Evaluation of aortic compliance in humans

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Basic Physiological Principles

The central aorta acts as a compliant tube that buffers and conducts pulsatile ventricular output and contributes mostly to total compliance of the arterial tree. The mathematical model introduced by O. Frank describing pulse wave propagation and arterial mechanical properties assumes that the arterial tree is an elastic chamber (windkessel) in which the diastolic pressure decays exponentially with a time constant that is determined by total arterial resistance and compliance. This model led to approaches of deriving systemic vascular compliance in humans from the diastolic arterial pressure decay and from pressure wave contour analysis.

The classic definition of compliance is the change in blood volume relative to a given change in distending pressure. The direct measurement of regional aortic compliance is difficult, because there is no simple means of estimating regional changes in blood volume. However, assuming that there is mainly radial and negligible axial vessel movement during pulse pressure, compliance can be estimated as a change in radius, diameter, or cross-sectional area for a given change in pressure. This assumption of negligible axial vessel movement during pulse pressure appears to be a valid approximation when compliance is estimated from radial extension of vascular cross sections for several reasons. It should be noted that neglect of longitudinal vessel axis is accounted for in the theory of pulse wave propagation developed by O. Frank, assuming that volume changes occur mainly because of expansion of the vessel wall along its radius. Also, when aortic pressure and radius are analyzed in the living dog, the ratio of pressure changes to changes in vessel radius obtained at identical sites compared well with measures of impedance, justifying the use of simpler radius measurements for estimating dynamic elasticity of the aorta.

Direct Imaging of Pulsatile Vascular Dimensions

Although the central aorta contributes most to total compliance of the arterial tree, most studies employing noninvasive imaging modalities have focused on evaluating superficial arteries such as the carotid, brachial, and radial arteries. Recently, the mechanical behavior of the aorta in humans has been studied using two-dimensional and transesophageal echocardiography and magnetic resonance imaging.

Initial studies have been published suggesting the use of intravascular ultrasound to determine aortic compliance in humans. Validity of intravascular ultrasound measurements has recently been proven by comparison with sonomicrometer measurements in conscious and anesthetized normal dogs. Intravascular ultrasound allows for mapping of viscoelastic behavior of multiple aortic segments and thus may help in investigating the mechanisms of changes in viscoelastic properties in experimental arterial hypertension and atherosclerosis models, avoiding the use of sonomicrometers. Potential limitations that need to be considered are misleading area measurements in oblique catheter positions and the need for careful calibration of ultrasound equipment.

Evaluation of Aortic Compliance from Pressure-Dimension Relationships

There are two approaches to evaluating compliance of large arteries from pressure-dimension relationships. The first approach is to measure instantaneous pressure-dimension relations of single pulse waves at varying levels of distending pressures, resulting in a family of hysteresis loops. Arterial compliance, representing the slope at each point of the pressure-dimension curve, is a nonlinear function of pressure. Higher distending pressures are accompanied by smaller changes in aortic dimensions that are determined by shifts along the same pressure-dimension curve when there is no change in arterial wall elasticity (Fig. 1). Changes in wall elasticity unrelated to acting distending pressures are eminent from shifts of pressure-dimension curves and changes in the slope of the pressure-dimension curve at a given pressure. The second approach is a simplification considering only peak systolic-to-diastolic differences in pressures and dimensions at varying blood pressure levels. However, the assessment of pressure-dimension relationships may give only an estimate of arterial
elastica properties, because a distinction has to be made between distensibility of the aorta as a tube and elastic material properties of the vessel wall components. Evaluation of elastic material properties needs to account for vessel geometry (vessel diameter-to-thickness ratio) to calculate stress-strain relationships (1). In the clinical setting, noninvasive measurements of aortic wall thickness are difficult but may presently be best approached semi-invasively using high-resolution transesophageal echocardiography (16).

Potential Clinical Importance of Estimating Aortic Compliance

Compliance of large conduit arteries has been found to be decreased as a result of aging (9), arterial hypertension (6), atherosclerosis (5), diabetes (22), and heart failure (15). Changes in the composition of the vessel wall and changes in vessel geometry accompanying these cardiovascular and metabolic disease states are the leading mechanisms explaining a decrease in vascular compliance. A decrease in aortic compliance increases cardiac and vascular load and leads to increases in systolic pressure and pulse pressure, an independent risk factor for development of cardiovascular disease (8). With recent reports suggesting that arterial wall compliance is improved by drugs acting on vascular structure or endothelial cell or smooth muscle function (2), estimation of central aortic compliance in clinical settings may become more relevant.

Table 1. Indices of aortic elastic properties

<table>
<thead>
<tr>
<th>Property</th>
<th>Equation</th>
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<tbody>
<tr>
<td>Static aortic compliance</td>
<td>$C = (D_s - D_d)(P_s - P_d)$</td>
</tr>
<tr>
<td>Peterson’s pressure-strain elastic modulus</td>
<td>$E_P = \ln \left( \frac{(P_s - P_o)(D_s - D_d)}{D_d} \right)$</td>
</tr>
<tr>
<td>Arterial stiffness constant</td>
<td>$\beta = \ln \left( \frac{(P_s - P_o)(D_s - D_d)}{D_d} \right)$</td>
</tr>
<tr>
<td>Young’s modulus</td>
<td>$E = \ln \left( \frac{(P_s - P_o)(D_s - D_d)}{D_d} \right)$</td>
</tr>
</tbody>
</table>

C, compliance; $E_P$, Peterson’s pressure-strain elastic modulus; $\beta$, arterial stiffness constant; $E$, Young’s modulus; $D$, diameter; $P$, pressure; $s$, peak systolic; $d$, end diastolic; $D_o$, arterial dimension at a standardized blood pressure; $h$, wall thickness.

REFERENCES


