Bicuspid aortic valve (BAV) disease and the aortopathy associated with are a complex condition, of which, the genetics and pathophysiology are still not entirely delineated. The clinical management of this group of patients has been a challenge for physicians in all disciplines. Borger and colleagues (1) are to be congratulated for their great efforts and success in condensing the best available knowledge and current evidence in the executive summary of the American Association for Thoracic Surgery guidelines on BAV-related aortopathy (2). The guidelines as well as the executive summary covers the major aspects of BAV aortopathy.

In accordance with previous recommendations, the current recommendations for aortic repair in this setting are as follows: (I) among patients with an aortic diameter ≥55 mm (level I); (II) among patients with an aortic diameter ≥45 mm, while undergoing cardiac surgery for a different indication (level IIa), or if certain risk factors exist (level IIa); and (III) among patients with an aortic diameter ≥50 mm if operated on by an aortic-experienced surgical team (level IIb).

But what happens if we try to address this information from the patient's perspective? We are aware of the fact that different clinicians have different thresholds for surgical intervention in patients with BAV-associated aortopathy (and aortopathy in general). That inconsistency probably stems from both the difference in surgical expertise and experience, the imaging modalities (and their standards of error) as well as from the laxity and variability in previous guidelines and recommendations.

In the current era of cardiac surgery, numerous options exist for repair and/or reconstruction of the different segments of the aorta, including the root, ascending, arch and descending aorta. When counseling the patient with a BAV, the decision tree typically begins with what led to the initial presentation: is it the aorta or the valve (or both). In the cases in which the aortic valve is pathologic and is indicative of surgery, we will have to recommend on whether to repair the aorta or not. What should we offer to a patient with a 43 mm (or 40–44 mm) ascending aorta in that case? If we propose that the current recommendation is to intervene on the aorta at 45 mm only, how do we account for the potential rate of growth (0.4–0.6 mm/year, or at a 10 years period 4–6 mm), the margin of error of current imaging modalities (1–2 mm), the very low (if any) added surgical risk for adding an aortic repair to an aortic valve replacement (3-6) and for the institutional volume and expertise which is known to affect outcomes as well (7).

In the cases in which the aortic valve is well functioning and is not indicative of surgery, then we would propose the diameter of 50–55 mm as a cut-off for surgery. Again, depending on other risk factors (both risk factors for aneurysm growth or dissection and risk factors for surgical morbidity and mortality), and whether you are or are not an experienced aortic surgical team in a center which has established, excellent surgical results. And how do we account for the attrition during follow-up?

The decision making becomes even more complex when
we deal with other indications for cardiac surgery (coronary artery disease or other valvular pathology) in a patient with a BAV aortopathy. Or with the concerned patient or the patient who does not want to have physical restrictions.

Probably the only current way to manage this clinically complex group of patients is to take all these (the aortic pathology, preoperative patient characteristics, required procedure, experience level of the surgeon, etc.) into consideration in an individualized manner, in an attempt to achieve a safe, durable and optimal outcome.

As always, a lot of work is still needed to be done to answer those complex clinical questions: (I) continue and contribute data to the body of evidence of BAV aortopathy. Should we propose a BAV global registry? (II) Improve our clinical results and aim at decreasing the surgical risk of ascending aorta replacement to near zero; (III) develop new grafts and material to potentially imitate the Windkessel function (or improve ventriculoarterial coupling); (IV) minimize attrition during follow-up monitoring.

If we return to answer the question in the title, our most frequent answer to the patient, is “I don’t really know”.

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Footnote

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