

# Scisense PV Technical Note

## Understanding Afterload

Afterload is the mean tension produced by a chamber of the heart in order to contract. It can also be considered as the 'load' that the heart must eject blood against. More precisely, afterload is related to ventricular wall stress by a modification of the LaPlace Law which states that wall tension (T) is proportionate to the pressure (P) times radius (r) for thin-walled spheres or cylinders. Therefore, wall stress is wall tension divided by wall thickness.

$$\sigma \propto \frac{P \times r}{h}$$

$\sigma$  = ventricular wall stress

P = ventricular pressure

r = ventricular radius

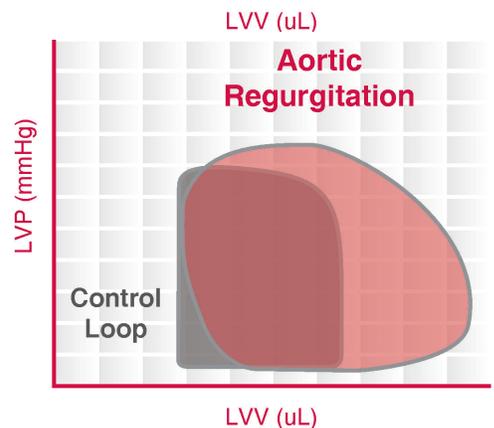
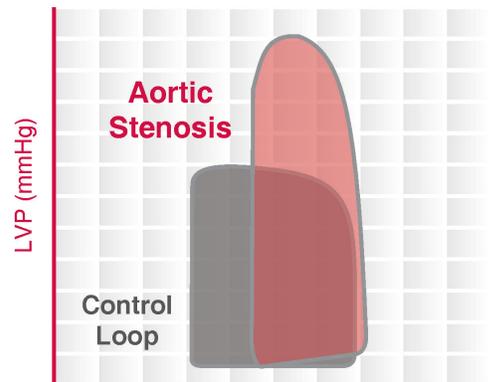
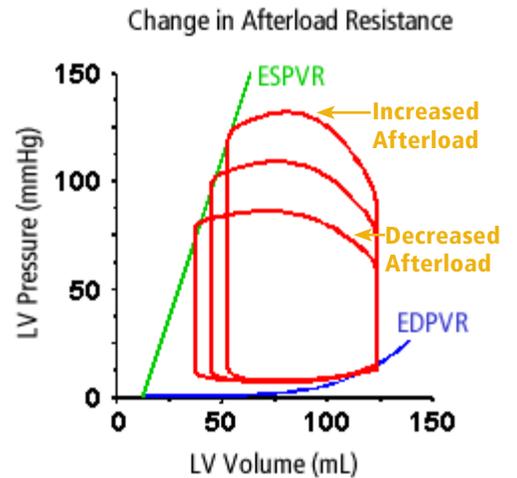
h = wall thickness

From this relationship it is apparent that wall stress (afterload) increases when the aortic or arterial pressure increases, ventricular radius increases (ventricular dilation), or wall thickness decreases.

Overall, wall stress is contractility dependent and is not constant during the contraction. LV cavity pressure, wall thickness and curvature vary with preload.

Afterload is composed of these major parameters: myocardial wall stress, arterial blood pressure, arterial resistance and arterial impedance.

Left ventricular afterload is affected by various disease conditions. Hypertension increases the afterload since the LV has to work harder to overcome the elevated arterial peripheral resistance and decreased compliance. Aortic valve diseases like aortic stenosis and insufficiency (regurgitation) also increase the afterload whereas mitral valve regurgitation decreases the afterload. Long-term afterload increases can lead to decreased stroke volume and deleterious cardiac remodeling.



## Understanding Afterload Cont.

Afterload cannot be measured directly, but several methods for assessing afterload indirectly are available. This is normally accomplished by characterizing the interaction between the heart and the arterial system.

### ECHOCARDIOGRAPHY

Echocardiography (both transthoracic and transesophageal) gives reliable measures of end systolic surface areas and wall thickness. In combination with ventricular pressure measurement this allows for the calculation of ventricular wall stress as described previously [1]. Additionally, long axis Doppler echocardiography can measure decreases in stroke volume (SV) during the period of increased afterload.

### COMBINED PRESSURE AND FLOW

Combination of pressure and flow measurement (transit-time Flowprobe and Pressure Catheter) can be used to determine afterload. Using flow as a measurement of cardiac output (CO) and both mean arterial pressure (MAP) and central venous pressure (CVP) can help to determine total peripheral resistance (TPR). Most investigators however omit CVP due to its minimal influence on the total, therefore:

$$TPR = (MAP / CO) = (MAP / SV * HR) \text{ [2]}$$

### PRESSURE-VOLUME LOOPS

Pressure-volume loops characterize afterload by total mechanical load on the ventricle during the ejection. During increased afterload heart muscle cells have to increase their metabolism while starting to use an oxidative phosphorylation pathway to obtain more ATP to handle calcium homeostasis and ultimately contract. Moreover, an increase in oxygen consumption will be characterized by an increase in PVA (pressure volume area) at the beginning of afterload challenge.

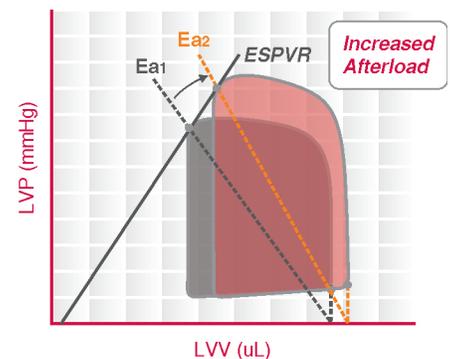
Afterload is also related to the arterial elastance (Ea), which is the ratio of ventricular end-systolic pressure to stroke volume:

$$Ea = ESP / SV$$

### REFERENCES

[1] Franklin RC, et al. "Normal values for noninvasive estimation of left ventricular contractile state and afterload in children." Am J Cardiol. 1990 Feb 15; 65(7): 505-10.

[2] Veras-Silva AS, et al. "Low-intensity exercise training decreases cardiac output and hypertension in spontaneously hypertensive rats." Am J Physiol. 1997 Dec; 273: H2627-H2631



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