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Understanding Ventricular Afterload

Jose Fernando Guadalajara Boo, Md, Facc*

National Institute of Cardiology "Ignacio Chavez", Mexico, City.

*Correspondence:

Jose Fernando Guadalajara Boo, Md, Facc, Instituto Nacional de Cardiología "Ignacio Chávez", Juan Badiano no. 1, Seccion XVI, Belisario Domínguez, 14080, Ciudad de México.

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Introduction
From the end of the 19th century, RH Woods, showed that Laplace's Law was applicable in the human heart to know the tension to which these tissues are exposed during different moments of cardiac function [1]. This issue was later studied by Sandler and Dodge in the human heart, who did cardiac catheterization and angiographic studies to describe the concepts of tension and stress [2]. Subsequently, WP Hood et al., demonstrated that when the systolic pressure is increased, by myocardial hypertrophy normalizes the systolic wall stress (SWS) in the left ventricle [3] and Sasayama S. et al. demostrated normal inotropic state of the hypertrophied left ventricle has hyperfunction [4] Gunther and Grossman with cardiac catheterization demonstrated in patients with aortic stenosis that there is an inverse relationship between SWS (estimated on Laplace's Law) and LVEF; this means that SWS in fact matches to left ventricular afterload [5]. With these findings, Carabello et al., studied 13 patients with severe aortic stenosis (SAS) with catheterization; LVEF less than 50% and found that in patients with a severely reduced LVEF but with high SWS, there was no surgical mortality when they were taken to aortic valve replacement; furthermore, LVEF was normalized; conversely, all patients with a severely reduced LVEF and a low or pseudonormalized afterload (SWS) died in the postoperative period [6].

This issue was studied extensively by John Ross Jr. and was called the afterload mismatch [7].

Noninvasive quantification of myocardial hypertrophy
Since 1976 Lincoln E. Ford, described that the relationship between the thickness of the wall and the radius of the cavity under normal conditions remains stable regardless the size of the heart, demonstrating that the mass/volume ratio and its derivative thickness/radius (h/r) in diastole, are constants that govern cardiac physiology [8]. Based on these principles and using 2D Echocardiography, Richard Stack et al, in 1981 described the technique for hypertrophy study at Duke University, (Figure 1) [9].
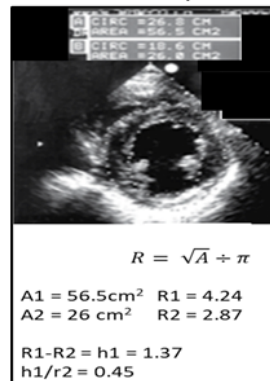
Figure 1: Parasternal short axis view of the left ventricle. Epicardial (A1) and endocardial (A2) areas are drawn on end-diastole. The radius (r) of both areas is estimated. r1 is subtracted by r2 and the thickness of the wall (h) in end-diastole is obtained

$$R = \sqrt{A} \div \pi$$

Square root of area 1 /π = r1
Square root of area 2 /π = r2
r1 − r2 = h1
h1/r2 = h/r in diastole

A1 = 56.5cm²   R1 = 4.24
A2 = 26 cm²   R2 = 2.87

R1-R2 = h1 = 1.37
h1/r2 = 0.45

Normal values: 0.42-0.56

h1/r2 < 0.40 inadequate hypertrophy
h1/r2 > 0.56 left ventricular hypertrophy

Cellular definition of stress
Parietal stress: It is the force that tends to separate the myofibrils from each other by squared centimeter² (cm²) (Figure 2) [5].

Cardiac Afterload Definition
It is the resistance that the myocardium has to overcome in order to raise intraventricular pressure, open the aortic valve and expel its content into the great vessels. *In human being systolic wall stress (SWS):* It is the force per unit of sectoral area, which opposes to ventricular contraction to achieve blood ejection to the great vessels [2,5] (Figure 2).
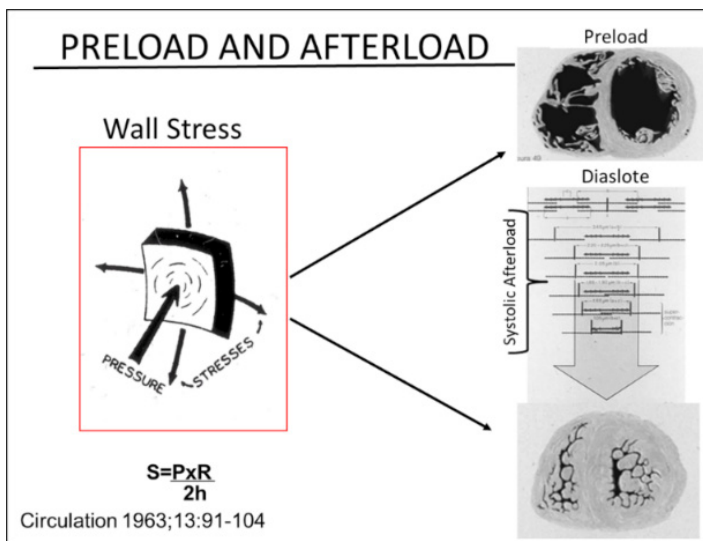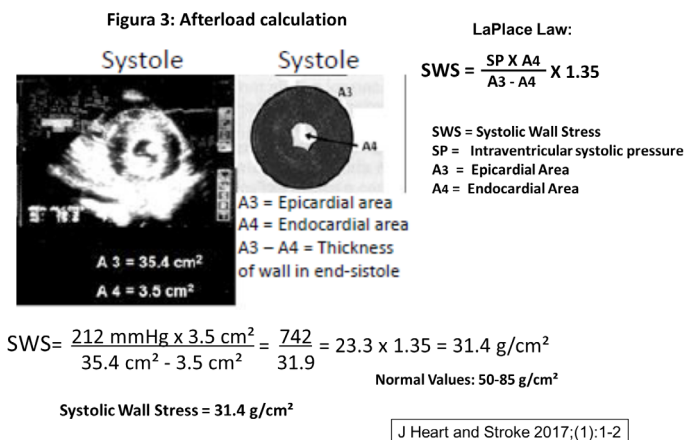
PRELOAD AND AFTERLOAD

Figure 2: **Preload** = Dyastolic Stress. **Afterload** = Systolic Wall Stress

Noninvasive Calculation of Afterload

Using a short parasternal axis in systole, at the level of the papillary muscles, the epicardial area (A3) and the endocardial area (A4), where meassured in the same axis, the epicardial area (A3), minus the endocardial area (A4), let us to estimate the thickness of the left ventricular wall in systole, an essential parameter for the calculation of SWS (afterload) (Figure 3).

Afterload depends on the pressure that it has to overcome, first to open the aortic valve and then to eject the blood towards the aorta, during the expulsive phase (thickening of the wall), that reduces the area of the cavity and with this, myocardial stress (reduction of the myocardium area) in such a way that the afterload, according of the Laplace Law [10] is directly proportional to the intracavitary systolic pressure multiplied by the radius of the cavity and is inversely proportional to the wall thickness in systole [11-13]. In this technique the results are multiplied by 1.35 for convert mmHg in g/cm^2. In the presence of aortic stenosis, the maximum systolic ventricular pressure is obtained by adding the brachial systolic pressure by the sphygmomanometer to the maximum systolic gradient obtained by Doppler.

Figura 3: Afterload calculation

LaPlace Law:

$$SWS = \frac{SP \times A4}{A3 - A4} \times 1.35$$

SWS = Systolic Wall Stress
SP = Intraventricular systolic pressure
A3 = Epicardial Area
A4 = Endocardial Area

A3 = Epicardial area
A4 = Endocardial area
A3 − A4 = Thickness of wall in end-sistole

A 3 = 35,4 cm²
A 4 = 3,5 cm²

$$SWS = \frac{212\ mmHg \times 3.5\ cm^2}{35.4\ cm^2 - 3.5\ cm^2} = \frac{742}{31.9} = 23.3 \times 1.35 = 31.4\ g/cm^2$$

Normal Values: 50-85 g/cm²

Systolic Wall Stress = 31.4 g/cm²

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All these findings are consistent that when a reduced LVEF is due to excessive afterload, the aortic valve replacement (critical aortic stenosis or regurgitation), (interventional treatment of aortic coartaction) or Bental & Bono procedure in aortic aneurism with significant regurgitation relieves this afterload, and because there is no intrinsic myocardial damage, the heart recovers its function and a better prognosis can be expected. These results have been reproduced, when afterload is excessively high and this accompanies a significant reduction in the LVEF and surgical treatment is capable of completely reversing heart failure and prolonging life [14,15].

Currently, the assessment of the surgical indication of patients with aortic disease or aortic coarctation, does not include the evaluation of the afterload to which the left ventricle is exposed, neither in patients with normal LVEF, nor in patients with a reduced LVEF (heart failure).

Preveously left ventricular afterload was accurately calculated by 2D echocardiography [11-14] and by nuclear magnetic resonance [16].

In this work, we aimed to assess left ventricular afterload (SWS) and myocardial hypertrophy[4-5] by noninvasive technics an attempt to complement the study of ventricular function and look for quantifiable parameters that allow us to approach the optimal time to indicate the surgical intervention of patients with aortic valve disease or aortic coarctation, especially in patients with severe heart failure.

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