Author Reply to Commentary: Don’t miss the forest for the trees

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I appreciate the metaphysical musings and commentary from Dr Rajagopal on our translational work focused on diseased aortic tissue.1 Also, Rafael’s school of Athens is a great painting. In Plato’s philosophies of Forms, reality is divided into the perfect ideal and the phenomena we perceive. In the ideal, theories about reality are constant and perfect, whereas the perceived world is deceptive and changeable.

In his hierarchy of Forms, Plato considered Good the highest Form. Fortunately, as cardiothoracic surgeons we do Good everyday by saving and improving lives. We lead teams in pursuit of this altruistic objective. However, when making decisions about prophylactic aortic repair, we only have diameter as our surrogate for risk—a “deceptive perception.”

Certainly, increased diameter is an important criterion for predicting risk of death from aortic disease. Average diameters and area to height indices for patients in our study were within the guidelines for surgery.2,3 As Dr Rajagopal points out, aortic size is too simplistic of a tool to precisely predict the complications of aortic disease. This is a point with which I agree. A key finding from our study was that the association between diameter and tissue failure was weaker than several other patient-specific factors. This in vitro finding reinforces previous clinical observations about the limitations of aortic size to predict risk.4

Dr Rajagopal states that our model is too simplistic because we are unable to account for the viscoelastic, anisotropic, and in-homogenous nature of the aorta. I agree that our model is simple compared with natural aortic behavior. I disagree that the assumptions negate the merit of our study. All mathematical mechanical quantities applied to biologic tissues are based on assumptions that simplify reality. Cells react to mechanical stimuli, but how mechanical forces affect tissue microstructure is not well understood. Collagen, elastin, smooth muscle cells, and extracellular matrix react to force in specific ways. Molecules change conformation. We have previously shown that adjacent regions of pathologic aortic tissue behave differently.5 With our current understanding, simplified assumptions must be made about tissue properties tested in vitro. In our study, these assumptions were made consistently across a large number of specimens as acknowledged in the paper. We are actively evaluating in vivo ultrasonography, 4-dimensional flow magnetic resonance imaging, and building constitutive models to better understand aortic tissue characteristics and biomechanics.

Another thing that is oversimplified is Dr Rajagopal’s description of pathology affecting the thoracic aorta when he states that it does not rupture. Although most patients who present with acute aortic syndrome have aortic dissection, repair prevents death from rupture. Ascending rupture is an important cause of death from aortic disease and often occurs before the delivery of care.6

We do not assume causation from the empirical correlations in this study; rather, we suggest that diameter poorly predicts tissue failure. Dilatation is the hallmark of aortic degeneration, but it is only part of the picture. We must improve our understanding of the complex characteristics of the aorta in normal and diseased states, so we can develop better tests and therapies to prevent death. We will achieve these objectives, one simple step at a time.

References