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Aortic impedance in older subjects: MR and applanation tonometry study by wave intensity analysis

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Introduction: Central pressure waveform can be decomposed into pressure transmission and reflection waves. Zc, characteristic impedance of aorta can be measured through the recording of pulse pressure and aortic flow signals and reflects structural and functional properties of elastic central and peripheral muscular arteries.

Methods: This study proposes a novel method using wave intensity analysis to assess reflected waves by decomposition of magnetic resonance imaging central aortic flow and central aortic pressure waveform measured by tonometry in 30 older subjects allowing to obtain the aortic characteristic impedance in the time domain and reflection index (IR) and reflection magnitude (MR).

Results: Elderly patients were mean aged of 75.25±5.85 years (14 female, 16 male). Mean Zc value was 424.3±94.1 DSC for the whole aging population. We found a strong negative association between Zc and LVET (r=−0.53, p=0.004). We found also a trend of negative association between Zc and aortic diameter, at the aortic root, (r=−0.36, p=0.05) and pressure augmentation (r=−0.36, p=0.06), and also with Aix (r =−0.32, p=0.09). Zc was positively associated with PPA (r = 0.41, p=0.02), and also strongly with HR (r=0.54, p=0.003).

Association between RM and IR were positively significant with aorta length magnitude by MRI, respectively (r=0.41, p=0.03) for RM and (r=0.42, p=0.03) for IR. RM associated positively with thoracic aorta length (r² = 0.15, p=0.04) after adjustment to age and height, IR also associated positively with thoracic aorta length (r² = 0.16, p=0.04) after adjustment to age and height.

Conclusions: This automatic signal treatment of aortic flow and pulse pressure waveform is a good method for assessment of wave reflections in older subjects allowing obtaining reflection magnitude. Aortic characteristic impedance is inversely correlated to left ventricular ejection time and aortic diameter as new insights suggest.

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Protective role of nucleotidases against the development of hypertension

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Hypertension is characterized by a hypertrophic remodeling of big arteries, increased tone in smallest, endothelial dysfunction and accompanied by oxidative stress, inflammation and fibrosis. Extracellular nucleotides, which are released under cellular stress, promote deleterious pathological responses (vasoconstriction, inflammation, vascular permeability) through P2 receptors activation although the contribution of purinergic signaling to cardiovascular pathologies remains to be established. Hydrolysis of these molecules is provided by nucleoside triphosphate diphosphohydrodases (NTPDases), especially NTPDase1 (CD39), highly expressed in the arterial wall. Together with ecto-5’ nucleotidase (CD73), these enzymes generate vasoprotective adenosine (ADO anti inflammatory, vasodilatory). Using Apyrase (APY, soluble potato nucleotidase) treatment and CD39 deficient (Entpd1−/−) mice, we evaluated the potential benefit of nucleotides hydrolysis in experimental hypertension. After 12 days of AngII (1mg/kg/day) infusion, with or without APY (45U sc, 15U ip every 3 days), the increase in systolic blood pressure (SBP) and the hypertrophic aortic remodeling were significantly reduced in AngII/APY-treated mice compared to AngII-treated mice. Reversely, in Entpd1−/− mice treated with intermediate dose of AngII (0.5mg/kg/day) the increase in SBP was greater than in Entpd1+/+ mice. This was associated with exacerbated hypertrophic aortic remodeling. Interestingly, RT-qPCR revealed a decreased CD39 expression level in resistance arteries of AngII-treated mice and SHR rats, suggesting a role for the enzyme in hypertension. The role of CD39 as a regulator of arterial tone through the control of P2Y6 receptor activation is likely, although its contribution in the prevention of vascular inflammation remains to be investigated. Consequently, nucleotidases protect against high blood pressure and represent new therapeutic area in the treatment of hypertension.