Aortic Stiffening and Dilation

Influence on Coronary Supply–Demand Balance

We read with great interest the article by Redheuil et al. (1), which reported that proximal aortic distensibility was an independent predictor of all-cause mortality and incidence of further cardiovascular events. Concerning congenital heart diseases, there are lots of reports about the decreased distensibility of native and post-operative aortas. We previously reported the decreased ascending aortic distensibility in patients with transposition of the great arteries after arterial switch procedure (2). The decreased aortic distensibility increases the afterload of the left ventricle and is a disadvantage for coronary circulation (3). Therefore, we also examined the subendocardial viability ratio in those patients (4). The subendocardial viability ratio is the ratio of the aortic diastolic pressure time integral and the aortic systolic pressure time integral (tension time index), that is, a measure of hemodynamic capacity for supply divided by myocardial oxygen demand. In that study, the tension time index, which indicates the myocardial oxygen demand, was higher than that in the control subjects, although the subendocardial viability ratio was the same. This pattern of the aortic pressure waveform, an elevated tension time index without a decrease of the subendocardial viability ratio, is similar to that in elderly people, although the patients in our study were elementary school-aged children.

Because preserving the coronary supply–demand balance is essential to sustain life, the subendocardial viability ratio should be maintained constant even in conditions with decreased aortic distensibility. Because the decreased aortic distensibility increases left ventricular workload, it is necessary to increase “supply,” although the stiff aorta is a disadvantage for coronary circulation (3). In our opinion, 1 of the solutions to the problem is aortic dilation. Although the aortic distensibility is decreased, an expanded aorta may be able to store enough blood during systole, which resembles the compensation of a failing heart; that is, although the left ventricular ejection fraction is decreased, the increased left ventricular end-diastolic volume can maintain sufficient cardiac output. Actually, our patients demonstrated aortic dilation (2) (so-called “aortopathy” in congenital heart diseases [5]). In addition, it is well known that the aortas in elderly people are not only stiff, but also dilated (3).

We would like to know whether the aortic diameter was related to its distensibility in Redheuil et al.’s study (1). Moreover, we are interested in the subendocardial viability ratio and the tension time index of the patients in their study.

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REPLY: Aortic Stiffening and Dilation

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We wish to thank Drs. Murakami and Niwa for their interest in our work and their very insightful comments. Concerning congenital heart disease, in particular arterial switch for transposition of the great arteries correction, we agree that altered myocardial oxygen supply is multifactorial and that the proximal aorta may play a role in several ways. First, the evolution and aging of the neoaorta-associating dilation, elongation, and rotation processes may lead to
late coronary complications found in up to 9% of cases (1). Second, accelerated stiffening of the proximal neoaorta as suggested by the authors may increase left ventricular (LV) afterload and alter LV function. However, the authors reported decreased aortic distensibility and increased diameter, but no change in indirect measures of myocardial perfusion based on indices of aortic pressure, in particular the subendocardial viability ratio (2) in these young patients (6.5 ± 1.1 years). Interestingly, the authors underline the potential benefit of aortic dilation to compensate for decreased compliance and normalize myocardial blood supply. We agree with Murakami et al. that this mechanism is probably at play within a certain degree of physiological aging and within a certain range of dilation in disease but cannot be generalized as beneficial in the extremes, because many complications are related to significant dilation of the proximal aorta. Nevertheless, as shown by the IRAD registry (International Registry for Aortic Dissection), many complications, including acute dissection, occur below the diameter threshold indicating prophylactic surgery. Risk stratification for acute aortic syndrome remains, therefore, currently insufficient. It is of great importance, as pointed out by Drs. Murakami and Niwa, to understand the relationships between aortic geometry and flow, as well as the coupling with the periphery downstream and LV upstream. The efficiency of the circulatory system in the young rests on a highly compliant proximal aorta of normal size and harmonious proportions. The systolic load buffering function of the aorta plays here a crucial role in regularizing and redistributing flow to the coronaries and other arteries during diastolic recoil. However, as we showed previously using magnetic resonance imaging (MRI), this dynamic function of the aorta is severely altered as early as mid-life in a general population sample (3). These functional changes in the ascending aorta are also associated and seem to precede aortic geometry alterations such as dilation, elongation, and increased tortuosity related to vascular aging (4). In turn, augmented static aortic volume and decreased wall distensibility are strong determinants of aortic flow patterns. We recently showed using quantitative MRI indices of proximal aorta that backward flow magnitude and its early onset time seen with aging were strongly related to ascending aortic diameter and arch morphology (5). Although aortic dilation may be considered a compensatory mechanism to a certain extent, it cannot necessarily be extrapolated to certain disease states. Indeed, the underlying mechanism for dilation in physiological aging rests mainly on elastin fibers fragmentation and on increase in the collagen-to-elastin content, whereas in inherited or acquired aortopathy, intrinsic anomaly of wall components predominates.

Increased aortic cross section and diameter were shown with increasing age and other risk factors in the MESA (Multi Ethnic Study of Atherosclerosis) study. We recently showed proximal aorta distensibility measured at baseline with MRI to be a predictor of mortality and hard cardiovascular events in the MESA cohort. In this study, distensibility was a better predictor of outcome than diameter alone while being intrinsically related, because distensibility is defined as aortic strain divided by pulse pressure. As expected, the relationship of distensibility was stronger with strain than with pulse pressure (respectively, Spearman \( \rho = 0.84, \rho = -0.51, p < 0.0001 \)). When broken down into constituent variables, relationships of distensibility were average diameter \( \rho = -0.33 \), minimal cross section \( \rho = -0.27 \), maximal cross section \( \rho = -0.37 \), systolic blood pressure \( \rho = -0.51 \), diastolic blood pressure \( \rho = -0.14 \), all \( p < 0.0001 \)). The subendocardial viability index was not available in the MESA study during MRI. However, in our prior study using tonometry, this index remained constant across groups of young to middle-aged individuals and decreased significantly only after 70 years of age. Although consistent with data from Murakami et al., it must be noted that these results were obtained in apparently healthy human adults, and whether this remains true during the later adult follow-up of their congenital disease patients remains to be studied.

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