What are (name) two physiological factors that directly affect left ventricular afterload; explain/discuss the effect(s) (on left ventricular afterload) of each factor. For each of these two direct factors name one (other) factor that affects the direct factor, either directly (on the direct factor) or through intermediates.

Two physiological factors directly affect left ventricular afterload: peripheral resistance and arterial compliance.

* Elevated systemic blood pressure increases the left ventricular afterloads because LV must work harder to eject blood into the aorta. Thinking of blood vessels as rigid pipes, using Poiseuille’s equation (Equation B&L[7] 17.3 of a flow which is proportional among other factors to the vessel radius), and using the equation of the resistance (R= ΔP/Q Equation B&L[7] 17.5), the vessel radius, r, is an important determining factor of arterial resistance and it is an indirect factor of the LV afterload.
* Compliance of the arteries is another direct factor affecting afterload. If the proximal aorta is stiff and non-compliant, it is resisting blood flow and storing none of the energy (see Module 8, Video 4, Slide 4).Therefore, to achieve the same systolic flow, the LV has to generate a higher pressure which translates into greater wall stress, i.e. an increase on afterload, because left ventricular afterload is related to ventricular wall stress and wall stress is expressed as: (P · r) / h where P: ventricular pressure, r: ventricular radius, h: wall thickness. Endothelial dysfunction results in reduced compliance (and usually is age related), and it affects compliance thus it is an indirect factor of LV afterload.

Two direct physiological factors directly affect left ventricular afterload: peripheral resistance and arterial compliance.

* Elevated systemic blood pressure increases the left ventricular afterloads because LV must work harder to eject blood into the aorta. Thinking of blood vessels as rigid pipes, using Poiseuille’s equation (Q = (𝜋 • ΔP • r4)/(8 • 𝜂 • l), Equation B&L[7] 17.3), and using the equation of the resistance (R= ΔP/Q Equation B&L[7] 17.5), the vessel radius, r, is an important determining factor of arterial resistance and it is an indirect factor of the LV afterload.
* Left ventricular afterload is related to ventricular wall stress. Wall stress is expressed as: (P • r) / h where P: ventricular pressure, r: ventricular radius, h: wall thickness.

Compliance of the arteries is another direct factor affecting afterload. If the proximal aorta is stiff and non-compliant, it is resisting blood flow and storing none of the energy (see Module 8, Video 4, Slide 4). Therefore, to achieve the same systolic flow, the LV has to generate a higher pressure which translates into greater wall stress, i.e. an increase on afterload. Endothelial dysfunction results in reduced compliance (and usually it is age related) and affects compliance and it is an indirect factor or LV afterload.

If an increase in heart rate altered cardiac output, arterial pressure would change; that is, afterload would be altered.

Left ventricular afterload is the pressure to open the aortic valve.

Peripheral vascular resistance is the main determinant of left ventricular afterload.

The factors that affect TPR are **blood viscosity**, total **vessel length**, and overall **vessel diameter**. When the **hematocrit** is high, such as for people living in high altitudes, the **viscosity** is greater and the blood is thicker and stickier.

* Length of the arterial tree (the longer the vessels, the greater the resistance)
* **Blood viscosity** (the higher the viscosity, the greater the resistance)
* **Vessel radius**(the smaller the radius, the greater the resistance)

**s***Systemic vascular resistance* (*SVR*), also known as total *peripheral* resistance

Any change in caliber of blood vessels alters the amount of blood that can flow through so dramatically, that reducing the diameter of a blood vessel by 1/2 allows for only 1/16 of the previous blood flow.

The viscosity of the blood also plays an important roll in SVR.[[6]](https://www.ncbi.nlm.nih.gov/books/NBK556075/) The more substances dissolved in the blood, the more viscous it will be.