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ARTERIAL COMPLIANCE

What is it?	Arterial compliance is the inverse property of arterial stiffness. Formally, arterial compliance is defined as the change in arterial blood volume due to a change in arterial blood pressure [Spencer and Denison 1963]. It can be calculated for an arterial segment, or for the total arterial system. *Segmental area compliance (C_A):* The change in arterial area (ΔA) for a given change in arterial pressure (ΔP) at fixed vessel length (L) [O'Rourke 2002]: $C_A = \frac{\Delta A}{\Delta P}$ Unit of measurement = [m^2kPa^{-1}] where area (A) can be calculated from the lumen diameter (D) of a blood vessel based on the assumption of circularity (*i.e.,* A = pi x $(D/2)^2$. See also **COMPLIANCE AND DISTENSIBILITY COEFFICIENTS** *Total compliance (C_T):* A global estimate of arterial compliance of the entire arterial tree typically expressed in ml/mmHg or ml/kPa. It is computed by the summation of the segmental volume compliance values of all arterial vessels. C_T is an expression of the compliance of the large, elastic central arteries as well as the small muscular peripheral arteries.
Why do we measure it?	Arterial compliance has been used to investigate the effect of ageing on vascular function [Obeid 2017]. It is interesting to note that the early growth phase is associated with increasing buffering capacity of the large arteries due to the expansion of lumen dimensions and vascular wall as the child grows [Eck 2016]. C_T is a major parameter to evaluate the relationship between structural and functional changes in the vascular system with respect to its elasticity and capacity [Heitmar 2010, Haluska 2010]. Alterations in C_T are associated to various physiological (aging) [Van Bortel and Spek 1998] or pathological (hypertension) conditions [Beltran 2001], which cannot be necessarily assessed by current biomarkers.
How can it be measured?	Segmental compliance is usually determined from simultaneous diameter and pressure measurements. Diameter changes of superficial arteries (*e.g.,* the carotid) can be measured non-invasively by ultrasound-based "wall tracking" techniques and for non-superficial vessels (*e.g.,* the aorta) by magnetic resonance imaging. From the local diameter the cross-sectional area is calculated assuming a circular cross-section. Pressure measurements can be acquired either by invasive catheterization (especially for central arteries such as the aorta) or by non-invasive applanation tonometry or oscillometry at superficial

	arteries. When the change in diameter is related to the change in pressure, one obtains segmental diameter compliance, C_D = $\Delta D/\Delta P$. Area compliance, C_A, and diameter compliance, C_D, are related by: C_A = pi x D x C_D/2. Volume compliance is computed as C_V = L x C_A, where L is the length of the vessel. Direct measurement of C_T is not feasible because: (i) there is no simple way to estimate the changes in blood volume in the systemic arterial tree, and (ii) the arterial compliance depends on pressure, and thus no single value of compliance exists over the whole physiological pressure range. The simplest estimate of C_T is the ratio of stroke volume (SV) to pulse pressure (PP), namely SV/PP [Chemla 1998], but this method does not account for arterial outflow in systole and leads to an overestimation. Indirect methods for estimation of C_T involve simultaneous recordings of the aortic pressure and flow waveforms. Some of the most reliable and accurate techniques include the decay time method, the area method, and the pulse pressure method [Stergiopulos 1995].
Where is it measured?	Segmental compliance can be measured either at large arteries such as the aorta or at small arteries of the distal vasculature such as the radial artery. C_T is indirectly derived from simultaneous pressure and flow measurements acquired at the aorta.
References	Spencer and Denison 1963, Pulsatile blood flow in the vascular system. Handbook of Physiology. Circulation. II Am. Physiol. SocWashington, DC, chapt. 25, p. 842. O'Rourke et al. 2002. DOI: 10.1016/s0895-7061(01)02319-6 Obeid et al. 2017. DOI: 10.1097/HJH.0000000000001371 Eck et al. 2016. DOI: 10.1002/cnm.2755 Heitmar et al. 2010. DOI: 10.1038/jhh.2009.106 Haluska et al. 2010. DOI:10.1016/j.atherosclerosis.2009.10.018 Van Bortel and Spek 1998. DOI: 10.1038/sj.jhh.1000669 Beltran 2001. DOI: 10.1016/S0895-7061(01)02160-4 Chemla et al. 1998. DOI: 10.1152/ajpheart.1998.274.2.H500 Stergiopulos et al. DOI: 199510.1152/ajpheart.1995.268.4.H1540

FEEDBACK AND SUGGESTIONS FOR THESE DEFINITIONS[1] CAN BE SUBMITTED AT

https://vascagenet.eu/feedback-for-official-glossary-of-key-terms

[*] These definitions have been downloaded from https://vascagenet.eu/official-glossary and were released on 1st April, 2023.