CHAPTER 6
BASIC CARDIAC PHYSIOLOGY

At this point, the student should have a working knowledge of cardiac and vascular anatomy, and understand basic physiological principles such as compliance, elastance, and resistance. Using this basic knowledge we will construct a foundation in cardiac physiology.

Section 6.1 Basic Cardiac Physiology Learning Objectives

1. End diastolic volume, EDV
2. End systolic volume, ESV
3. Stroke volume, SV
4. Minimum filled volume (dead volume), Vo
5. Cardiac cycle (time domain)
   • Isovolumic relaxation
   • Isovolumic contraction
   • Ejection
   • Filling
6. PV loop (pressure-volume domain)
7. External work
8. Potential work
9. Starling’s law
10. Basic Cardiac Muscle physiology

Section 6.2 Cardiac Volumes Defined

The heart operates on a cyclical contraction basis. As the cardiac muscle contracts, pressure inside the ventricles increases, and if the ventricular pressure exceeds the aortic (or pulmonary) pressure, ejection of blood volume occurs. The amount of blood expelled from one ventricle during a single contraction cycle is termed the stroke volume (SV). It is the difference between the volume in the ventricle at the start of the contraction process and the volume left in the ventricle at the end of the contraction process. The volume in the ventricle at the start of the contraction process is termed, end-diastolic volume (EDV). The end diastolic volume is the largest volume of the ventricle during that particular contraction cycle. After expelling a stroke volume, the resulting volume left in the heart is
termed end-systolic volume (ESV). The end-systolic volume is the smallest volume in the ventricle for that particular contraction cycle.

\[ SV = EDV - ESV \]

The ratio of stroke volume to end-diastolic volume is termed ejection fraction (EF).

\[ EF = \frac{SV}{EDV} \]

Substituting the stroke volume equation into the ejection fraction equation and rearranging it results in:

\[ EF = 1 - \frac{ESV}{EDV}. \]

In Chapter 4 we discussed a blood vessel’s dead volume, \( V_0 \). The heart also has a “dead” volume, \( V_0 \). If a ventricle is completely void of blood, it will take a certain amount of blood volume to fill the empty ventricle and just begin stretching the myocardium -- this volume is termed the dead volume.

Now that we’ve defined the volumes of the contracting heart, we can construct the time domain behavior of the heart – the cardiac cycle. A keen understanding of the cardiac cycle is imperative for the cardiovascular engineer. The cardiac cycle is shown graphically in Figure 6.1. We will start the analysis of the cardiac cycle by beginning with the electrical stimulus for contraction, denoted by the bottom most ECG plot (the QRS peak). Shortly after this electrical event, the heart muscle begins to contract at EDV, causing the blood pressure within the ventricle to increase. At this point the ventricular pressure is greater than the atrial pressure and the A/V valve is closed. The semi-lunar valves are also closed because the aortic and pulmonary artery pressures are greater than their respective ventricular pressures. Because both valves are closed, no volume can leave the ventricle. This is known as the isovolumic contraction phase.
FIGURE 6.1 Cardiac Cycle (time domain)
As the cardiac muscle continues contracting, the ventricular pressure rises isovolumically until the ventricular pressure exceeds the aortic (or pulmonary artery) pressure, at which time the semi-lunar valves open and the heart begins to eject. Note that the ventricular volume curve begins to descend until it reaches ESV. End of ejection occurs when the ventricular pressure falls below the aortic (or pulmonary artery) pressure and the semi-lunar valves shut. The change in volume from EDV to ESV is the stroke volume and this phase is aptly termed the ejection phase. As the ventricular pressure falls both the A/V valves and now the semi-lunar valves are closed resulting in the isovolumic relaxation phase. This phase continues until the ventricular pressure falls below the atrial pressure, which causes the A/V valves to open and thus starting the filling phase of the ventricle. Towards the end of the filling phase, the atria contract and produce a rise in ventricular pressure in both ventricles as a small amount of blood is forced into the ventricles by the atrial contractions. Now the ventricles begin to contract due to the ECG (QRS) stimulus and the process repeats itself.

Thus there are four major divisions of the cardiac cycle; isovolumic contraction, ejection, isovolumic relaxation, and finally filling. The cardiovascular engineer should be able to identify the individual waveforms, phases, valve opening and closing points, volume changes and the electrical stimulus.

Another way to view the cardiac cycle is through a plot of pressure and volume, known as the pressure-volume (PV) loop. Figure 6.2 depicts a typical PV loop. Once again, after the QRS electrical event, the myocardium begins to contract at EDV, causing the blood pressure within the ventricle to increase. Looking at just the left heart, both the biscupid and aortic valves are closed, resulting in the isovolumic contraction phase. As the cardiac muscle continues contracting, the ventricular pressure rises isovolumically until the ventricular pressure exceeds the aortic pressure (AoP), at which time the ejection phase begins. End of ejection occurs when the ventricular pressure falls below the aortic pressure and the ventricle has expelled its stroke volume.
Now that both the aortic and mitral valves are closed the isovolumic relaxation phase begins. This phase continues until the ventricular pressure falls below the left atrial pressure (LAP), which causes the A/V valves to open and thus starts the filling phase of the ventricle. Now, the ventricles begin to contract due to the ECG (QRS) stimulus and the process repeats itself. Thus, there are four major divisions of the PV loop, isovolumic contraction, ejection, isovolumic relaxation, and finally, filling. The cardiovascular engineer should be able to identify the phases on a PV loop.

**Left Ventricular Pressure**

![Figure 6.2 PV Loop](image)

The area within the PV loop is referred to as the external work performed by the myocardium on the ventricular blood. As the stroke volume increases and/or the ventricular pressure during ejection increases, so will the external work created by the “pump”. Figure 6.3 shows the external work (red shading).
Now, let’s examine the filling phase in more detail. If the heart didn’t contract, the heart would be very similar to a “water” balloon. We could add and subtract volumes from the non-contracting heart and measure the resulting pressures. The result might look like Figure 6.4. If one reduced the volume in the ventricle until the pressure was zero, this would be the heart’s dead volume, $V_o$. To remove any more ventricular blood, the pressure would have to be brought below zero and the heart would start to collapse. Now as we begin to fill the non-contracting ventricle with blood the pressure rises but not in a linear fashion. For most cases, the filling phase is similar to the diastolic, or non-contracting properties, of the myocardium.
Reducing the inflow to the right heart will also reduce the volume in the right ventricle which, in turn, reduces the ventricular pressure. Therefore, the EDV and pressure will fall along the filling curve in Figure 6.3. In addition, recalling Figure 4.10, the outflow of the heart will also reduce and the left heart will also experience decreased filling pressures and volumes. Conversely, if one increases the EDV by increasing venous volume, the filling phase ventricular pressures and volumes will increase along the filling curve. The resulting PV loops at different EDVs are shown in Figure 6.4. One can draw a tangent to the upper left corners of the PV loop. By extrapolation, this line comes close to the ventricle’s $V_o$. This line is referred to as the **End Systolic Pressure Volume Relation** (ESPVR) and its slope has the units of elastance. This characteristic of the PV loop has been used for many years as an index of cardiac performance and it is important for the cardiovascular engineer to have a good understanding of its basis and relevance.
Now, let us allow the heart to contract at different EDVs, but not eject any volume by clamping the outflow vessels. As shown in Figure 6.4, as the EDV gets larger the peak pressure generated in a totally isovolumic beat also gets larger up to a certain point. Beyond that point, the peak pressure begins to decline, even though the EDV continues to get larger. There comes a point, where the amount of pressure in the ventricle at the start of the isovolumic contraction doesn’t change throughout the “contraction”. One can “connect the dots” of the peak pressures and define the operating envelope of that particular ventricle as shown in Figure 6.5. The operating envelope can change with neural and humoral activities. It should be noted that the line formed by connecting the dots of the peak pressures will differ slightly from the ESPVR and the reasons for this will be discussed later.
The global behavior of the heart can be better understood by examining cellular properties of cardiac muscle. As we learned in Cardiac Anatomy Unit 3, the cells of the heart interconnected with intercalated disks (see Figure 6.6), and because of the excellent cell-to-cell communication the cells appear to be one large cell (the syncytium). If one delves further into the cardiac muscle cell, striations similar to skeletal muscle appear. These striations are due to the structure of the contractile machinery. Figure 6.7 illustrates the source of the striations.

**FIGURE 6.5** Isovolumic contractions at different filling volumes
The anchor of the force generating unit – the sarcomere – is the Z disk. Attached to the Z disk and extending towards another Z disk are filaments called actin.

Figure 6.6 The “syncytial,” interconnecting nature of cardiac muscle.

Figure 6.7 Sarcomere Structure
The bridge between the actin filaments (one from each Z disk) is myofilaments called myosin. When viewed by a light microscope where the myosin and actin overlap is termed the A-band. The space between the Z disk and the end of the myosin is the I-band. The H-band is the space between the ends of the actin filaments. A contraction occurs when the myosin heads “ratchet” along the actin filaments. This causes the A-band to get larger, and the H- and I-band to get smaller.

Thus, as the contraction progresses the sarcomere shortens by pulling the Z disks closer together. When relaxation occurs, the sarcomere is pulled back by external forces, increasing the distance between the Z disks. As it turns out, there are optimum settings for the sarcomere. Figure 6.8 illustrates the dependence of sarcomere length and the force it can generate.

Figure 6.8. Length-tension diagram for a single sarcomere, illustrating maximum strength of contraction when the sarcomere is 2.0 to 2.2 microns in length. At the upper right are shown the relative positions of the actin and myosin filaments at different sarcomere lengths from point A to point D. (Modified from Gordon, Huxley, and Julian: J. Physiol., 171:28P, 1964.)
Here the maximum force is generated at a sarcomere length of 2.2 microns. If the length varies from either side of this length, the amount of force generated drops off significantly. This sarcomeric property is the basis for much of the volume dependence of the heart. At low volumes (low levels of sarcomere stretch) the heart can produce little pressure (smaller sarcomeric force). At larger levels of stretch (larger sarcomere length) the heart can generate higher pressures (higher sarcomeric force). But, if the volume of the heart gets too large, (over-stretched sarcomeres) the heart can produce less pressure and this, in part, explains the operational envelope of the heart as depicted in Figure 6.5. The difference of the upper curve (total pressure developed) and the lower curve (amount of pressure stretching the myocardium) is the amount of active pressure that the heart can develop. When the pre-contraction pressure (force) equals the amount of total pressure (force) that the myocardium can generate, there is no active force.

The combination of increased stretch of the myocardium (up to a certain point) and the increase in cardiac pressures and cardiac output is referred to as Starling's law of the heart and it has its basis in cardiac muscle physiology.

While there will be more to say about the PV loop later in this course, this chapter provides a basic overview of cardiac physiology. In the next few chapters we will modify this view somewhat.