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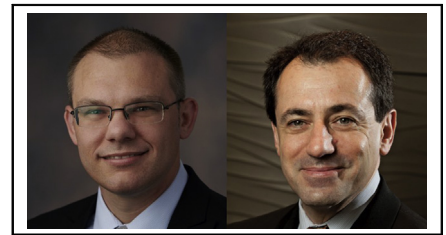
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Commentary: Anomalous coronary arteries and car crash testing

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An anomalous right coronary artery from the left coronary sinus (AAORCA) that is incidentally discovered, or even those with vague symptoms, continues to pose a conundrum.¹⁻³ Surgical outcomes, in terms of short-term survival, are excellent.⁴ However, recent reports note worsening ventricular function, aortic valve function, or even death in a very small number of patients undergoing surgery for an anomalous coronary, even asymptomatic patients.⁵ Therefore, a better understanding of who will benefit from surgical intervention is necessary to avoid surgery when the anatomy is unlikely to cause pathology. Currently, this decision is largely based on cross-sectional imaging. These current imaging modalities are insufficient, as they obtain static images and of the patient at rest. In this issue of the *Journal*, Lo Rito and colleagues⁶ attempted to address these problems. Building off their earlier work using finite element analysis (FEA), recognizing that the aortic root and coronaries are dynamic, they created a model replicating the loading conditions associated with activity.⁷



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CENTRAL MESSAGE

Structural finite element analysis allows for the dynamic evaluation of an anomalous coronary arteries. It may, eventually, help determine if surgical intervention is necessary.

FEA is used to evaluate the impact of a force on a complex structure. To do this, the structure is broken down into a finite number of elements. A simulation is run evaluating the strain on each element based on the force applied. When the elements are reassembled, the impact on the entire structure can be inferred. Commonly, it is used in the simulation of motor vehicle accidents. The car is broken down into multiple elements, each with different properties. The force of an impact on each element is evaluated, and they are reassembled and the damage to the automobile and its occupants determined.

Similarly, the authors divided the aortic root and coronaries (including their ostium and intramural course) into a finite number of elements. Using FEA simulations, the models were inflated at a baseline pressure and 2 greater pressure loads to simulate modest and strenuous activity. For each, the cross-sectional area of the coronaries was evaluated every 0.5 mm. This was performed for 10 patients, 5 controls and 5 with AAORCA. For patients with AAORCA, the model demonstrated that the normally arising left coronary appropriately increased in cross-

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sectional area similar to controls, whereas the anomalous right did not. At maximum loading conditions, the anomalous right demonstrated only an 8% increase in cross-sectional area, compared with 19% in controls. A more modest, yet significant, difference occurred at lower loading conditions. In addition, although a small sample size, the only 3 patients who were symptomatic demonstrated the least expansibility.

In these simulations, the patient's aortic wall is not replicated, as well as other potential elements that could be contributory, including any factor distal to the aortic root. For example, the pulmonary artery is omitted, neglecting a potential contributor to the pathophysiology. Certainly, much remains before this approach gains clinical applicability. Despite this, the authors are to be congratulated on their study. It pushes the field in the right direction, evaluating these lesions in a dynamic fashion. Only by visualizing the coronaries in "real time" will we be able to identify the at-risk population.

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