Editorial Commentary

Terminology for Describing the Elastic Behavior of Arteries

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The Current State
The ability to characterize and quantify the elastic behavior of arteries has become increasingly important, because its application has broadened from basic physiology to clinical domains and the prediction of cardiovascular risk. Consequently, it is imperative that terminology to communicate across these disciplines is consistent and meaningful. In 1960, Peterson et al1 suggested coining a new definition of elastic modulus, the ratio of stress to strain, in terms of the pulse pressure, ΔP, and the directly measurable parameters ΔD and D (diameter). This has subsequently become known as Peterson’s modulus (Ep), where

\[ Ep = \Delta P / (\Delta D/D) \]

In 1975, one of us (R.G.G.)2 suggested calling the inverse of Ep the arterial compliance, C, where

\[ C = (\Delta D/D) / \Delta P \]

However, since that date, various authors have used the term distensibility for this quantity (eg, most recently O’Rourke et al3), and the term compliance has also become strongly linked with adherence to medical advice. The authors would therefore suggest that this difference in terminology is best resolved by using the long-established, engineering term compressibility when referring to the inverse of the elastic modulus.4,5

Thus, use of the well-defined, longer-established terms, elastic modulus and compressibility, would replace the need to use the terms compliance and distensibility, about whose mathematical definition some confusion exists in the literature.

Essential History
The classic physics of elasticity is often said to have started in 1600 with the discovery by Hooke that the ratio of stress to strain in isotropic materials, within their elastic limit, was constant. He defined strain as the fractional deformation caused by the deforming stress. Later, this ratio became known as the elastic modulus. For a change ΔV in a volume V of isotropic material in response to an applied pressure change ΔP, the value of ΔP/(ΔV/V) is known as the bulk modulus and is usually denoted by the letter K.

In 1926, Otto Frank6 derived an expression for the forward-going velocity of the pressure pulse, ΔP, in an infinitely long, thin-walled elastic tube filled with an essentially incompressible fluid and with the elasticity of the tube wall considered to be isotropic. This has become known as the characteristic pulse wave velocity and obtains in an elastic artery in the absence of any pressure reflections from the periphery, ie,

\[ PWV = \sqrt{(K/\rho)} \]

where \( \rho \) is the density of the blood and K is the elastic modulus of luminal volume change per unit length of artery, and

\[ K = \Delta P / (\Delta V/V) \]

It may be noted that because \( V = \pi R^2 \), where R is the luminal radius, then \( dV = 2\pi R dR \), and if \( \Delta V \) is small, \( \Delta V/V = \Delta V/V = 2\Delta R/R = 2\Delta D/D \), then equation 2 may be written \( K = \Delta P / (2\Delta D/D) \).

Path to Consensus in Terminology
Following Laplace’s theory for thin-walled tubes, Bergel in 19617 argued that the hoop tension, T, in the artery wall of thickness, h, was related to the luminal pressure, P, by the equation \( P = Th/R \), ie, \( T = Pr/h \). If P changes by a small amount \( \Delta P \), then \( \Delta T \), the increment of stress, is given by \( \Delta T = \Delta P r/h \). The circumferential strain caused by \( \Delta T \) will be \( (2\pi R + \Delta R - 2\pi R)/2\pi R \), ie, \( \Delta R/R \).

Thus, it is possible to define \( E_{inc} \), the static incremental Young’s modulus, for the material of the arterial wall, where \( E_{inc} = \text{stress/strain} = (\Delta P \cdot R/h)/\Delta R/R \); ie,

\[ E_{inc} = \Delta P \cdot D^2 / 2hD \]

Since \( \Delta P / (2\Delta D/D) = K \), equation 4, for the pulse wave velocity can be written

\[ PWV = \sqrt{(E_{inc} \cdot h / 2\pi R)} \]

Equation 6 was derived independently by Moens and Korteweg and is often referred to as the Moens-Korteweg equation.8,9 This equation assumes that the artery wall is isotropic and experiences isovolumetric change with pulse pressure. However, the details of the architecture of the arterial wall, eg, Clark and Glagov,10 show us that this
assumption is not justified. The 3 principal elastic component materials of the artery wall—collagen, elastin, and smooth muscles—have values of K in descending order of $10^7$ to $10^6$, $10^5$, and $10^4$ N/m², respectively. Furthermore, these components are influenced both in physiology and pathology by changes in the mucopolysaccharide matrix, or "ground substance," in which they are embedded. The result is that the value of K for any artery varies nonlinearly with pressure (eg, Berry and Greenwald 14) and with the frequency of the applied stress (eg, Bergel 15 and O’Rourke and Taylor 16). For these reasons, we would suggest that it is not realistic to attempt to attribute values of E to an artery wall for in vivo noninvasive clinical purposes, because these values would only obtain for very particular pressures, compositions, and thicknesses of the wall.

Work in Japan (eg, Hayashi et al in 197417 and 198018 and Kawasaki et al in 198719) raised the hope that the nonlinear effect of pressure on the value of K could be accounted for by the calculation of a value at a standard referred pressure. However, we have found that this approach only holds for 20- to 40-year-olds who are cardiovascularly “normal.” It is not true for hypertensives, menopausal women, or other clinical subgroups.20–23

In practice, therefore, we would suggest that the elastic behavior of the artery wall be described in terms of (1) K, the elastic modulus for volume change per unit length of lumen and/or (2) its inverse, the compressibility. An average value of K may be found for any known length of artery by measuring the velocity of the pressure pulse in the absence of reflected pressure waves or by direct measurement of D and ΔD at a particular artery cross-section. In clinical practice, the practical difficulty with the latter approach is the measurement of ΔP in the same cross section as D and the identification of that section if the values are to be repeated in order to follow the effect of treatment. However, approximations of these parameters may be derived from applanation tonometry at a distal site or from arterial distension waves recorded by echo tracking.23

**Implications for Practice and Previous Work**

The use of K or its inverse need not be a source of difficulty in interpreting results of previous work in the literature (eg, published values for compliance or distensibility) as long as the term has been clearly defined in the article and its relation to the definition of K is known.

For example, the variation in compliance with age and sex published by Laogun and Gosling 24 in 1982 for white subjects in the London area at their prevailing resting blood pressures could easily be re-expressed with compressibility on its ordinate axis (Figure 2).

**Conclusion**

In this editorial, we have underlined a current problem, reviewed the necessary historical context, and opened a dialogue for the future. If our suggestions were adopted, the proliferation of currently used terms to describe the noninvasively measured elastic behavior of arteries in the clinical and physiological literature would be replaced by clearly understood and clinically relevant terms: stress, strain, elastic modulus, and compressibility.
References


Key WORDS: elasticity □ arteries □ aorta □ compliance □ pulse
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