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Biomechanical Factors Analysis in Aneurysm

Kleiber Bessa[1], Daniel Legendre[2] and Akash Prakasan[2]
[1]Department of Environmental Sciences and Technological
Rural Federal University of Semi-Arid
[2]Institute Dante Pazzanese of Cardiology
Brazil

1. Introduction

All the cells in the body need to receive food (nutrients, metabolic products) and to dispose of waste products. The responsible system for that is cardiovascular system. It is responsible to supply food through the arteries and to return waste products through the veins for all living cells in the human body. This task is reached by a circulating fluid, the blood. The central location which all lines of supply originate from and return to is a small, very small, pump, the heart. The heart keeps the fluid in circulation. In the heart, there are two pumps, propelling blood into the pulmonary and systemic circulation and are combined into a single muscular organ to synchronously beat. Any disruption in the blood flow causes a disruption in food supply. Life is not possible without blood, but in the truth life is not possible without the circulation of blood. It must pump at all times, which it does by contracting and relaxing in a rhythmic pattern, approximately once every second, more than 86 thousand times every day, and about 2 billion times in a lifetime of 75 years, nonstop (Zamir, 2005). The blood ejected by the heart follows in the direction the arterial tree. Along the arterial tree, the arteries successively decrease in size, increase in number, undergo structural changes, and finish in arterioles that are as little as 10 μm in diameter. The structure of the artery is quite complex. The main components of the vessel wall are endothelium, smooth muscle cells, elastic tissue, collagen, and connective tissue. The arteries are targets for diseases such as atherosclerosis or aneurysms that each year claims the lives of scores of people worldwide. The cardiovascular disease may be triggered or aggravated by mechanical stimuli, such as wall stress or stretch resulting from the blood pressure, or shear stress resulting from the blood flow (Wernig and Xu, 2002). Arteries can also adapt to long-term physiological conditions by thinning or thickening the muscular layer, and altering the relative composition and organization of the various assemblies of structural proteins in process generally know as remodelling. Bessa et al. (2011) showed that occurs remodelling in tail arterial bed from normotensive and hypertensive rats. As shown in Figure 1, the internal diameter of the proximal portion of the tail artery did not differ between Wistar rats and spontaneously hypertensive rats (SHR), whereas the diameter of the intermediate and distal portions of SHR tails arteries were significant smaller than those of normotensive rats.
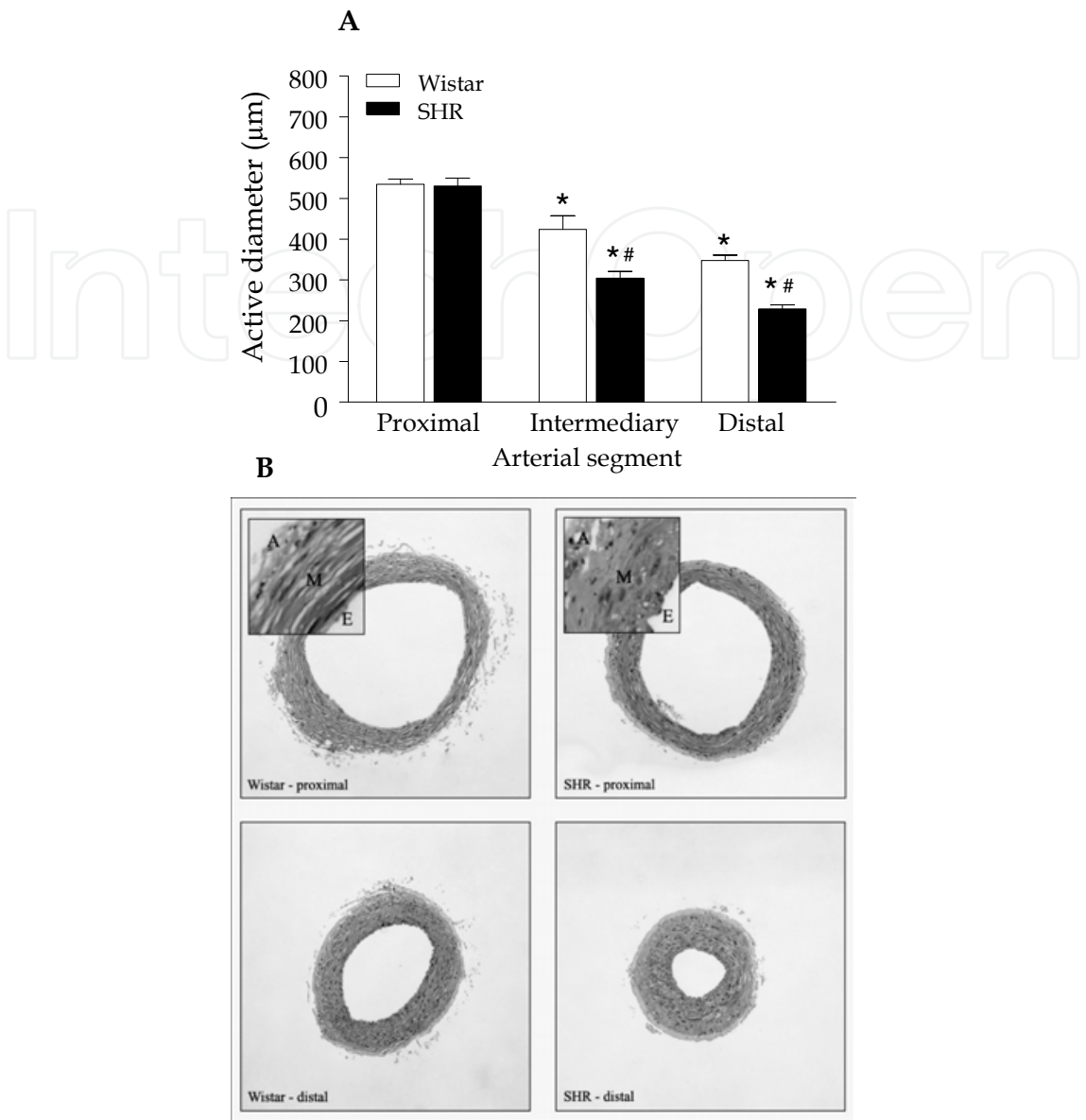
Fig. 1. *A*, Active internal diameters of tail arteries from Wistar rats (N=4animals) and spontaneously hypertensive rats (SHR, N=5 animals) fixed at a volumetric flow rate of 2.5 mL/min. *P <0.05 *vs* proximal portion. #P < 0.05 *vs* wistar rats (two-way ANOVA followed by the Bonferroni *post hoc* test). B, Photomicrograph of transverse section of proximal and distal tail artery from Wistar rats and SHR. Magnification: 10X. Inset panels in the proximal tail artery photomicrographs of Wistar and SHR are amplified images showing the endothelial (E), medium (M) and adventitial (A) layers of the artery. Extracted from Bessa et al. 2011.

An aneurysm is defined as a focal dilation of a blood vessel when compared with the original artery. Aneurysms are widening of the lumen in any artery, most commonly in the aorta for fusiform types, and in the head for saccular types. The arterial fusiform aneurysms and intrinsic stenosis are possible complications of atheroma. Saccular aneurysms can occur as complications of arterial wall trauma or infection. Intracranial aneurysms (IA), rare in childhood and adolescence, are observed in 3% to 5% of the population, with a gender ratio

of 3 women for 2 men. Abdominal aortic aneurysms (AAA) most of the affect men between 40 and 70 years old (5-7% of people older than 60) (Thiriet, 2008). Genetics and risk factors play important roles in the development of the aneurysms, but is universally accepted that biomechanical factors (including increased pressures in hypertension) also play fundamentals roles. Vascular endothelial and smooth muscle cell are constantly exposed to the biomechanical factors caused by the blood flow. Cellular responses to these biomechanical factors influence vessel wall homeostasis (Hisai, 2008). Once the aneurysm forms, the biomechanical factors caused by the pulsatile flow in the aneurysm can cause gradual expansion of it. When the wall of the distended artery fails to support the stress occurs rupture of the aneurysm. This rupture often leads to death or severe disability.

This chapter introduces analysis about pathogenesis, hemodynamic forces acting on the wall vessel that could be important factor to the origin and progression of the disease, and computational fluid dynamic (CFD) associated to the medical imaging. Thus, the interaction between pathogenesis of aneurysm, medical imaging and CFD are important to understand the development of aneurysm.

2. Pathogenesis of arterial aneurysms

2.1 General considerations

An arterial aneurysm is one of the most common vascular diseases causing disability and death. True aneurysm represents a degeneration of the artery wall with loss of structural integrity leading to gradual dilatation of all artery wall layers. Aneurysms have been reported in almost all segments of the arterial tree, but are more frequently localized at the aorta below the renal arteries. Aneurysms are most commonly diagnosed in the sixth and seventh decades of life, with a rising incidence for unknown reasons. The prevalence of aneurysms in a given population depends on the presence of risk factors associated, including older age, male gender, white race, positive family history, smoking, hypertension, hypercholesterolemia, peripheral vascular occlusive disease, and coronary artery disease. Although these risk factors are associated with increased abdominal aortic aneurysms (AAA), they may not be independent predictors and may be markers rather than causes of AAA prevalence.

Although a precise cause of AAA remains unknown, much has been learned about the pathophysiology of the aneurysmal aorta. Research has linked the development of AAA with chronic aortic wall inflammation, increased local expression of endogenous proteinases, and the degradation of structural connective tissue proteins (Shah, 1997).

Most arterial aneurysms arise at the bifurcation of major arteries, and this is also true for the intracranial location. Around 85% of all intracranial aneurysms originate from the anterior circulation. The most common location (30%–35%) is the anterior communicating artery. The prevalence of intracranial aneurysms among first-degree relatives of patients with cerebral aneurysms is higher than in the general population. The risk for a first-degree relative harbouring an aneurysm is about three to four times higher than for someone from the general population (Raaymakers 1999; Ronkainen et al. 1997).

Although the pathogenesis and etiology of cerebral aneurysms has been studied extensively, both are still poorly understood. Endogenous factors like elevated blood pressure, the special anatomy of the Circle of Willis or the effect of hemodynamic factors, particularly originating at vessel bifurcations, are all known to be involved in the growth and rupture of an aneurysm. Arteriosclerosis and inflammatory reactions, however, might also have an

impact. Exogenous factors like cigarette smoking, heavy alcohol consumption or certain medications are thought to be risk factors in the pathogenesis of an aneurysm or at least increase the risk of rupture.

2.2 Basic mechanisms

The normal aortic wall is composed of a thin endothelial lined intima, a thick elastic media dominated by vascular sooth muscle cells, and a fibrocollagenous adventitia. Medial elastin fibers and interstitial collagens are normally responsible for the tensile strength, resilience, and structural integrity of the aortic wall. Aneurysmal dilatation and rupture are due to mechanical failure of these fibrillar extracellular matrix proteins.

Basic research on the pathophysiologic processes of aortic aneurysm formation and growth has identified several putative mediators of the disease. These mediators include both bacteriologic and enzymatic agents, especially matrix metalloproteinases. Aneurysm formation and aneurysm growth represent two distinct phases of aortic aneurysm disease. Aneurysm formation encompasses the metabolic processes that cause degradation of the structural components of the aortic wall and loss of biomechanical integrity. This phase of aneurysm disease appears to be directed at the destruction of elastin within the aortic wall. Although all layers of the aortic wall may be involved, the most fundamental structural alteration that results in a loss of biomechanical integrity and aneurysm formation is degradation of adventitial elastin. The metabolic processes that cause adventitial elastin degradation are nonselective. They most likely begin in the intima or media, associated with an inflammatory cell infiltrate. Destruction of the elastin within these layers does not cause loss of the structural integrity of the aortic wall. These processes, however, ultimately invade the adventitia, cause degradation of the elastin within this layer, destroy the structural integrity of the aortic wall, and permit its pathologic expansion. Because humans are unable to synthesize and deposit elastin in the aortic wall in any detectable quantity beyond the first year of life, the degraded elastin is replaced by collagen I and III during the process of aneurysm formation. (Mesh et al., 1992) Once formed, even the smallest aneurysm represents the end-stage of aortic wall destruction, a significant alteration in the biomechanical properties of the aortic wall and a reduction in structural integrity. Aneurysm growth appears to be a self-sustaining process. Data from both human and animal aneurysm cells demonstrate a sustained increase in collagen types I and III m RNA and new collagen deposition within the aneurysm wall. These processes continue until the rate of collagen degradation exceeds the rate of deposition or the pressure per unit area of aneurysm wall exceeds the ability of the collagen fibers to withstand the load. In either of these situations, aneurysm rupture occurs.

3. Evaluation of aneurysms

3.1 Physical examination and imaging

Although most clinically significant AAA are potentially palpable during routine physical examination, the sensitivity of this technique depends on size, obesity of the patient, skill of the examiner, and focus of the examination. With physical examination alone, the diagnosis is made in 29% of AAAs 3 to 3.9 cm, 50% AAAs 4 to 4.9 cm, and 75% of AAAs 5 cm or larger (Lederle & Simel, 1999).

Several imaging modalities are available to confirm the diagnosis of AAA. Abdominal B-mode ultrasonography is the least expensive, least invasive, and most frequently used examination, particularly for initial confirmation of suspected AAA and follow up of small AAAs. Computed tomography is more expensive than ultrasound and involves exposure to radiation and intravenous contrast material, but it provides more accurate measurement of diameter, with 91% of studies showing less than 5 mm interobserver variability (Chervu et al., 1995).

3.2 Factors affecting clinical decision making

The choice between observation and prophylactic surgical repair of an AAA for an individual patient at any given time should take into account the risk for rupture under observation, the operative risk associated with repair, the patient's life expectancy, and the personal preferences of the patient. For the scope of this chapter we will only analyse the risk of rupture.

Estimates of the risk for AAA rupture are imprecise because large numbers of patients with AAAs have not been observed without intervention. Data are insufficient to develop an accurate prediction rule for AAA rupture in individual patients, which makes surgical decision making difficult. Some aspects may be considered:

- Aneurysm diameter

For the past 5 decades maximal aneurysm diameter has been the primary determinant of rupture risk. Several studies firmly established the effect of size on AAA rupture and provided a sound basis for recommending elective repair for large AAAs because of marked improvement in survival after repair. Despite differences in precise estimates, most studies show that rupture risk increase substantially with AAA diameter between 5 – 6 cm.

- Aneurysm wall stress

From a biomechanical perspective, AAA rupture occurs when the forces within a AAA exceed the wall's "bursting strength. The application of engineering principles to the analysis of actual aneurysms has only recently been possible. Multiple studies have demonstrated that finite element analysis of AAA wall stress with three dimensional CT reconstructions is better than diameter for estimating rupture risk.

- Finite element analysis

At this point the strength of the data and the size of the patient cohorts already rival or exceed that of the data initially used to determine the clinical use of aneurysm diameter to estimate rupture risk in the 1960's. The technique of using aneurysmal wall stress to predict rupture risk remains to be validated in a large multicenter cohort using a standardized, broadly applicable technique, although one such study is currently under way (Fillinger et al., 2004).

- Aneurysm shape

Clinical opinion holds that shape is important and eccentric or saccular aneurysms present a greater risk for rupture than do more diffuse fusiform aneurysms. Vorp et al. (1998) associates using computer modelling showed that wall stress is substantially increased by an asymmetric bulge in AAA. The presence of calcification in the wall may increase wall stress focally, but may not be useful as a clinical tool. The effect of intraluminal thrombus on rupture risk is also debated, with studies suggesting that thicker thrombus may increase the risk for rupture, decrease the risk for rupture, or have no effect. The practical impact of these variables on AAA rupture risk requires further study.

4. Biomechanical analysis

This section provide an overview of the fundamental basis of biomechanical factors acting on the vessel wall and their influences under endothelial cells and the development of the aneurysm, once that there are several studies showing disturbed flow conditions and unsteady turbulent stresses damage endothelial cells and may provide a first step to the degradation of the wall (Davies et al., 1984, 1995). There will be analysed biomechanical factors in intracranial aneurysm and an abdominal aortic aneurysm.

4.1 Intracranial aneurysm

At present, there is not completely satisfied the theory about the origin, growth and rupture of intracranial aneurysm. The walls of intracranial arteries exhibit the same general organization and composition of all arteries. However, in most cerebral arteries, there is no external elastic lamina. Moreover, in these arteries, the arterial walls are thin, there is less elastin, and there are "medial defects" (gaps in the muscle layer) present frequently, thus intracranial arteries are more susceptible to aneurysm formation than extracranial arteries. Other arteries show similar medial defects, particularly in renal, mesenteric, splenic, and coronary arteries. However, these defects are smaller and less frequent and the aneurysm formation is rarely (Crompton, 1966). In the literature, there are some hypothesis about the location and structural changes in the wall of the artery that contribute to development of the aneurysm. Eppinger (1887)(apud Thubrikar, 2007) showed that certain aneurysms occurred at the site of the medial defects, however, other researchers, such as, Forbus (1930), Toth et al. (1998) and Zhang et al. (2003) showed that the aneurysm could be developed through combination of degeneration of the elastic and defects in the muscular layer. Meng et al. (2007) recently demonstrated in animal models that the aneurysm initiation and development occurred at the apices of arterial bifurcations where there was high wall shear stress and aneurysm-type wall remodelling (disrupted internal elastic lamina and endothelium, thinned media and smooth muscle cells loss) at histology. These factors appear to render these arteries susceptible to a local weakening under the persistent action of hemodynamic loads, particularly in hypertension (Inci and Spetzler, 2000). The hemodynamic loads resulting from the direct impingement of the central streams at the apex of bifurcations are probably the most important factor contributing to the focal degeneration of the internal elastic membrane and the early origin the aneurysms. These forces could enlarge medial defects already present. The impingement of central axial streams results in a much greater velocity gradient and shear stress at the apex than is experienced in the main stem or branches of bifurcations. As flow is pulsatile, the peak force will be great, because there is a brief impact time and in the moment of impact the kinetic energy of the moving blood is changed to pressure energy (stagnation pressure) (Thubrikar, 2007). This extra pressure is the force responsible for focal degeneration of the internal elastic membrane and thus the cause of initiation of aneurysms at the apex. Shojima et al. (2005) reported that local rises in pressure due to flow impingement are less than 2 mmHg, which is small compared with nominal pressure levels in cerebral arteries, and concluded that dynamic pressures acting at bifurcations and on the walls of intracranial arteries may be less significant to enlargement and rupture than previously assumed. Acevedo-Bolton et al (2006) concluded that regions that continued to enlarge experienced low wall shear stress and speculated that this might be due to increased residence time of particles that degrade the aneurysm wall. There is a great interest to study the wall shear stress in aneurysms because there are many works show that it plays a role in the evolution of aneurysmal

disease. It is known that the wall shear stress is a major physiological stimulus for the vessel endothelium. The endothelial cells submitted the physiological levels of wall shear stress in the arteries [1 (10) – 7 (70) Pa (dyn/cm²)] and in the veins (0.1 (1) – 0.7 (7) Pa (dyn/cm²)] provide a physiological stimulus for the vessel endothelium contributing to selective barrier for macromolecular permeability, can influence vascular remodelling via the production of growth-promoting and –inhibiting substances, modulate hemostasis/thrombosis through the secretions of procoagulant, anticoagulant, and fibrinolytic agents, mediate inflammatory responses via the surface expression of chemotactic and adhesion molecules and release of chemokines and cytokines (Malek et al., 1999; Paszkowiak & Dardik, 2003; Li et al., 2005). On the other hand, when there is excess or lack of the stimulus can lead to pathological phenomena that cause changes in the arterial wall biomechanical properties. The high values of wall shear stress can cause damage in the endothelial cells (Fry, 1968). A prolonged high wall shear stress fragments the internal elastic lamina of vessels (Masuda et al., 1999) and gives rise to the initial change involved in the formation of cerebral aneurysm. Low wall shear stress has been reported to be related to aneurysmal growth (Jou et al., 2003) and rupture (Shojima et al., 2004) as it promotes various mechanisms that cause arterial wall remodelling. When the wall shear stress is lower than 0.4 Pa, it generates endothelial proliferation (Malek et al., 1999) and apoptosis (Kaiser et al., 1997). It is also responsible for abnormal vascular reactivity and vasospasm that can cause ischemia, angina, and myocardial infarction, increased permeability to macromolecules such as lipoproteins, increased expression of chemotatic molecules and adhesion molecules, enhanced recruitment and accumulation of monocytes/macrophages in the intima as foam cells, altered regulation in growth and survival of vascular cells (Lerman & Burnett, 1992; Bonetti et al., 2003; Gimbrone et al., 2000). This excessive low wall shear stress may be one of the main factors underlying the degeneration, indicating the structural fragility of the aneurysmal wall (Shojima et al., 2004). It appears that, after an initial injury that might result from excessive wall shear stress on the endothelial cells (Meng et al., 2007), progressive changes in aneurysm shape occur with a trend for the cross section to become more elliptical (Boussel et al., 2008). This generates a progressive decrease of wall shear stress leading to endothelial dysfunction, wall remodelling, and aneurysm growth (Ahn et al., 2007; Utter & Rossman, 2007).

4.2 Abdominal aortic aneurysm

Abdominal aortic aneurysms (AAAs) most of the affect men between 40 and 70 years old (5-7% of people older than 60) (Thiriet, 2008). AAAs rarely appear in individuals under 50 years old, but their incidence increases drastically at age 55 and peaks in the early 80s. Norway in 1994-1995 showed that AAAs are present in 8.9% of men and in 2.2% of women over 60 years old (Singh et al., 2001). Reed et al. (1992) showed that AAAs increased steadily with age after 60 years old and aortic dissections (aortic wall dissects and blood enters the wall causing the enlargement) showed peak between ages 70 and 75 years old and decreased after that (Figure 2).

Although aneurysms may develop throughout ht length of the aorta, AAAs are at least 5 times more prevalent than thoracic or thoracoabdominal aneurysms (Dua & Dalman, 2007). This fact can be explained from two points: physiologic and anatomic features unique to the distal aorta. The infrarenal aorta is the most common site of extracranial aortic aneurysm formation. Hemodynamic through the aorta combined with regional factors can explain this preferential distribution. In the infrarenal aorta the number of elastic lamellae (and therefore

elastin) is markedly decreased in comparison with the thoracic aorta which become fragmented and unorganized (Lakatta et al., 1987; MacSweeney et al., 1994) contributing to reducing elasticity and wall motion (Ailawadi et al., 2003). The degeneration of elastic fibers is accompanied by an increase in the collagenous substance (the stiffer structural component). As the ratio of elastin to collagen decreases, the vessel progressively loses its elasticity. The stiffening of the wall causes an increase in speed of the pulse wave. For example, in the aorta, the wave speed increases from 6.5 m/s in a 10-year-old child to upwards of 11 m/s in a 60-year-old adult (Nichols & O'Rourke, 1990). Reduced distal aortic elasticity, in combination with augmented pressure due to pulse wave reflections from the aortic bifurcation and other downstream arteries, may increase wall strain and aneurysm susceptibility (Humphrey and Taylor, 2008). The collagen-to-elastin ratio is the principal determinant of wall mechanics in the aorta. Changes in composition and structure of the arterial wall will alter the wall mechanics. An increase in collagen-to-elastin ratio results in a higher wall stiffness and lower tensile strength. Clinical observations show that most AAA walls become progressively stiffer as the diameter increases. This is because of biomechanical restructuring of the wall (Kleinstreuer et al., 2007). In the normal abdominal aorta, the collagen-to-elastin ratio is approximately 1.58 (Nichols and O'Rourke, 1990), however, the collagen-to-elastin ratio is much higer in AAAs (table 1).
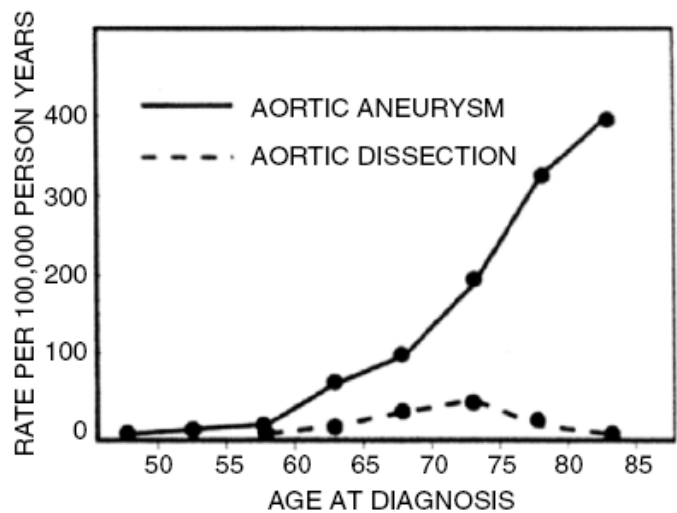

Fig. 2. Plots of incidence rates of aortic aneurysm and dissection by age at diagnosis. Extracted from Reed et al., (1992).

As commented above, hemodynamic of the infrarenal is one the main factors to cause the development of AAAs. Hemodynamic forces relevant to AAA pathogenesis can be obtained into three components: 1) wall shear stress (explained above), 2) hydrostatic pressure, the perpendicular force acting on the vascular wall; and 3) relative wall strain (RWS), the circumferential stretch of the vessel wall exerted by cyclic luminal pressure changes and resulting tensile stress. Several works in the literature showed that cultured vascular endothelial cells studies when submitted the disturbed flow conditions and unsteady turbulent stresses damage the endothelium, and the loss or malfunctioning of their regulatory processes may provide a first step to the degradation of the wall (Davies et al. 1984, 1995, 2009; Chiu and Chien, 2011). Hemodynamic conditions vary markedly along the aorta, from high Reynolds numbers (*Re*)

$$\mathrm{Re} = \frac{\rho V d}{\mu} \tag{1}$$

where ρ is the specific mass, *V* is media velocity, *d* is the diameter of the vessel and μ is the viscosity dynamic, at the aortic root to low and oscillatory shear conditions at the aortic bifurcation (Greve et al., 2006). Most relevant to AAA disease pathophysiology, and its predilection for the distal-most aortic segment, is the marked difference between resting aortic wall shear stress in the thoracic and abdominal aorta. In suprarenal aortic segments, flow is antegrade throughout the cardiac cycle, providing continuous antegrade laminar wall shear stress. In infrarenal aorta, wall shear stress values are lower, and reverse flow is present in late systole and diastole. In response to reduced distal arterial resistance and increase flow, such as is demonstrated in the response to even modest lower extremity exercise, wall shear stress becomes antegrade and laminar throughout the cardiac cycle, mimicking those characteristic of more proximal aortic segments. These distinct regional differences in hemodynamic influences may account for some component of the differential aneurysm risk noted between the thoracic and abdominal aortic segments (Dua and Dalman, 2010).

	Normal Aorta	Aneurysm
Elastin		
Average	22.7	2.4
Maximum	32.5	6.7
Minimum	16.1	0.2
Muscle		
Average	22.6	2.2
Maximum	33.6	6.4
Minimum	15.5	0.4
Collagen and ground substances		
Average	54.8	95.5
Maximum	63	98
Minimum	48	91.4

Table 1. Composition of normal aorta and aneurysm. Extracted from Nichols & O'Rourke, 1990.

As commented above, there are several work in the literature that show a correlation between very low shear stresses and the loss of permeability of the endothelial cell membrane (Helmlinger et al., 1991; Chiu et al., 2003). Studies have been the basis of an alternative, or even complementary, mechanism responsible for the origin of these aneurysms. During the normal course of aging, the abdominal aortic artery gradually undergoes conformal changes in its geometry (increasing its length and diameter, thickening its wall, etc.). Over time, the relative unconstructed nature of this artery inside the abdominal cavity may lead to the formation of bends, kinks, and other morphological changes that, in turn, create "disturbed flow" conditions inside the vessel (i.e., unsteady flow separation and weak turbulence). It is the argued that the anomalous response of the VEC to the high shear stresses, very low shear stresses, low, but oscillating shear stresses, and the anomalous temporal and spatial gradients of wall shear stress associated with these disturbed flow conditions could contribute to an unstable progressive degradation of the arterial wall and to the formation of the aneurysm (Lasheras, 2007). To understand more about the risk for and progression of aneurysm disease

is necessary introduce DFC to analyse hemodynamic in site specific, for example, in the aorta infrarenal. Compared with the suprarenal aorta, the infrarenal environment in resting subjects is characterized by increased peripheral resistance, increased oscillatory wall shear stress and stagnant flow (Dua and Dalman, 2010).

5. Computational fluid dynamics (CFD)

Since the beginning of the computer age, the computational study of fluid dynamic problems has been of interest to researchers studying both fundamental problems and engineering applications. Vast numbers of real-world problems require accurate viscous flow solutions to meet requirements for supporting engineering and science tasks – such as achieving ideal fluid dynamic performances and satisfying cost effectiveness. For example, computational analysis is indispensable, as well as economical, for developing advanced rocket-engine turbopumps and biomedical devices handling blood flow in humans. The computational fluid dynamics (CFD) for viscous, incompressible flow has been of interest for many decades to investigate fundamental fluid dynamic problems as well as engineering applications. The pioneering work by Harlow and Welch (1965) opened a new possibility of applying a computational approach to solving realistic incompressible fluid engineering problems, especially for three-dimensional problems (Kwak and Kiris, 2011).

Development of image-based modelling technologies for simulating blood flow began in the late 1990s. Since that time, many groups have developed and utilized these techniques to investigate the pathogenesis of occlusive and aneurysmal disease in the carotid artery (Long et al., 2000), the coronary arteries (Gijsen et al., 2007), the aorta (Tang et al., 2006) and the cerebral circulation (Cebral et al., 2005). Patient-specific modelling techniques have also been applied in solid mechanics analyses to predict rupture risk of aneurysms (Vorp, 2007).

There are many methods for quantifying vascular anatomy for patient-specific modelling of cardiovascular mechanics include noninvasive imaging techniques such as computed tomography (CT), magnetic resonance imaging (MRI), 3D ultrasound (3DUS) and an invasive method combining angiography and intravascular ultrasound (IVUS). Contrast-enhanced CT and MRI are particularly suited for generating high-resolution volumetric images of many parts of the vascular tree. Generally, iodinated contrast is used in CT angiography, and a gadolinium-based contrast agent is used in magnetic resonance (MR) angiography. MRI has the additional advantage of being able to quantify physiologic parameters, including blood flow, wall motion and blood oxygenation (Taylor & Figueroa, 2009).

A recent serial MRI-based case study demonstrated an association between the eventual site of plaque ulceration and elevated wall shear stress (WSS) (Groen et al., 2007) , a finding corroborated by an IVUS-based CFD study of 31 plaques that showed a clear association between elevated WSS and elevated strains within the plaque (Gijsen et al., 2008). Tang et al. (2006) quantified hemodynamic conditions in subject-specific models of the human abdominal aorta constructed from magnetic resonance angiograms (MRA) of five young, healthy subjects. Image-based modelling techniques are also being used to provide hemodynamic data in a clinical study testing the hypothesis that exercise can be employed to slow the progression of small (3-5 cm diameter) abdominal aortic aneurysms (Dalman et al., 2006). Rayz et al. (2008) performed a study on the effect of vertebral artery flow in four patients with basilar artery aneurysms. Computed flow fields were found to agree well with measurements made using PC-MRI. Subsequently, these authors reported good correspondence between regions of slow flow predicted by CFD at baseline and the deposition of thrombus observed at follow-up of three basilar aneurysms cases.

5.1 Real model of artery

One way to analyze and estimate the hemodynamic behaviour in an artery is the use of computational analysis of a real model of blood vessel (Legendre, 2010). The artery model is built based on images obtained from a patient. For example, in order to evaluate an abdominal aortic aneurysm in a patient, this model can be obtained by using multi-slice CT scan of the patient chest (Figure 3).
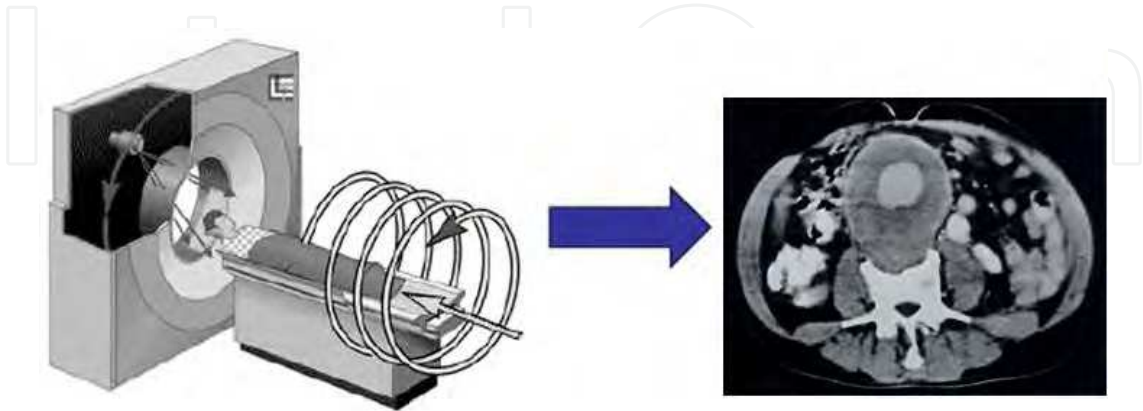

Fig. 3. CT images acquisition of a patient. Extracted from Invesalius (2008).

These DICOM (Digital Image Communication in Medicine) images should be treated by dedicated software, for example InVesalius, Mimics, etc., to select only the region of interest. Then, it is generated a compatible extension file that can be imported by a software to generate a three-dimensional mesh model of the arterial segment under study.

Overlapping the two dimensions (2D) images obtained through computed tomography or MRI, it can be created the computational model in three dimensions (3D) corresponding to the patient anatomical structure (Figure 4) and (Figure 5).
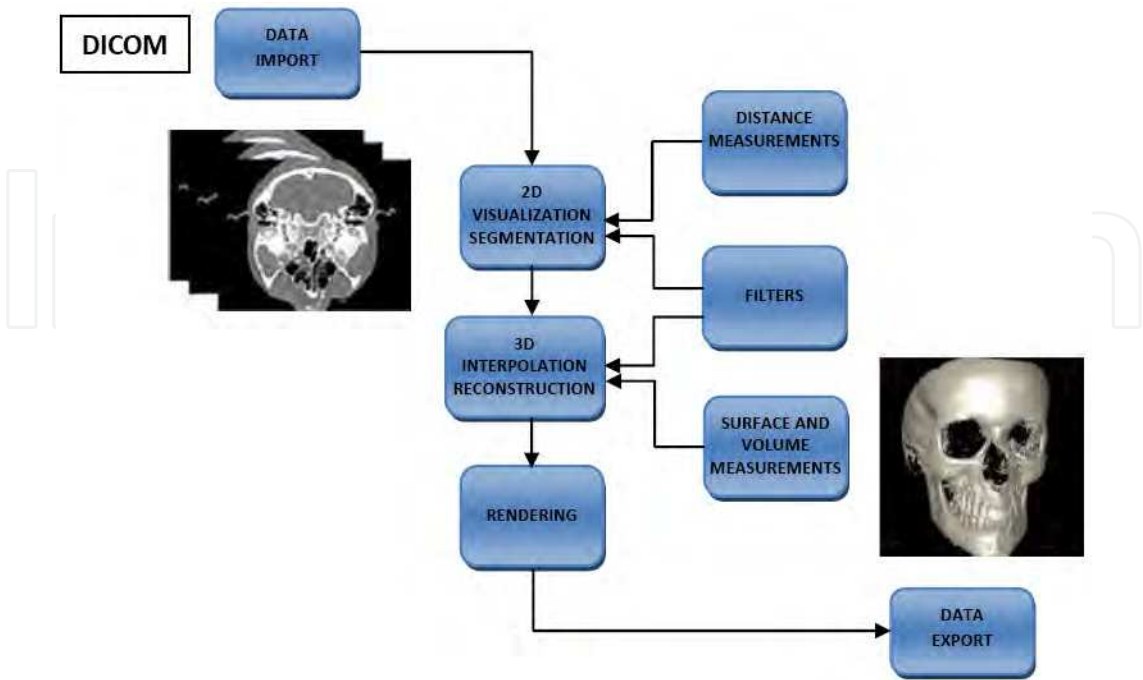

Fig. 4. Flowchart of the reconstruction of medical images. Extracted from Invesalius (2008).

The program makes, from tomographic medical imaging, a three-dimensional computational model in genuine size of several anatomical structures. This model can be manipulated and observed from different angles and it is also possible to separate specific parts of the image obtained from CT for a detailed analysis. The different densities of bone and tissue are easily identified by using tools such as color map. This feature is an important tool for creating computational models. Different methods of volume segmentation can be used for the treatment of the obtained images. Those methods are useful to adequately separate the regions of tissues, organs, anatomical structures etc.

The computational model generated by the software enables computer simulations in order to evaluate hemodynamic patterns developed by each patient according to their individual characteristics (Figure 6).

Fig. 5. Three-dimensional reconstruction of the abdominal aorta segment obtained from CT Scan. Extracted from Invesalius (2008).
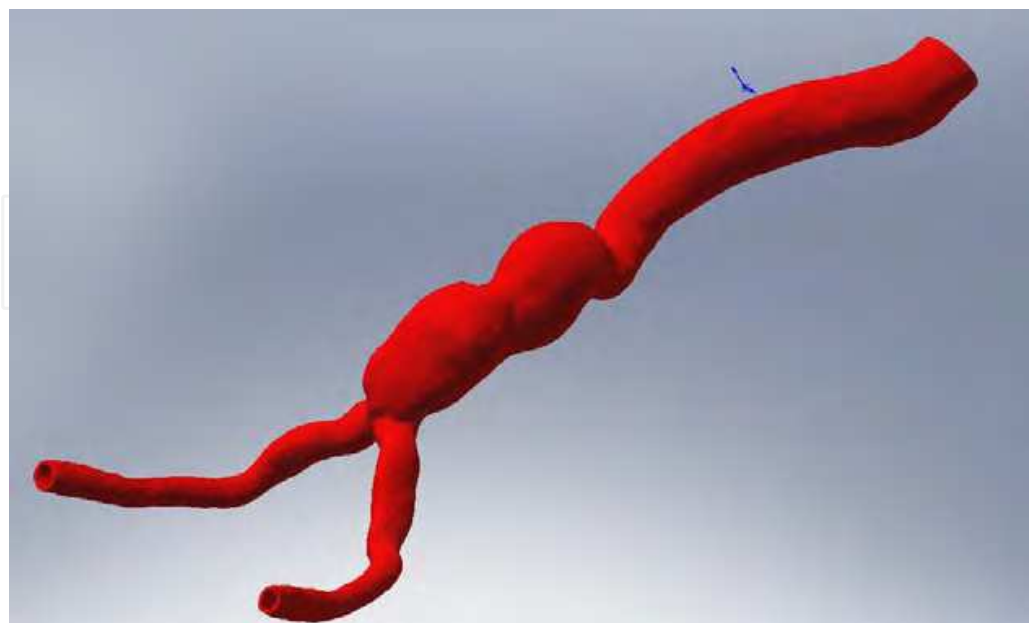

Fig. 6. Abdominal aortic aneurysm model obtained after image processing. Extracted from Legendre (2010).

5.2 Computer analysis

5.2.1 Finite volume method

The finite volume method (FVM) is a numerical technique for the solution of partial differential equations. The domain under study is subdivided into control volumes or computational cells. This method consists in the integration of the equations that governing the fluid flow over each control volume. The numerical solutions obtained by techniques of FVM (Patankar, 1975) have problems or errors known as false numerical diffusion. The numerical diffusion occurs when the interpolation function used in the discretization of equations differs from the exact solution. It can be understood as any effect that tends to moderate gradients or discontinuities present in the exact solution of a problem. There are some functions that were developed in order to minimize these effects of false diffusion, for example, the "upwind" method of second order.

For instance, the Fluent software solve the governing integral equations for the conservation of mass and momentum and also for scalars such as turbulence. The technique based on the volume control works as the following:

- The domain is divided in discrete control volumes using a computational mesh;
- Integration of the governing equations of the individual control volume to construct algebraic equations for discrete dependent variables (unknown), such as speed, pressure, etc.;
- Linearization of the discretized equations and solution of the resulting linear equation system to update the field values of the dependent variables.

5.2.2 Segregation solution method

In the segregated solution method, the governing equations are solved sequentially. Thus, to solve the equations that governing the phenomenon, many interactions are carried out until the solution convergence. Each interaction consists of the following steps outlined below:

- The fluid properties are updated based on the current solution. If the calculation has been started, the fluid properties are updated based on the initialized solution;
- The equations of momentum in x, y and z are solved one by one using the current values of pressure and mass flow to update the velocity field;
- Since the velocities obtained in step 2 cannot satisfy the conditions of continuity, pressure corrections are made so that continuity is satisfied;
- Other equations for scalar quantities such as turbulence are solved using the previously updated value of the other variables;
- It is made a convergence confirmation of the equations set.

These steps are performed until the convergence criteria are achieved.

5.2.3 Discretization

The technique based on control volume converts the governing equations into algebraic equations so that they can be solved numerically. This control volume technique consists of integrating the governing equations about each control volume, generating discrete equations that conserve each quantity on the volume control.

5.3 Overview

In general, the computational analysis tool can be viewed as a data interpolation in which the uncertainties of the results will depend on the model, the boundary conditions and mesh refinement adopted. To perform a study for hemodynamic performance evaluation, the

computational model adopted from the arterial segment should be able to represent the real model in a reliable way, as much as possible. The boundary conditions should be properly employed, since they are directly related to the consistency of the numerical results. The use of the finite volume method for solving the problem requires a proper discretization of the model in order to enable the convergence of analysis and also minimize the uncertainties in the results. On the other hand, it is also needed a good sense to perform the mesh refinement and computational cost in order to obtain a good quality of results in the shortest time. The quality of the mesh has an important role in the accuracy and stability of numerical computation. The convergence of results is intrinsically related to the size of mesh elements of the model and thus its quality is measured by its "asymmetry".

The choice of the optimal mesh for the simulation of the model should consider the performance and accuracy. The performance will depend on the total number of elements to be analyzed and thus a computational mesh made by larger elements will require lower computational cost due to lower number of elements required to cover the whole region of interest. However, this may affect the consistency of results. On the other hand, smaller mesh elements means better results, but this will require a greater computational cost (Figure 7).

One way to obtain more information regarding the arterial hemodynamic behavior is through the use of computer simulation. By using this kind of tool, it is possible to evaluate wall stress, recirculation areas, pressure, pathlines, velocity field and speeds distribution in the arterial segment.
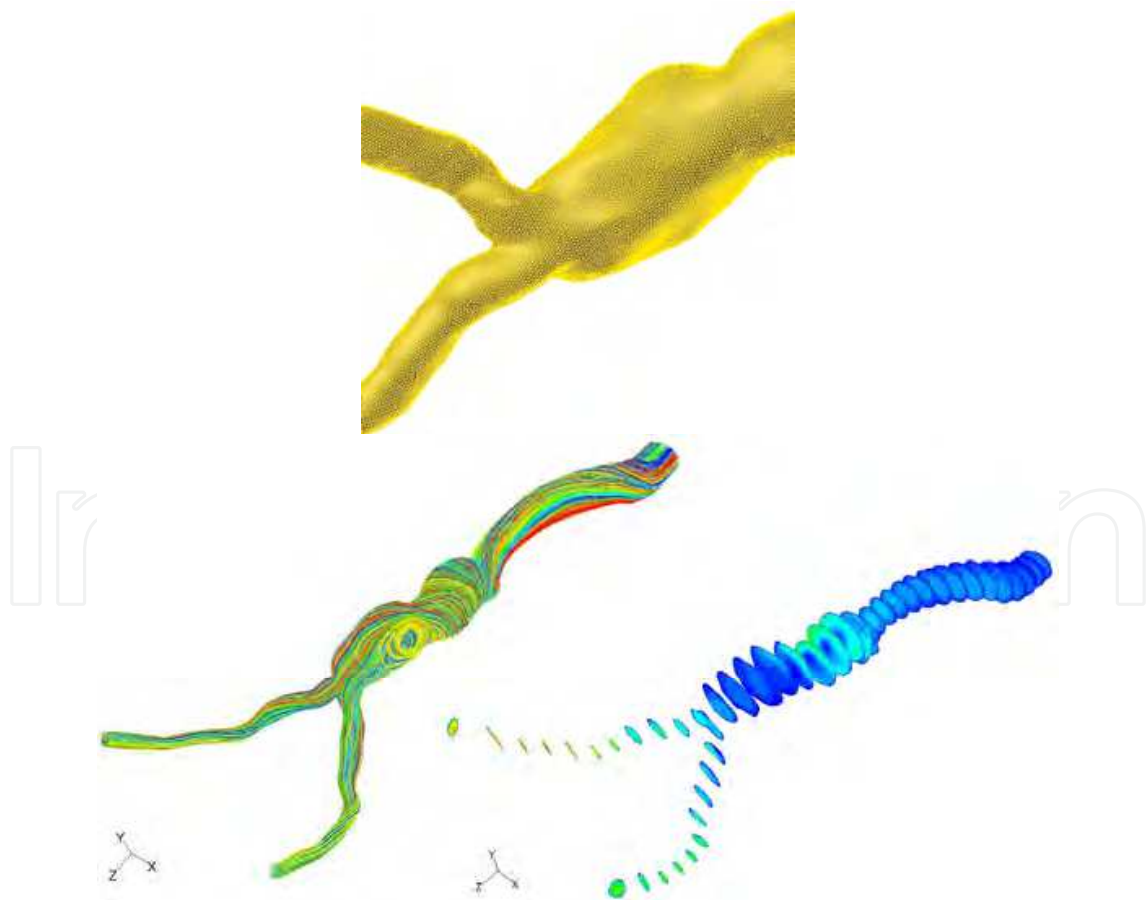

Fig. 7. Computational mesh, pathlines and velocity field of an arterial aneurysm. Extracted from Legendre (2010).

There are many works numerical and experimental fluid mechanics in the literature aimed at determining the characteristics of the flow shear stresses on the walls of AAAs at different stages of their development. These studies occur in ideal symmetric and nonsymmetric shapes of fusiform aneurysms and in realistic geometries obtained from high-resolution CT and angiographies (Egelhoff et al. 1999, Finol & Amon 2003, Salsac et al. 2006, Bessa & Ortiz 2009, Legendre 2010). Although, these studies show limitations about the appropriate initial and boundary conditions, as well as account for the precise elastic properties of the wall, they clearly show that once a fusiform aneurysm forms, the flow is dominated by the onset of an unsteady, massive separation from the walls that occurs immediately after the peak systole. When the flow separates from the walls during the deceleration portion of the cardiac cycle, a relatively coherent array of large vortices forms and the blood flow slowly recirculates (Lasheras, 2007). Bessa & Ortiz (2009) showed that occur massive separation from the walls and the velocity profile becomes inverted inside of the aneurysm, forming a vortex in the bottom and in the top of the aneurysm. After that, the velocity profile has been completely inverted and the vortex pair travels towards to the center of the aneurysm (Figure 8).
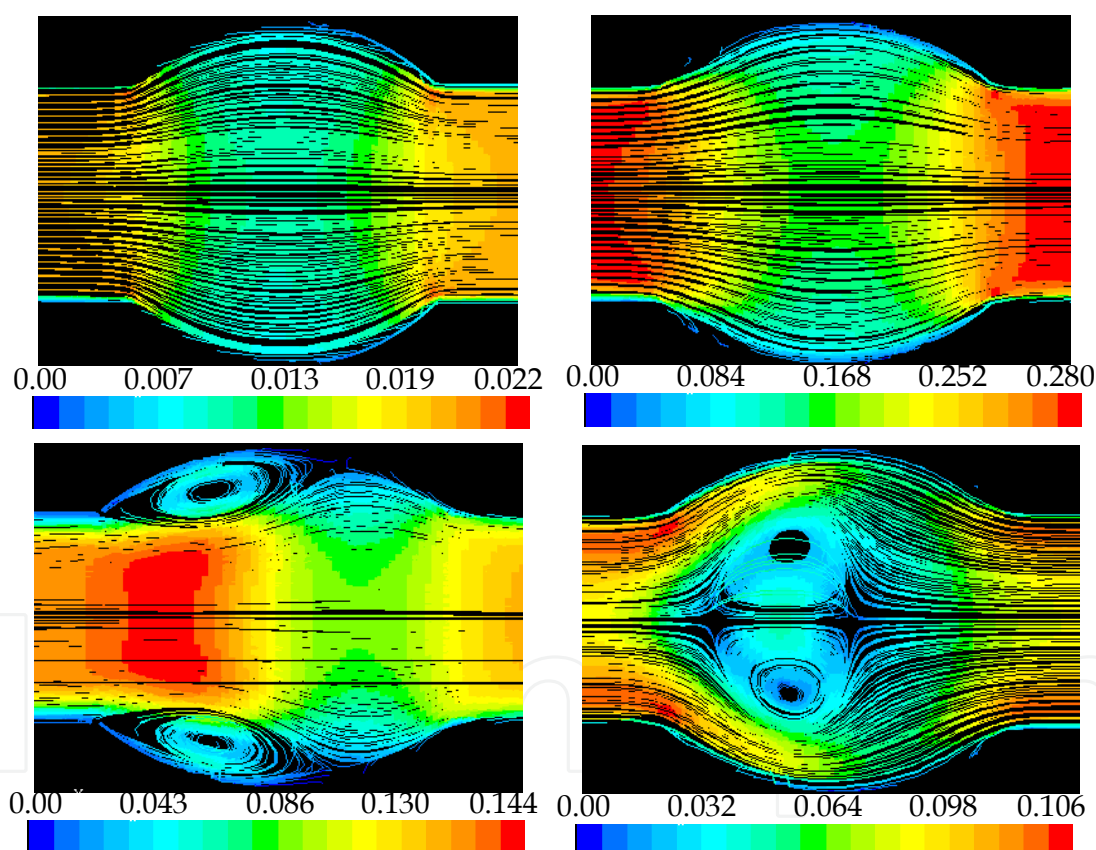

Fig. 8. Streamlines at various instant time of the cardiac cycle for aneurysm model. Extracted from Bessa & Ortiz (2009).

6. Conclusion

In the last few decades, it had a great effort of the scientific community to identify the beginning and the progression of the aneurysms. For this, several visualization techniques and 3D reconstruction of the vessels were developed associates with the computational fluid

dynamics with intention to analyze hemodynamic parameters that contributed to the development of the aneurysms. However, many questions exist on aneurysms that continue without answers: What cause aneurysms? When occur will the rupture of the aneurysms? Nowadays, advances in computational methods and computing hardware are also making it possible to solve increasingly more challenging problems, notably those involving flow instabilities and turbulence associated with a variety of vascular pathologies. However, to advance the research is the necessary to improve the related uncertainties the inserted boundary conditions in computational model. Exactely, even if these issues are fully resolved, still the mechanotransduction knowledge is essential to understand the endothelial cells behaviour. Therefore, still it has great challenges for the understanding of the origin and development of the aneurysm.
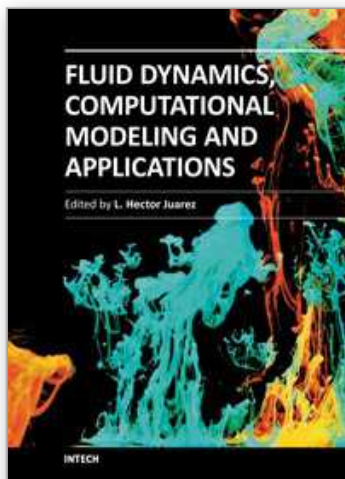
7. Acknowledgment

8. References

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