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EDITORIAL COMMENT

Aortic Dimensions and Stiffness in Normal Adults*

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Practicing physicians are familiar with the term "aortic unfolding," which is used in chest X-ray reports to describe an aging change and generally assume that this term represents proximal aortic dilation. "Unfolding" is often associated with aortic calcification, which implies aortic degeneration. The degree of ascending aortic dilation with age is relatively small and out of proportion to the aortic unfolding observed in the chest X-ray.

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Aortic dimensions can be measured by ultrasound, invasive angiography, X-ray computed tomography, and cardiac magnetic resonance. Considerable controversy has arisen recently over the degree of aortic dilation with age, on sex differences, and on the implications of diameter change to the development of arterial hypertension and to the management of isolated systolic hypertension in older subjects. Mitchell et al. (1) have reported little change in aortic diameter with age and attribute isolated systolic hypertension to relative narrowing of the ascending aorta. This view has been challenged by us (2) and others (3) and now appears to have been retracted in the latest contribution from the Boston/Framingham group (4). In all this controversy, no attention has been directed at possible change in aortic length.

In this issue of *iJACC*, Sugawara et al. (5) from Tsukuba, Japan, and Austin, Texas, examine change in length in the ascending aorta and in other major arteries in the trunk and neck. The cardiac magnetic resonance method permitted measurement in all 3 dimensions; however, their report did not address changes in aortic diameter with age. The authors found little or no change in length of the carotid artery or descending aorta or in the iliac or femoral arteries with age. The major, and unexpected, finding was that length of the ascending aorta (defined as from aortic annulus to apex of arch) increased almost 2-fold between 20 and 80 years of age. The authors compared their findings of increased proximal aortic length to previously published values of aortic diameter and noted a far greater change in length than diameter (some 12% increase per decade compared with approximately 3% increase per decade for diameter) (Fig. 1).

The authors explained the increase in dimension of the proximal aorta as due predominantly to fatigue and fracture of elastin fibers with subsequent remodelling. This view (6), with which we concur, applies the principles of material fatigue that relate fracture of nonliving components with extent of pulsatile strain and the number of applied cycles of strain (i.e., heart beats). This theory explains both aortic dilation (from fracture of elastic components) and stiffening (from transfer of tension from elastin to collagenous fibers in the wall). The theory explains greater dilation and lengthening of the proximal aorta because of its greater content of elastin fibers and a greater extent of pulsation (in youth, approximately 10%), with each beat of the heart. In this case, it also explains the greater increase with age in wave velocity and impedance of the most proximal ascending aorta—some 3-fold between 20 and 80 years of age (6).

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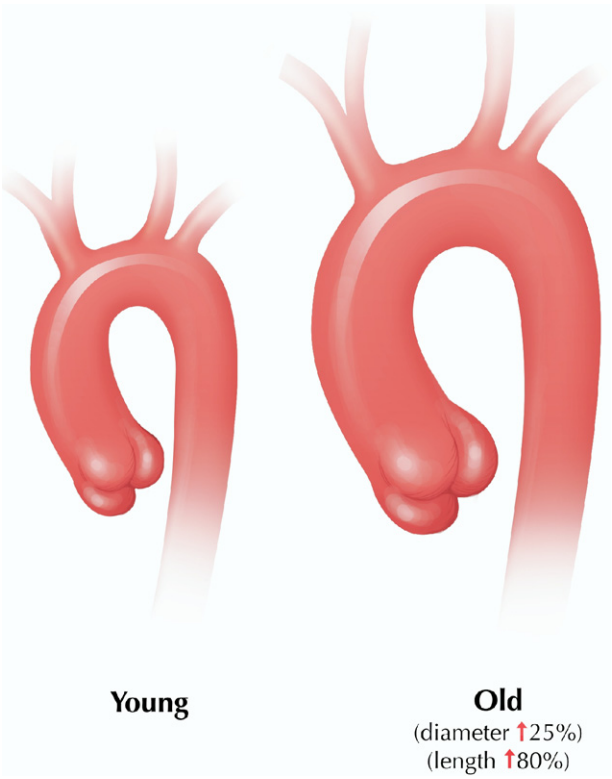

Figure 1. Typical Change With Age in the Ascending Aorta

Typical change in the ascending aorta is noted between a 20-year-old **(left)** compared with an 80-year-old man **(right)**. The length increases by approximately 12% per decade, whereas the diameter increases by just 3% per decade. Figure illustration by Rob Flewell.

But why does length of the proximal aorta increase more than diameter? The most likely explanation is that strain in the longitudinal direction is greater then strain in the circumferential direction. Pressure in the aorta is virtually the same in all directions. However, strain (change in length) depends on the orientation and strength of elastin fibers in the longitudinal axis of the vessel (6). Another factor affecting pulsatile strain is the capacity for movement in the longitudinal direction. The 3 branches of the aortic arch are tethered to surrounding structures, and the proximal descending aorta is tethered to the spine by intercostal arteries whereas the proximal aorta is attached to the heart which moves within the pericardial sac, with the aortic valve annulus moving down as the ventricle contracts. Longitudinal movement of the most proximal aorta may be enhanced by return of the reflected wave from the lower body and its re-reflection either from the contracting ventricle during systole or from the closed aortic valve during diastole (2,6). If pulsatile strain in the longitudinal direction of the proximal aorta is greater than pulsatile strain in the circumferential direction, one would expect greater lengthening with age in the longitudinal direction than increase in diameter (i.e., the findings of Sugawara et al. [5]). Because other arteries—the carotid, descending aorta, iliac, and femoral—are tethered directly or through branches to the spine and surrounding structures, one would expect little change in pulsatile strain with each beat and so little or no change in length with age—again as found by Sugawara et al. (5). They found that changes in aortic dimensions were associated with increased aortic pulse wave velocity (indicating greater aortic stiffness) and lower amplification of the pulse between the aorta and brachial artery (indicating greater wave reflection from the lower body) (2,6).

There are a number of clinical implications that arise from this study that apply to the ascending aorta. First, in type 1 aortic dissection as a consequence of medionecrosis from aging effect or Marfan syndrome, the ascending aortic tear is usually transverse rather than longitudinal, pointing to greater disruptive strain in the longitudinal than in the radial direction. The second implication relates to selection of patients for ascending aortic replacement to prevent aortic dissection. This is conventionally decided on the basis of aortic dilation (6–8). Measurement of ascending aortic length may be a better predictor of dissection and a better guide to selection of patients for Bentall's procedure. Indeed, this phenomenon may explain localization of aortic damage to the ascending aorta in Marfan syndrome.

Carotid/femoral pulse wave velocity has been accepted as a measure of aortic degeneration in the guidelines for management of hypertension published by the European Societies of Hypertension and of Cardiology in 2007 (9). A value >12 m/s is described in these guidelines as abnormally high. Such a value has been challenged on the basis that the pulse wave generated by the heart travels from the arch in opposite directions (up in the carotid artery and down in the descending aorta) so that values calculated from the distance between carotid and femoral sites will give falsely high values of "aortic" pulse wave velocity. Sugawara et al. (5) concluded that the carotid-femoral distance gave a falsely high (by approximately 25%) pulse wave velocity and that the appropriate distance was be-

tween the suprasternal notch to the femoral site. This distance is similar to that measured at cardiac catheterization, during withdrawal of the catheter from the ascending aorta to the femoral artery (10). These findings support the view that one cannot use carotid-femoral distance and that the upper range of normal for "aortic" pulse wave velocity in an older adult is closer to 9 m/s than the value of 12 m/s proposed by the European Societies of Hypertension and of Cardiology (9).

This study adds substantially to our knowledge of aortic degeneration with aging and to assessment of aortic function and vascular ventricular interaction and is relevant to timing of surgery in Marfan syndrome.

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Key Words: aorta ■ aortic stiffness ■ pulse wave velocity ■ Marfan syndrome.