



# *Presión del pulso, variaciones de la onda arterial y supervivencia en la enfermedad renal terminal*

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The ill effects of hypertension are usually attributed to a reduction in the caliber or the number of arterioles, resulting in an increase in total peripheral resistance (TPR). This definition does not take into account the fact that BP is a cyclic phenomenon with systolic and diastolic BP being the limits of these oscillations. The appropriate term to define the arterial factor(s) opposing LV ejection is aortic input impedance which depends on TPR, arterial distensibility ( $D$ ), and wave reflections (WR).  $D$  defines the capacitive properties of arterial stiffness, whose role is to dampen pressure and flow oscillations and to transform pulsatile flow and pressure in arteries into a steady flow and pressure in peripheral tissues. Stiffness is the reciprocal value of  $D$ . These parameters are BP dependent, and arteries become stiffer at high pressure. In to  $D$  which provides information about the «elasticity» of artery as a hollow structure, the elastic incremental modulus ( $E_{inc}$ ) characterizes the properties of the arterial wall biomaterials, independently of vessel geometry. As an alternative, arterial  $D$  can be evaluated by measuring the pulse wave

velocity (PWV) which increases with the stiffening of arteries. Arterial stiffening increases left ventricular (LV) afterload and alters the coronary perfusion. With increased PWV, the WR impacts on the aorta during systole, increasing systolic pressures and myocardial oxygen consumption, and decreasing diastolic BP and coronary flow. The arterial stiffness is altered primarily in association with increased collagen content and alterations of extracellular matrix (arteriosclerosis) as classically observed during aging or in arterial hypertension. The arterial stiffening estimated by changes in aortic PWV and intensity of WR are independent predictors of survival in end stage renal disease (ESRD) and general population. Improvement of arterial stiffening could be obtained by antihypertensive treatment as observed with the calcium-channel blocker and ACE inhibitors. ACE inhibitors increased AC and reduced WR, and it has been shown that reversibility of aortic stiffening and use of ACE inhibitors had favorable independent effect on survival in hypertensive patients with advanced renal disease.