

Frank-Starling Law of Cardiovascular Dynamics

BME 362 Lecture Notes *Ying Sun*

Cardiovascular Dynamics

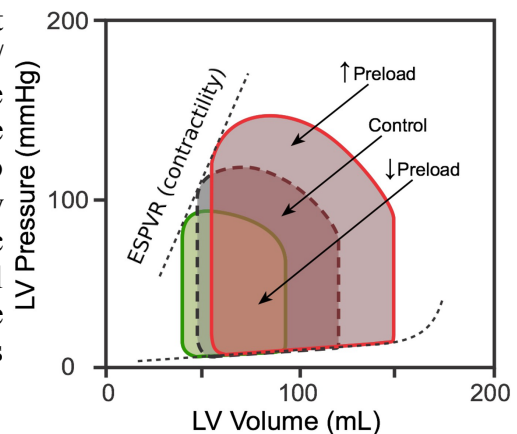
The main function of the heart is to pump blood. The right ventricle pumps blood into the pulmonary artery to drive the pulmonary circulation. The left ventricle pumps blood into the aorta to drive the systemic circulation. The following four variables affect the cardiovascular dynamics.

Heart rate: A normal resting heart rate for adults ranges from 60 to 100 beats a minute. Generally, a lower heart rate at rest implies more efficient heart function and better cardiovascular fitness. For example, a well-trained athlete might have a normal resting heart rate closer to 40 beats a minute.

Preload: Preload is the filling pressure of the heart at the end of diastole. The left atrial pressure at the end of diastole will determine the preload of the left ventricle. The greater the preload, the greater will be the volume of blood in the heart at the end of diastole. (Like blowing up a balloon, the more pressure that is applied, the bigger it will get.)

Afterload: Afterload is the pressure against which the heart must work to eject blood during systole (systolic pressure). The lower the afterload, the more blood the heart will eject with each contraction. Afterload is primarily determined by the resistance and capacitance of the systemic vascular bed. High blood pressure (hypertension) presents a larger workload to the heart and impedes cardiac output.

Contractility: Contractility is the intrinsic contractile function of the ventricle, independent of preload and afterload. Experimentally, contractility may be evaluated by pressure-volume loops. If a number of loops are obtained by varying preload, the coordinates at end-systole form a straight line, known as the end-systolic pressure/volume relationship. The slope of this line is a reasonably load-independent measure of contractility. Unfortunately, there is no simple method of assessing contractility clinically. Ejection fraction and cardiac output, the commonly used clinical parameters of cardiac performance, are influenced by loading conditions, as explained subsequently.



Frank-Starling Law

The Frank-Starling law (or Starling law) states: “The larger the volume of blood entering the heart during diastole (i.e. the end-diastolic volume), the larger the volume of blood ejected during systole (i.e. the stroke volume).”

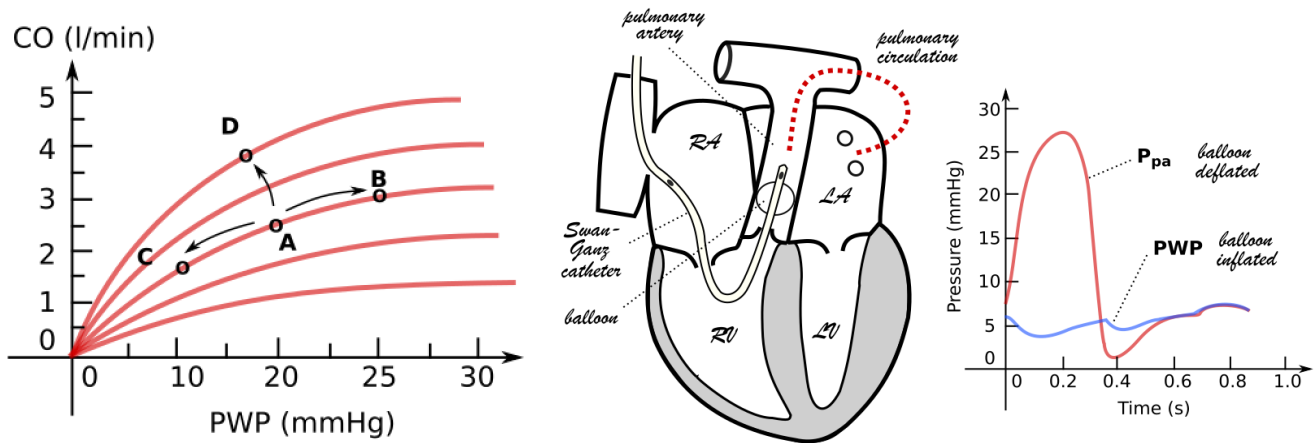
$$SV = EDV - ESV$$

$$EF = SV \div EDV$$

$$CO = SV \times HR$$

where SV: stroke volume
EDV: end-diastolic volume
ESV: end-systolic volume
CO: cardiac output
HR: heart rate

Clinically, the Starling curves are a family of curves by plotting cardiac output (CO) against the pulmonary wedge pressure (PWP) under different levels of contractility as shown in the figure below. Both CO and PWP can be obtained with the Swan-Ganz catheter. CO is obtained by thermodilution as discussed previously. As shown in the figure, the Swan-Ganz catheter has an end hole that can access the pulmonary arterial pressure (P_{pa}) with the balloon deflated. When the balloon is inflated, the blood flow is blocked momentarily. The pressure measured at the tip of the catheter is the pulmonary wedge pressure (PWP), which is about the same as the left atrial pressure (P_{la}). This phenomenon can be explained by use of the Ohm's law. Let's model the pulmonary circulation as a simple resistance. The pressure difference between P_{pa} and P_{la} is the flow times the resistance. If the flow is zero, there should be no pressure difference between P_{pa} and P_{la} . Thus, the catheter can access the the left atrial pressure via the pulmonary circulation by momentarily stopping the flow.



The Starling curves have been used to manage patients with congestive heart failure (CHF) in the Cardiac Care Unit of the hospital. PWP is an measurement of the preload, which can be controlled by fluid management. For example, the present operating point is A, which corresponds to CO = 2.5 l/min and PWP = 20 mmHg. Moving the operating point to B by retaining fluid, CO can be raised due to a higher PWP (preload). While a higher CO can be beneficial, the high PWP may accentuate pulmonary edema and increase breathing difficulty. Another adverse effect of high PWP is hypertension. Conversely, the fluid volume can be decreased by moving operating point C with a diuretic drug. Diuretic drugs increase the flow of urine (termed diuresis) by excreting sodium and chloride to the urine, and the sodium and chloride in turn draw excess water from the body. Examples of the diuretics include furosemide, hydrochlorothiazide, spironolactone, and acetazolamide.

If the present Starling curve is too low and flat, the operating point could be moved to point D on a higher curve. This curve corresponds to a higher contractility of the left ventricle. This may be achieved by use to an inotrope, which is an agent that alters the force or energy of muscular contractions. Examples of a positive inotropic agent include digoxin, inamrinone, milrinone, and beta adrenergic receptor agonists.

Afterload management could be done with vasodialators (such as hydralazine, ACE inhibitors, and minoxidil) and vasoconstrictors (such as amphetamines, antihistamines and cocaine). However, managing afterload has been controversial. Lowering the systemic arterial pressure could reduce the work of the left ventricle. However, a lower blood pressure reduces perfusion and could have significant adverse effects.

Autoregulation of the Preload

Atrial natriuretic peptide (ANP) is a peptide hormone which reduces an expanded extracellular fluid (ECF) volume by increasing renal sodium excretion. ANP is synthesized, and secreted by cardiac muscle cells in the walls of the atria in the heart. These cells contain volume receptors which respond to increased stretching of the atrial wall due to increased atrial blood volume. The discovery of ANP was reported by de Bold et al. [Life Sciences 28: 89, 1981], who found a substance in rat atrial extracts increasing salt and urine output in the kidney. Later, the substance was purified from heart tissue by several groups and named atrial natriuretic factor (ANF) or ANP [de Bold AJ, Science 230: 767, 1985].

When preload is elevated, the stretched atrial muscles release ANP. ANP acts on the kidney to lower preload by increasing sodium and water excretion (natriuresis) via the renin-angiotensin II system. ANP has the opposite effect of aldosterone on the kidney: aldosterone elevates preload by increasing renal sodium retention.

The blood pressure is mainly regulated via the carotid baroreceptors. When the blood pressure is too high, the baroreceptors signal the brain to reduce the heart rate. This is accomplished by modulating the firing rate of the sinoatrial (SA) node via the parasympathetic (vagus) nerve. The diagram below summarizes the neuronal and hormonal control mechanisms for the heart rate and the preload.

