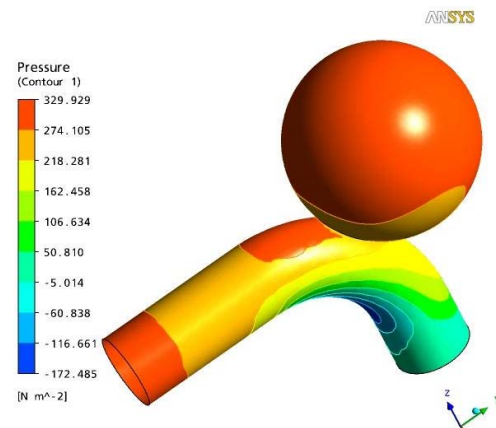

Fig. 6 Velocity contour in normal model (Peak systole)

Fig. 9 Pressure contour along wall in NNA model (Peak systole)

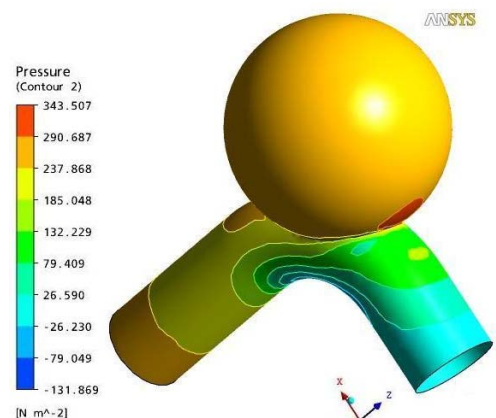Fig. 7 Velocity contour in BNA model (Peak systole)

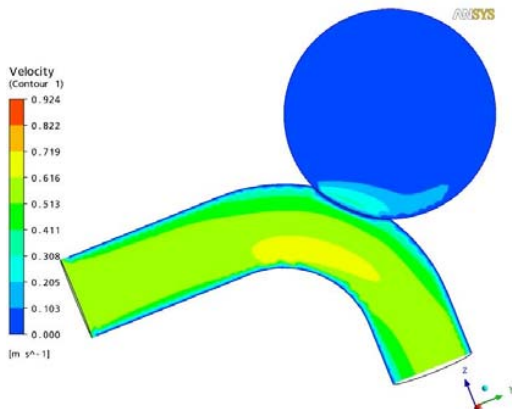Fig.10 Pressure contour along wall in BNA model (Peak systole)

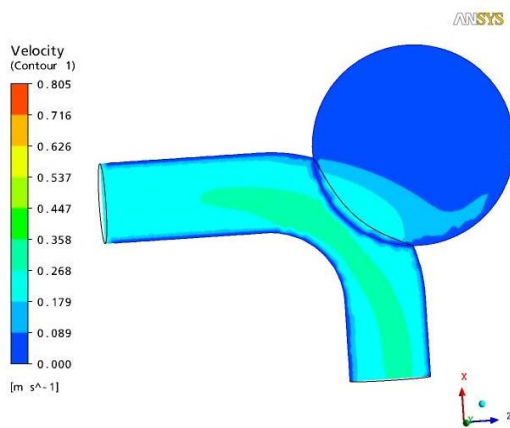Fig. 11 Velocity contour in NNA model (early diastole)

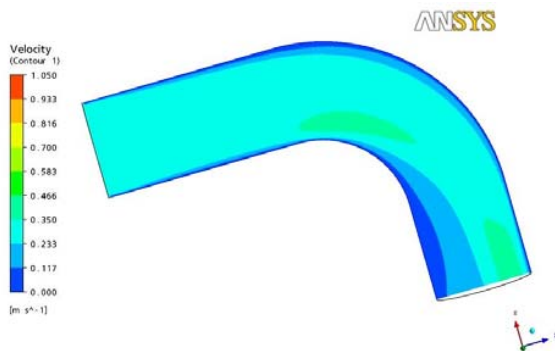Fig. 12 Velocity contour in BNA model (early diastole)

Fig.13 Velocity contour in normal model (early diastole)

Fig. 11 and fig.12 depicts the velocity contour during early diastole for NNA and BNA models. The flow pattern changes abruptly leading to turbulence in downstream. The flow in the most part of the sac appears to be stagnant and filling of sac diminishes as the neck size decreases. Also the time required for exit of the flow from the sac is less in BNA than in NNA. Due to the flow deceleration, the extent of the formation of eddies is high in normal as shown in fig.13 than both the abnormal models and it tends to increase as it reaches the end part of the pulse cycle.
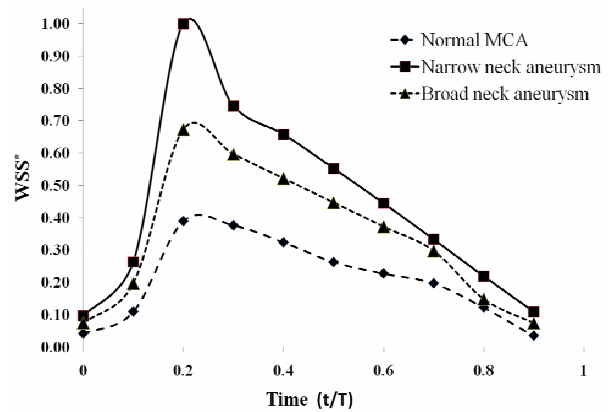

Fig. 13 Normalized WSS comparison

The significance of the evaluation of Wall Shear Stress (WSS) is high in case of saccular aneurysms, because the entire haemodynamics is altered as it varies with time due to pulsatility and the maximum value occurs generally at peak systole [11, 12]. The normalized WSS is compared for the three cases as shown in fig.14. It is observed that the variation of WSS is highest in NNA model followed by BNA and normal model and the location is not constant and varies with pulse cycle. In case of normal model, the maximum WSS is observed towards the inner bend during peak systole and it decreases with declaration of pulse. In case of BNA and NNA models also, the maximum WSS is observed along the bend. The nature of variation of WSS shows the pulsatile behavior.

V. CONCLUSIONS

The transient analysis is performed on normal MCA, NNA model and BNA model to exhibit the difference in flow pattern due to the variation in geometry. The results show that the maximum flow is observed at peak systole in all the three models. The sac filling is observed to be maximum in BNA model than NNA model and flow is nearly stagnant in the dome. The dome of NNA model is more pressurized than BNA model. During the flow deceleration, the formation of eddies is common in downstream in all the three cases. The comparison of WSS suggests that the maximum WSS is observed to be in NNA model than in BNA model.

In the present study, the numerical simulations results are compared for the changes in flow behavior for normal and diseased cases for an approximated model and agree well with the clinically observed results. In order to accomplish the realistic physiological simulations, the present methodology could be extended to realistic models. The generation of such models is possible by considering the images obtained from the various imaging modalities.

VI. NOMENCLATURE

MCA   Middle Cerebral Artery
BNA   Broad neck aneurysm

NNA   Narrow neck aneurysm
WSS *   Normalized Wall Shear Stress
           $= WSS_t / WSS_{max}$
WSS $_{max}$ Maximum WSS in time period, T
WSS $_t$ WSS at time, t

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