

Occlusion in anomalous coronary arteries: a parametric structural finite element analysis

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Abstract— Although anomalous origin of coronary arteries represents one of the most dangerous pathologies for young athletes, being related to sudden death, the underlying mechanisms are still to be elucidated. The present study aims to better understand how the lumen of the anomalous coronary may narrow during aortic expansion. To this aim, we created a parametric geometrical model of the aortic root and anomalous coronary, performing a static finite element analysis (FEA). In particular, we have analysed nine models with different take-off angles and intramural penetration, showing the functional effect of these geometrical features of the anatomical anomaly.

Keywords— Structural finite element analysis, coronary arteries, anomalies.

I. INTRODUCTION

CORONARY artery anomalies (CAAs) consist of congenital anatomical alterations of coronary arteries pattern characterized by specific features (e.g. origin, course, termination...) that is out of physiologic condition [1].

In particular, the anomalous aortic origin of coronary artery (AAOCA) occurs when coronary arises from the opposite or non-coronary *sinus* of Valsalva Figure 1. Different anomalous courses of the coronary are associated with this condition [2]; among these, the interarterial (between the aorta and the pulmonary artery) and/or intramural (inside the aortic wall) are thought to carry the major risk for sudden death due to coronary occlusion during aortic expansion [1].

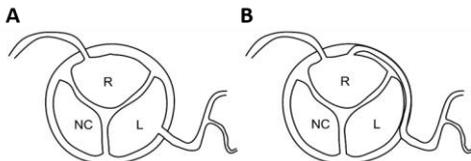


Figure 1 - Caudal perspective of aortic root with A) normal coronary and B) AAOCA with intramural course.

Although this pathology “rarely” (0.3-1%) occurs in the general population [1], its clinical incidence is principally found in individuals subjected to prolonged physical efforts, such as young athletes and soldiers, which undergo larger aortic dilatations comparing to others. For instance, Maron *et al.* showed that 11% of cardiovascular deaths among competitive athletes were due to AAOCA [3].

The pathophysiology of AAOCA is not clear: Angelini and colleagues ascribed the reduced coronary blood flow during aortic expansion to the compression of intramural portion [4]; Kaushal *et al.* suggested also that an acute angle of take-off could lead to a slit-like orifice [5]; on the other hand, Taylor and co-workers did not find any correlation between these geometrical features and sudden cardiac death [6]. Other authors suggest that coronary occludes only for compression

between aortic root and pulmonary artery [7].

Given such premises, the present study proposes to use structural finite element analysis (FEA) to assess the impact of coronary inclusion rate in the aortic wall and of the take-off angle on the coronary lumen narrowing during aortic exertion. This is, to the best of our knowledge, the first biomechanical study on AAOCA.

II. MATERIALS AND METHODS

A. Parametric CAD model generation

First, we created a *fully parametric* geometrical model of an idealized aortic root with AAOCA using the CAD software Rhinoceros v. 5.0 working with the plug-in Grasshopper v. 2014 (McNeel and associates, Seattle, Washington, USA). The model has twenty-three free parameters that allow to obtain an aortic root with the desired geometry and permit to simulate the AAOCA varying the positioning of the coronary on the root, the take-off angle (γ), the amount of intramural penetration (δ) and the length of intramural course (Figure 2).

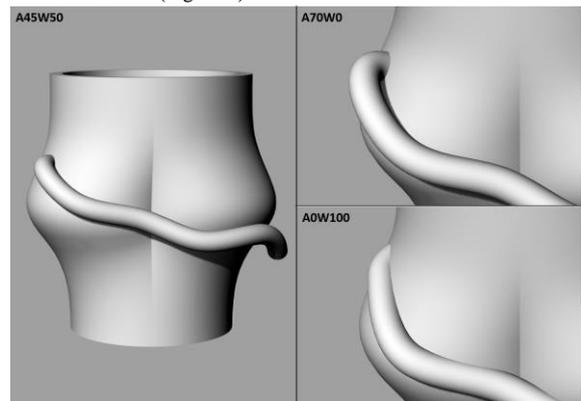


Figure 2 - Example views of CAD model. For labels, see “Material and Methods, B. FEM analysis”.

Second, we built the model by setting the parameters using typical values encountered in the literature, selecting studies on individuals subjected to prolonged physical efforts whenever possible. All parameters were fixed except γ and δ , which are defined as follows: a) γ is the angle formed by the coronary axis and the plane tangential to the external surface of the root in the intersection point and b) δ is the amount of intramural penetration and is related to the distance ρ between the points of the coronary axis and the external surface of the aortic root through the following equation:

$$\delta = \frac{R_{est} - \rho}{R_{est} - R_{int}} \bullet 100\% \quad (1)$$

B. FEM analysis

Nine models were selected for the FEM simulations, in the following referred as: A0W0, A45W0, A70W0, A0W50, A45W50, A70W50, A0W100, A45W100, A70W100, where “A” refers to γ ($^\circ$) and “W” refers to δ . Static simulations were performed with Abaqus Standard solver (Dassault Systèmes, Providence, RI, USA). The model was discretized with tetrahedral elements with quadratic interpolation (element C3D10). An approximate size of 1 was chosen for the elements of the aortic root, and 0.3 for those of the coronary, resulting in FE models having, on average, 263856 nodes and 172348 elements. Taking into account a pre-tensioning of the model, hydrostatic pressures of 100 mmHg and 15 mmHg were then applied to the internal surfaces of the aortic root and coronary to simulate the systolic pressures during exercise. As boundary conditions, the extremities of the aortic root were prevented to translate longitudinally and rotate planarly, but allowed to move radially to follow the dilatation of the root. Furthermore, a translational rigidity was added to the free extremity of the coronary using Abaqus spring elements “Spring1” to simulate the linking of the vessel to the tissues.

C. Post-processing

After the simulations, the internal surfaces of the deformed coronary were extracted as .WRL files and converted to triangular meshes with Rhinoceros. The .STL files were then imported in the VMTK software v. 1.3 (Orobix S.R.L., Bergamo, Italy) to compute the centerlines using a sampling length of 0.1 mm. The centerlines were then exported to Paraview v. 5.3.0 (Kitware Inc., New York, USA) for data visualization and manipulations.

III. RESULTS AND DISCUSSION

FE analysis revealed that a) aortic expansion leads to coronary occlusion and b) there is a dependence of coronary occlusion on both take-off angle and intramural penetration. Figure 3 relates the percentage reduction of the *minimum* radius along the coronary to δ , evidencing that the more is the intramural penetration the more is the coronary occlusion.

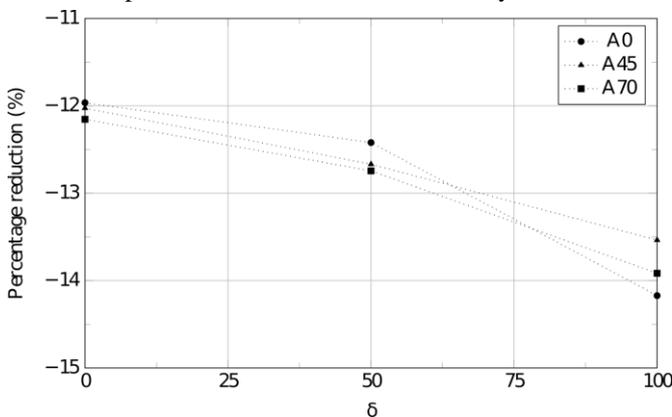


Figure 3 - Percentage reduction of coronary radius at different wall penetrations. The reduction increases with intramural penetration.

Although there is not such a result in the literature, the magnitude of the reduction of the *radii* agree with that of Angelini and colleagues, who found that diameters of intramural portion of coronaries narrowed by 8-10% during exercise conditions [4]. Figure 4 shows that acute take-off

angles lead to a reduction of the coronary lumen at the *ostium* level, as evidenced by the values at the left extremes of the graphs. These results confirm the hypothesis of Cheitlin *et al.* [8]. Furthermore, Figure 4 shows that the maximum coronary occlusion (evidenced by red lines) is found in correspondence of the *sinuses*, where the expansion is maximum.

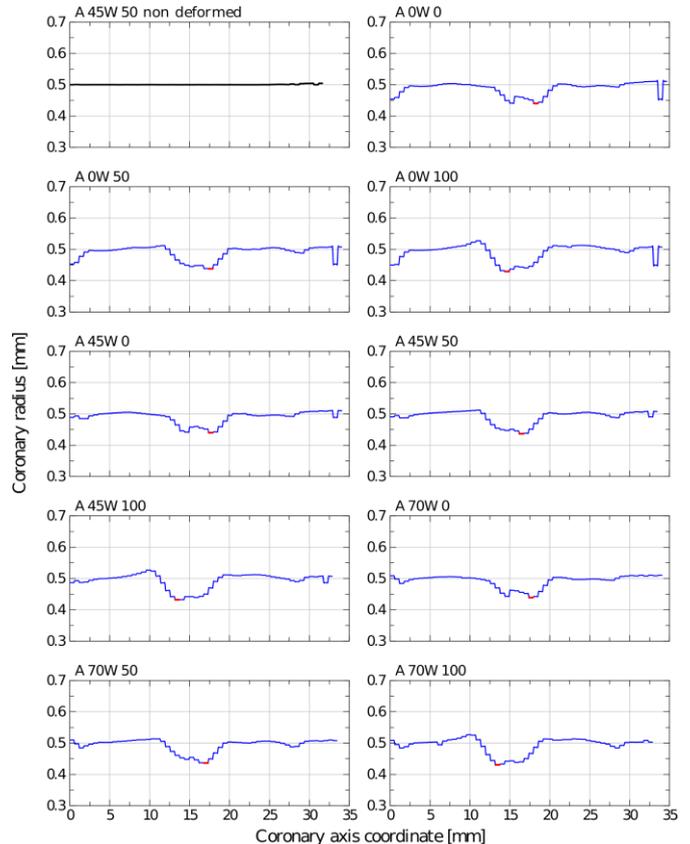


Figure 4 - Plot of the coronary radius for nine studied models. The maximum reductions (red marks) are in correspondence of the Valsalva *sinuses*. Acute take-off angles lead to a lumen narrowing at ostial level.

IV. CONCLUSION

This preliminary study reveals that a possible mechanism of coronary occlusion can rely on biomechanical reasons. Further studies should be made with more physiological pressures and boundary conditions to better understand this phenomenon.

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