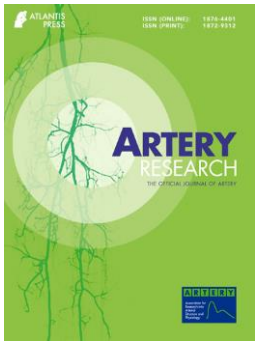

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P2.33
PERIPHERAL VERSUS CENTRAL PULSE PRESSURE VALUES IN CALCULATIONS OF CAROTID DISTENSIBILITY AND COMPLIANCE

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The aim of our study was to compare carotid compliance and distensibility calculations derived from central aortic pressure and peripheral brachial blood pressure measurements.
For this study 232 healthy, lifelong non-smoking, normotensive subjects (111 male & 121 female) were recruited (age 40±11 years, BMI 25.7±4.1 $kg.m^2$). Augmentation index (AIx), central aortic pressure (Sphygmacor, Skidmore Medical, UK), pulse wave velocity (PWV; Vicorder, Skidmore Medical, UK) and brachial blood pressure (Dynamap Pro, GE, USA), were measured using applanation tonometry. Stroke changes in common carotid diameter and intima-media thickness (CIMT) were measured from ultrasound (Philips HDX7E, Philips, UK) images using semi-automated software[1] (QLAB, Philips, UK). Carotid compliance and distensibility were subsequently calculated using brachial and aortic pulse pressure values.
Mean AIx, PWV and CIMT was 16.45±14.79 %, 7.04±1.22 $m.s^{-1}$ and 0.52±0.07 mm respectively. Carotid distensibility & compliance values calculated using brachial blood pressure (35.08±22.25 $kPa.10^{-3}$ & 11.23±7.95 $m^2.kPa.10^{-7}$) were significantly ($p<0.001$) lower compared to aortic derived measurements (48.63±34.81 $kPa.10^{-3}$ & 15.68±13.09 $m^2.kPa.10^{-7}$). Spearman's analysis showed that aortic derived calculations of compliance & distensibility were more strongly correlated with indices of arterial stiffness compared to brachial derived calculations (Table 1).

Table 1 Spearman's correlation analysis between brachial/aortic pulse pressure derived distensibility/compliance measurements.* $p<0.05$, ** $p<0.01$, *** $p<0.001$.

	Brachial		Aortic	
	Distensibility	Compliance	Distensibility	Compliance
	r	r	r	r
PWV	-0.0308	-0.0367	-0.3028**	-0.2674**
AIx	0.0843	-0.0452	-0.4298***	-0.5250***
CIMT	-0.0598	0.0741	-0.2327*	-0.1430

The results of the study reveal important considerations for blood pressure derived calculations of arterial stiffness.

1. Eric Y. et al. Carotid Arterial Wall Characteristics Are Associated With Incident Ischemic Stroke But Not Coronary Heart Disease in the Atherosclerosis Risk in Communities (ARIC) Study. *Stroke* 2012;43:103-108.

P2.34
EFFECT OF DECREASED PERFUSION PRESSURE ON THE DILATATION AND NORMALISATION PROCESSES OF FOREARM SKELETAL MUSCLE VESSELS AFTER ARTERIAL OCCLUSION

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Objective: The purpose of this investigation was to determine the effect of decreased perfusion pressure (Pperf.) on the dilatation and normalisation processes of skeletal muscle vessels.
Methods: Ten healthy subjects were investigated in supine position. Blood flow (I) and oxygen consumption (VO2) were studied during reactive hyperemia (RH) caused by arterial occlusion (AO) of 30 sec., 1, 3, 5, 7, 15 and 30 min. in the forearm at the level of the heart and after passive raising the arm above heart level. I was determined by venous occlusion plethysmographic method. VO2 was determined according Fick principle. Total cross-sectional area of forearm blood vessels (Q) was calculated.
Results: Analysis of the forearm I and VO2 in the discrete points of determination during RH revealed that raising the arm above heart level evoked the decrease of maximal values of I and VO2 in the early phase of RH, but further these values becomes greater than those in the horizontal arm position. Comparison of maximal values of forearm Q revealed that the reduction of Pperf. did not affect dilatation process of skeletal muscle precapillary vessels, but caused a delay of Q normalisation during RH.
Conclusions: Dilatation and normalisation processes of skeletal muscle precapillary vessels are two different phenomenons which are determined by different local factors. Dilatation reaction of precapillary vessels is determined by disappearing of dynamic component of transmural pressure after AO, but normalisation of resistance vessel tone is dependent from the repayment of O2 debt and blood supply conditions during RH.

P2.35
VASCULAR CONTROL IN DIFFERENT PARTS OF FOREARM ARTERIAL VESSEL TREE IN HEALTHY SUBJECTS DURING RESTING CONDITIONS AND DECREASED PERFUSION PRESSURE

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Objective: To investigate the changes in the forearm magistral and precapillary vessel tone in two different situations — during spontaneous changes in sympathetic activity and after reduction of perfusion pressure ($P_{perf.}$).
Methods: Ten healthy volunteers were studied in supine position. Blood flow (I) and volume pulse amplitude (ΔV) in the forearm were recorded by venous occlusion plethysmographic method. Systemic arterial pressure was determined auscultatory on the upper arm. Distensibility (D) of magistral arteries was calculated as a ratio between ΔV and pulse pressure (ΔP). Hemodynamic resistance (R) was calculated as a ratio between mean arterial pressure and I in the forearm. All investigated parameters were studied during resting conditions and after passive raising the arm above heart level.
Results: In the resting conditions I in the forearm oscillated from 0.8-6.3 $ml/100cm^3$ min. and corresponding changes was observed in D — when I increased D also increased and visa versa. After reduction of $P_{perf.}$ forearm D always increased and after the increase of $P_{perf.}$ - decreased. These changes in D occurred very rapidly (within 2-3 sec.) and remained permanent after the changing of $P_{perf.}$ Whereas I in the forearm after reduction of $P_{perf.}$ always decreased, but afterwards in 30% of the cases when initial value of I was below 2 $ml/100cm^3$ min. isovolumic autoregulation occurred - I increased and within a minute stabilised on a new increased level.
Conclusion: Intramuscular vessel tone is submitted not only to sympathetic activity and Ptransm. changes as extramuscular arteries, but also to metabolic control.

P2.36
FLOW-MEDIATED VASODILATION PULSE BY PULSE

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The measurement of flow-mediated dilation (FMD) is a standard method to assess endothelial function in the arteries (1,2). In practice, FMD measures arterial dilation after abruptly releasing the flow in previously clamped arteries (1,2). This clamping-releasing process might be considered as an experimental mimic of pulsation and, thereby, an FMD-equivalent measure might be determined by simply recording dilation that is induced by the initiation of flow during the rise of a pulse.
By using piezoelectric and photo-plethysmographic sensors, pressure (PP) and volume pulse (VP) waves were simultaneously recorded from adjacent digits, then their kinetics were compared. The systolic peak in the VP appeared with considerably slower kinetics as compared to that in the PP. The difference in the kinetics—either max. rate of rise or delay time (Fig. 1) computed after length-normalizing the pulses—was found to relate to the (*a*) subjects' age, (*b*) systolic blood pressure and (*c*) pulse wave velocity. Importantly, the kinetic differences between the PP and the VP of older subjects were apparently eliminated by the administration of sublingual nitroglycerin, suggesting that the rate of rise in the VP is a measure of endothelium-dependent vasodilation.
Our results imply the existence of a nitric oxide-dependent, flow-mediated mechanism of arterial dilation that operates pulse-by-pulse, on which basis a simple pulse contour analysis method, which might provide equivalent results as FMD, is developed to that assess endothelial function in the arteries.