

CHAPTER 7 (Draft) ADVANCED CARDIAC CONCEPTS

The purpose of this chapter is to expose the cardiovascular engineer to more advanced cardiac concepts. The rationale is that by understanding cardiac function in more complete terms, insights into better diagnostics and therapies can be achieved.

Chapter 7 learning objectives:

1. Time varying elastance concept
2. Mechanical and electrical models of time-varying elastance
3. Equations defining time-varying elastance
4. Infinite resistance PV loop
5. Infinite capacitance PV loop
6. Problems with time-varying elastance
7. Source parameters (visco-elastic properties)

7.1 Time-varying Elastance Concept

To begin our study of the cardiac time-varying elastance, let us examine a simple coiled spring (Figure 7.1). A real spring has an initial unstretched length, L_0 . Now, to stretch the spring we must apply a force to it. The greater the force, the greater the length.

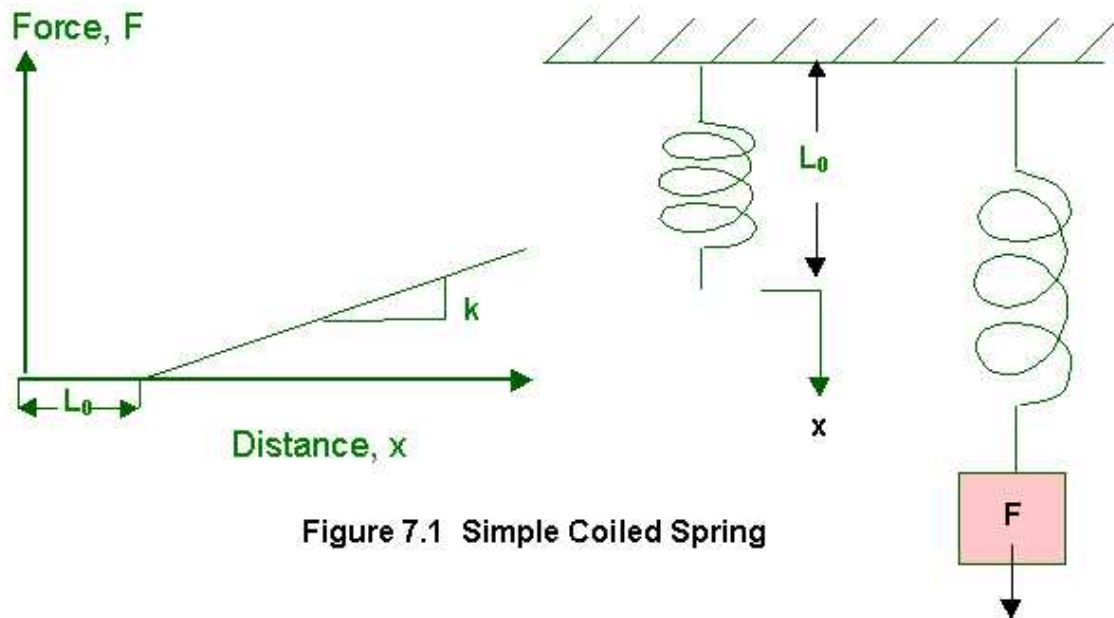
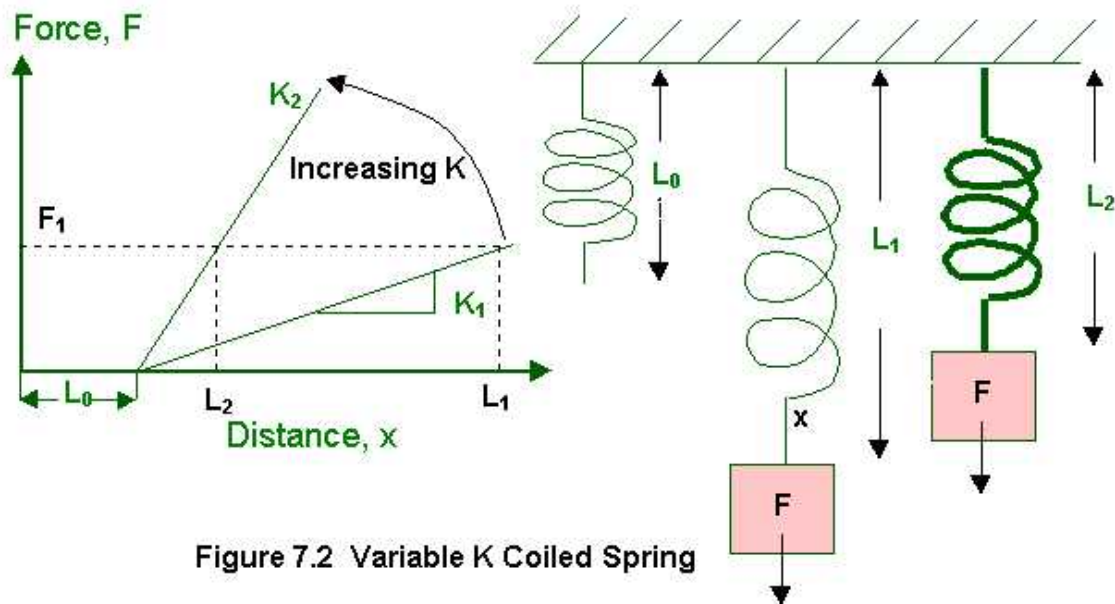


Figure 7.1 Simple Coiled Spring

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The slope of the force-distance relationship is termed the “spring-constant”, K . The stiffer the spring, the larger K will be and the more force is required to lengthen the spring. Let’s make the spring constant, K , variable. Further, let’s assume that if we electrically stimulate this special spring, the spring constant will increase. One can visualize this by a much thicker, stronger spring. So let’s take the elongated situation of Figure 7.1. We’ve stretched the spring by hanging a weight, F , from L_0 to some new length, L_1 .



At this point let’s apply some electrical stimulation to the spring and as a result the spring becomes thicker and stronger. This will tend to pull the weight up, because it is equivalent to hanging the weight from a thicker, stronger spring and having it stretch less. The more we electrically stimulate the spring, the thicker and stronger it becomes and the more we shorten the spring resulting in lifting weight, F , even higher, as shown in Figure 7.2.

If the spring shortens with a constant force it is termed isotonic. If the spring is electrically stimulated and not allowed to shorten (clamped firmly at both ends) it is termed isometric. Figure 7.3 depicts an isometric process. In an isometric

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process, the spring doesn't shorten, but the force generated increases from F_1 to F_2 due to the changes in K .

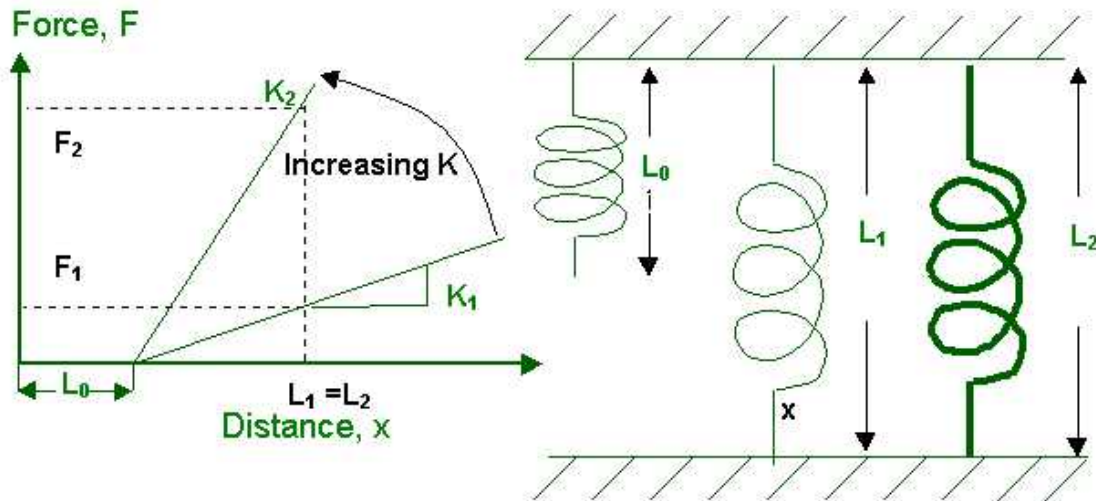


Figure 7.3 Variable K Coiled Spring (Isometric)

To an approximation, cardiac and skeletal muscle can be represented by the idea of a spring that can change its spring constant. The varying spring constant, K , is the one dimensional version of time-varying cardiac elastance. Cardiac elastance, however, relates pressure (force/area) and volume (area x height). Rather than isometric and isotonic processes, we speak of isovolumic and isobaric processes, respectively. Rather than a force-length curve, we speak of pressure-volume curves. Rather than an unstretched length, L_0 , we speak of filling volume, V_0 . Thus, the concepts of a mechanical spring with a variable spring constant and cardiac muscle with a time varying elastance are quite similar. We can also represent a time varying elastance using an electrical component – the capacitor. Instead of pressure, we can use voltage and instead of volume we can refer to charge. Thus, we can convert cardiac concepts into either mechanical or electrical terms.

Armed with this information, let us return to the PV loop and examine it from a time-varying elastance point-of-view. First, let's construct a model to help us view pressure volume loops. Figure 7.4 shows such a model. Here we have a

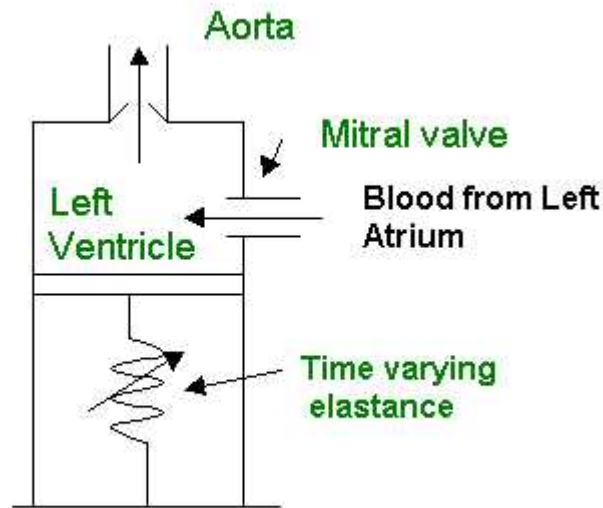


Figure 7.4 Mechanical Model of Ventricle

compressed time-varying elastance connected to a piston. As the elastance increases, the compressed elastance becomes stronger and tries to “push” the piston upward. In actuality, the elastance shortens, but for graphical simplicity, the elastance is shown here to lengthen. The results derived for this are no different than if the elastance shortened. In any event, as the elastance gets stonger, it pushes against the piston isovolumically until the pressure inside the ventricle gets larger than the aortic pressure, at which time the aortic valve opens and the spring is allowed to lengthen, ejecting volume. As it lengthens, the piston sweeps out a stroke distance. The amount of stroke of the piston (multiplied by the cross sectional area of the cylinder) is the stroke volume. If the elastance now decreases, the ventricular pressure falls, the aortic valve closes and the elastance decreases isovolumically until the ventricular pressure falls below the atrial pressure. Now the heart fills with blood and the process continues. Let’s view this process on the PV plane in Figure 7.5. Starting at point 1, the elastance is represented by the diastolic or filling elastance. Now the elastance begins to increase to point 2. Currently most researchers believe that V_0 remains constant and that the elastance line rotates about V_0 . Similar to the variable spring constant example, the elastance continues to increase to point 3 and then to point 4. This process from point 1 to 4 is the isovolumic contraction

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phase. Once the ventricular pressure rises above the aortic pressure ejection begins (point 4). Depending on what the heart is ejecting to, a complex pressure volume trajectory is followed until point 6 is reached. Here the ESPVR is reached and from this point on the elastance begins to decrease isovolumically until point 9 where ventricular pressure falls below atrial pressure and filling begins. As filling progresses along the diastolic elastance the pressure in the ventricle rises until point 1 is reached and the process starts all over again.

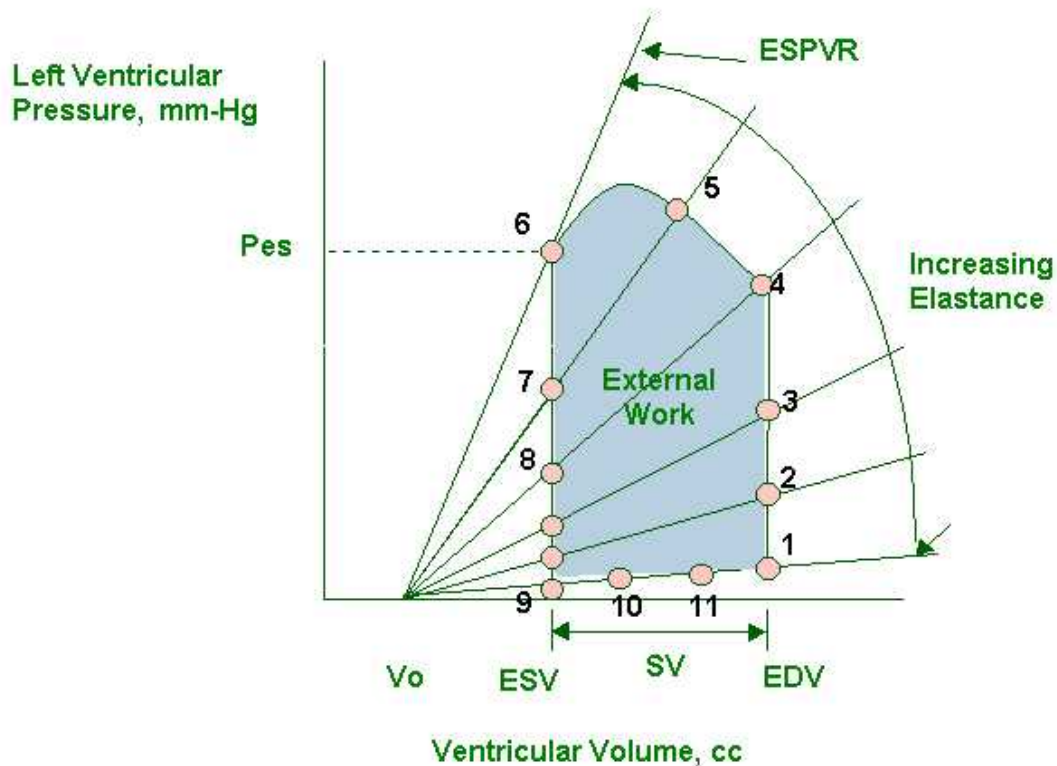


Figure 7.5 PV loop and time Varying Elastance

The work that the elastance performed is the external work area and the area between ESPVR, diastolic filling, V_o and ESV is the potential work. Thus, our first model of the heart can be thought of as a time-varying elastance. Armed with this knowledge, we can examine what the effects of various arterial loads on the heart might be when viewed from the PV loop.

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First, let's place an infinite resistance on the outflow of the ventricle. This could be accomplished by clamping the aorta shut and not allowing any ejection to occur. The result would be a totally isovolumic beat and would look similar to Figure 7.6.

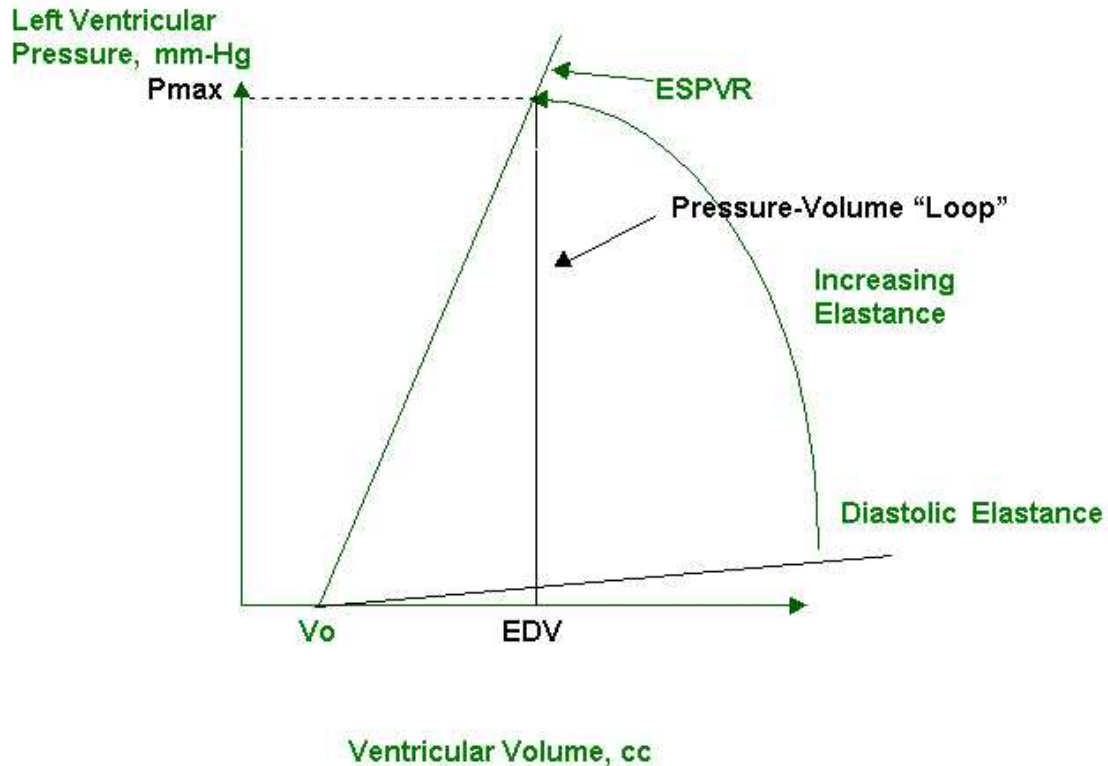


Figure 7.6 Infinite Load Resistance PV "Loop"

Thus, for an infinite load resistance, no outflow can occur and the PV loop becomes a straight vertical line. The maximum pressure during the PV loop will be attained at ESPVR, according to this model.

Another interesting type of arterial load is a purely compliant vessel, essentially a balloon with compliance, C , attached to the outflow of the ventricle (Figure 7.7). Here as the heart ejects its volume, the balloon receives that volume and the pressure in the balloon and ventricle will be approximately the same. Thus as we add volume to the balloon its pressure increases in a linear fashion. At the point

the ESPVR is reached, ejection will cease. The point of these various examples is to show that the filling phase profiles are determined, in part, by the type of the load attached to the outflow of the ventricle.

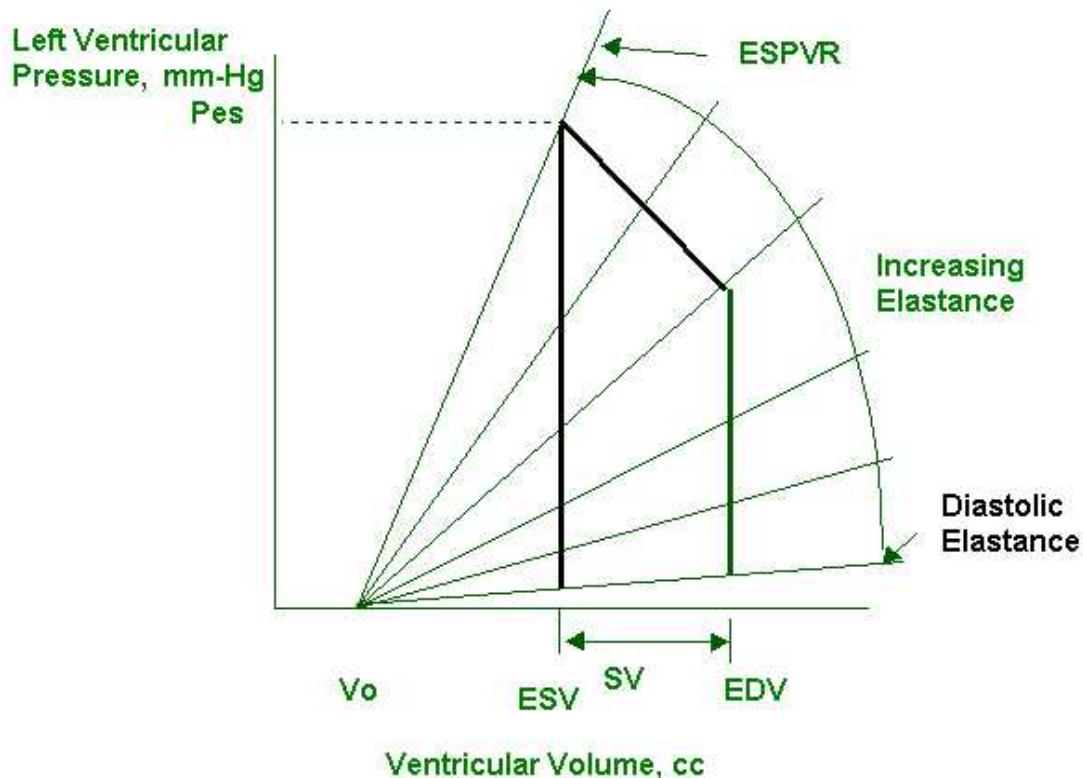


Figure 7.7 PV loop for compliant arterial Load

Let's try another example. In this case, let's apply a balloon with a very large compliance. Let's make compliance so large that any volume added to the balloon does not increase the pressure. Figure 7.8 shows the ejection phase for this situation. Because the large compliance doesn't experience a pressure increase when volume is added to it, the ejection phase of the PV loop will be horizontal.

In the final example, we will place a finite compliance that has a leak in it. Thus, as we push more volume into the compliance, some will leak out again reducing the pressure. This PV loop can take on an infinite number of profiles depending on the relative size of the compliance and the amount of leak. One possible

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profile for this situation is shown in Figure 7.9. As luck would have it, this one looks similar to actual PV loops and might indicate that the arterial load on a ventricle may have compliant and resistive properties. The rapid initial upswing in the pressure is because of the compliance and the later fall in pressure is due to the leak resistance. Figure 7.10 shows the ventricle model with an arterial model. Important in all this is that the afterload on the heart will affect measures of its performance.

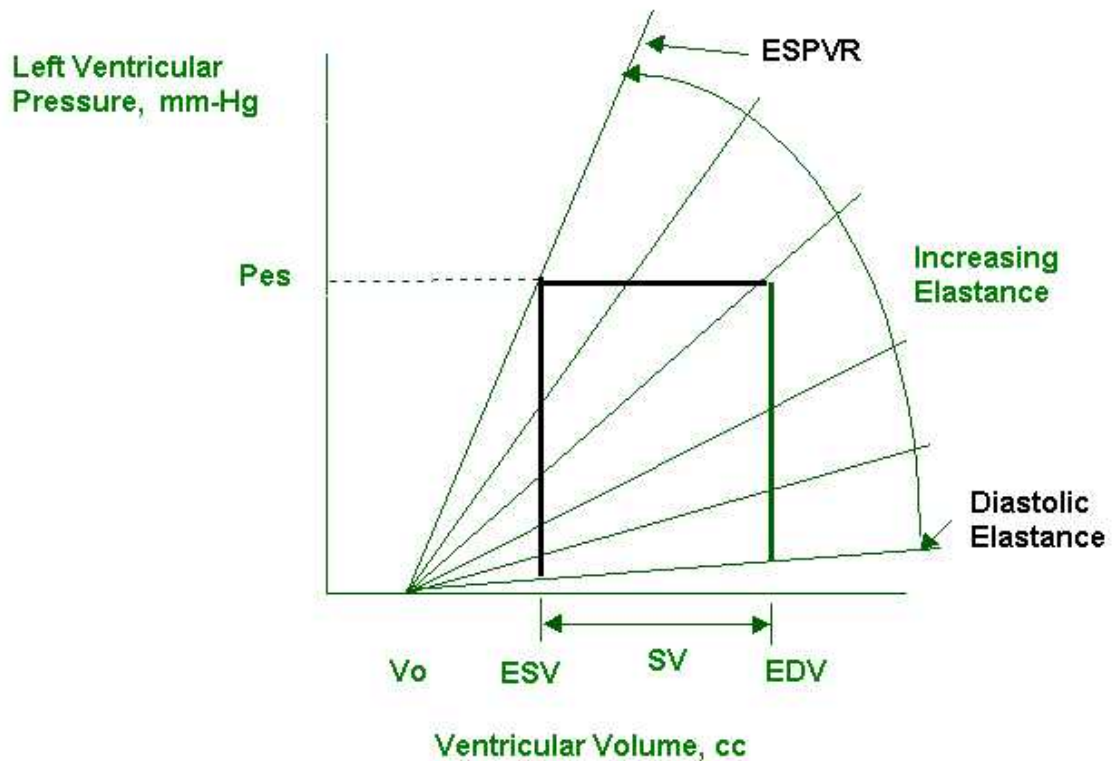


Figure 7.8 PV loop for very large compliant arterial load

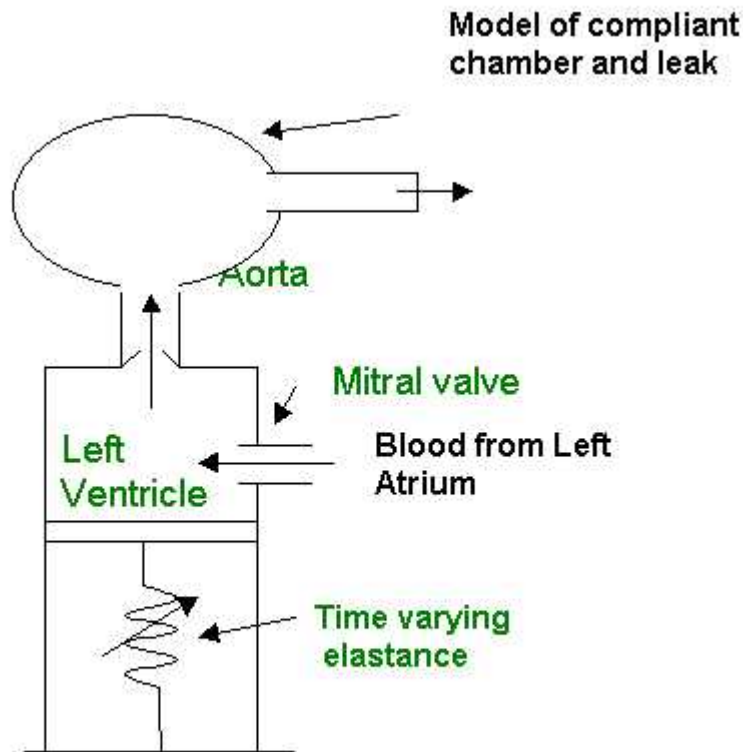


Figure 7.10 Mechanical Model of Ventricle with Compliant and Resistive Arterial model

Section 7.3 Mathematical Descriptions

The time-varying elastance is described by the equation for a straight line:

$$LVP = \varepsilon(t)[EDV - V_o - V_{EJ}(t)]$$

where:

LVP = left ventricular pressure

$\varepsilon(t)$ = Time Varying Elastance

EDV = End Diastolic Volume

V_o = Filling Volume

$V_{EJ}(t)$ = Volume Ejected as a Function of Time

and,

$$V_{EJ}(t) = \int AoF dt$$

$$V_{EJ}(t_{EJ}) = SV = \int_{Ejection} AoF dt$$

where:

AoF = Aortic Flow

t_{EJ} = total time of ejection

The aforementioned set of equations describes cardiac function from the point of view that the driving element of a cardiac contraction is a variable elastance. This time varying elastance generates an increase in pressure and ultimately sweeps out a volume – thereby delivering volume to the arterial (or pulmonary) system. Because cardiovascular engineers come from various backgrounds we will also represent cardiac function in circuit representation. Just as a spring stores mechanical energy, a capacitor stores electrical energy.

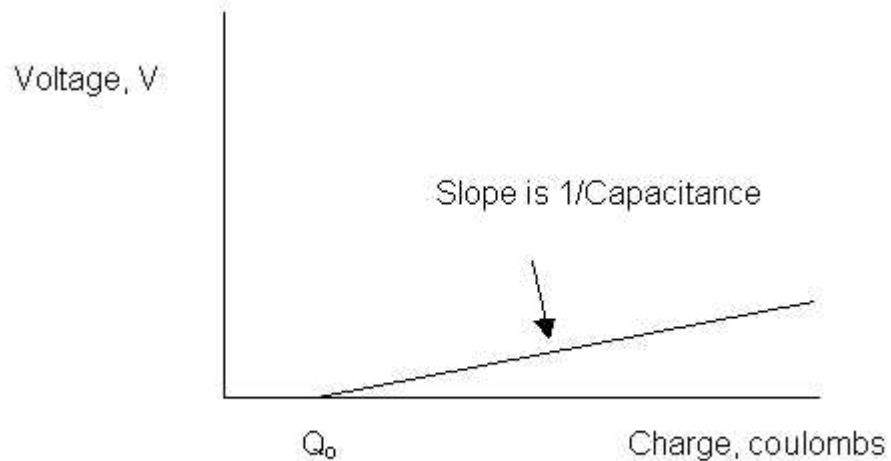


Figure 7.11 Electrical Capacitor

This plot is similar to the force-distance curve for a spring. In this regard, voltage is analogous to pressure and charge is analogous to volume. Thus one can have a time varying electrical capacitor (compliance) that will mimic a basic property of cardiac function.

Figure 7.12 illustrates the mechanical and electrical models of the heart. In summary, the heart is a three-dimensional object, whose walls are constructed of a contractile material that is oriented in many directions. We can abstract this into a regular geometry (such as a sphere or ellipsoid) that has a contractile material shell. However, this requires a three-dimensional analysis, thus we can opt for a simpler view by using a lumped one-dimensional models such as those shown in Figure 7.12. No model is perfect and with each choice of model comes

its own limitations. Usually a good principle to follow is to use the simplest model that sheds light on the information desired.

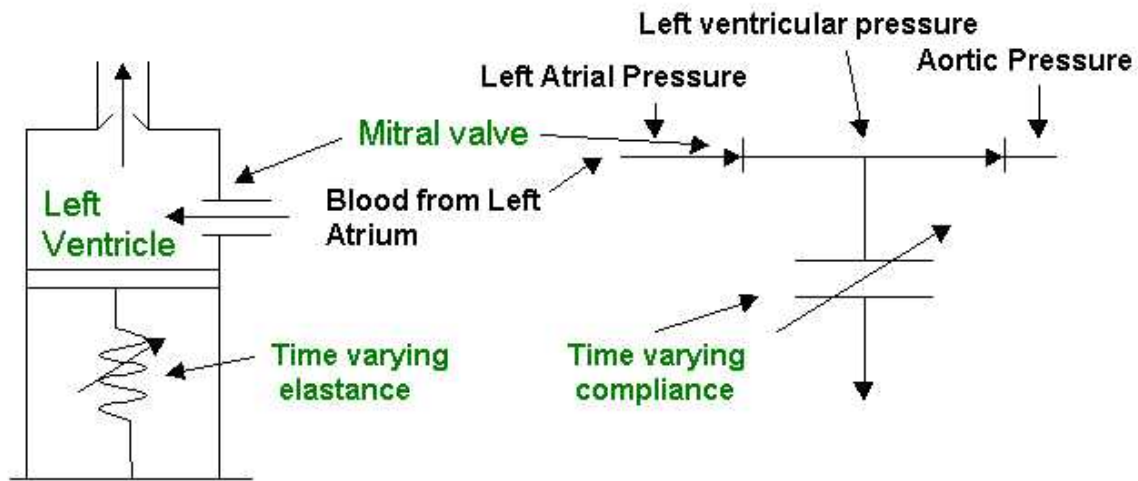


Figure 7.12 Mechanical and Electrical Model of Ventricle

If one looks at examining cardiac function, it quickly becomes clear that finding the elusive load-independent metric that reflects cardiac function becomes difficult. For example, the maximum pressure a ventricle can generate is dependent on the initial stretch of the myocardium, that is to say pre-load and the amount of volume it ejects is dependent on afterload. The greater the pre-load the greater the pressure, thus just measuring pressure and/or flow will not be adequate to estimate cardiac performance. Our present model, albeit a simple one, introduces another concept, termed elastance, which may be a better estimate of cardiac performance, but as we will see in later chapters, this also has its limitations.

If we measure LVP, EDV, V_o and V_{EJ} we can estimate the time varying elastance of the myocardium surrounding the ventricle from the above equation. If the simple model is a good one we should be able to describe both isovolumic beats and ejecting beats.

Let's use a test set of data to examine the effectiveness of our model. We'll use two cardiac loading conditions – ejecting and non-ejecting. If our model is a

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good one, it should predict the cardiac behavior in both instances. But why would we use two loading conditions – specifically the non-ejecting beat? You may remember from basic circuits class that one way to characterize an energy source such as a voltage or current source is to test it under open circuit (infinite load resistance) and short circuit (zero load resistance) conditions. We can obtain the former condition easier in the intact cardiovascular system by clamping the aorta (a non-ejecting beat).

Figure xx shows a time sequence of an ejecting beat followed by an isovolumic beat. Look closely at the isovolumic beat and you'll see that it has a greater amplitude and duration than the ejecting beat.

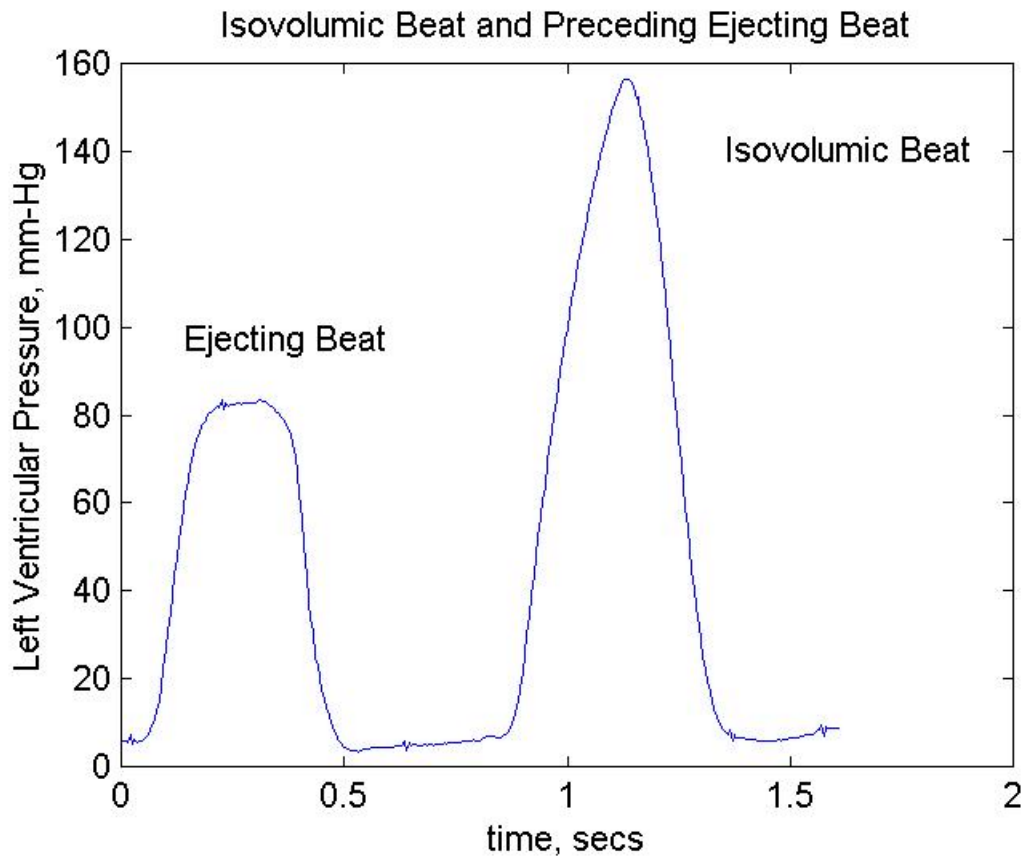


Figure 7-13

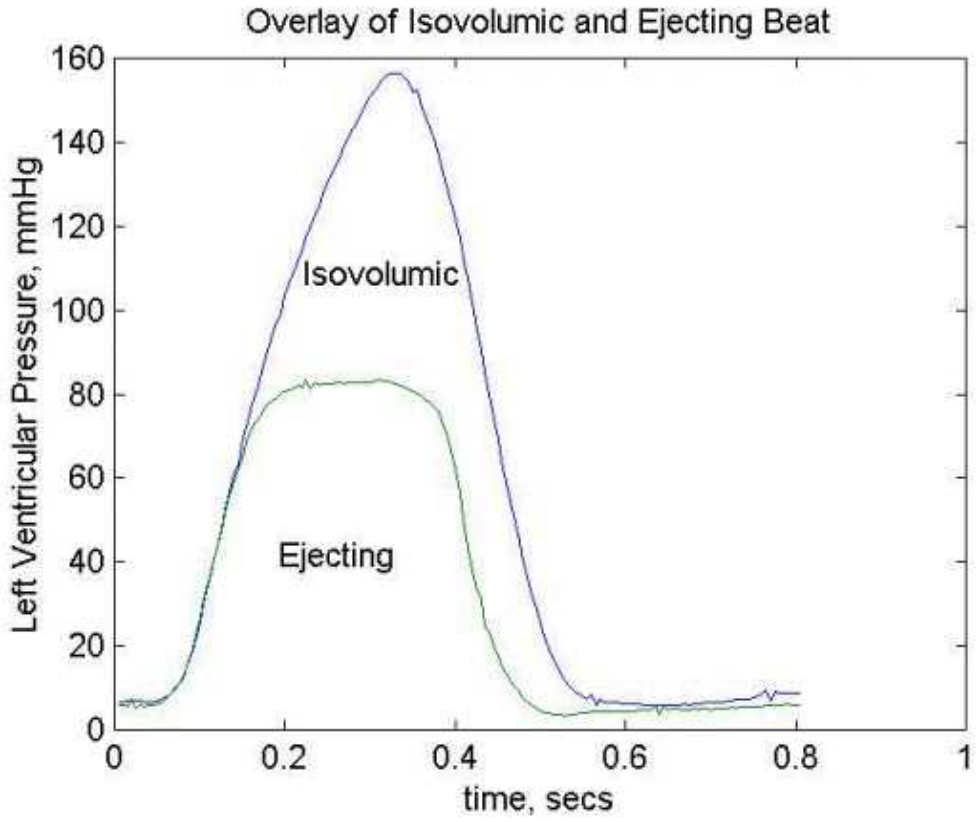


Figure 7-14a

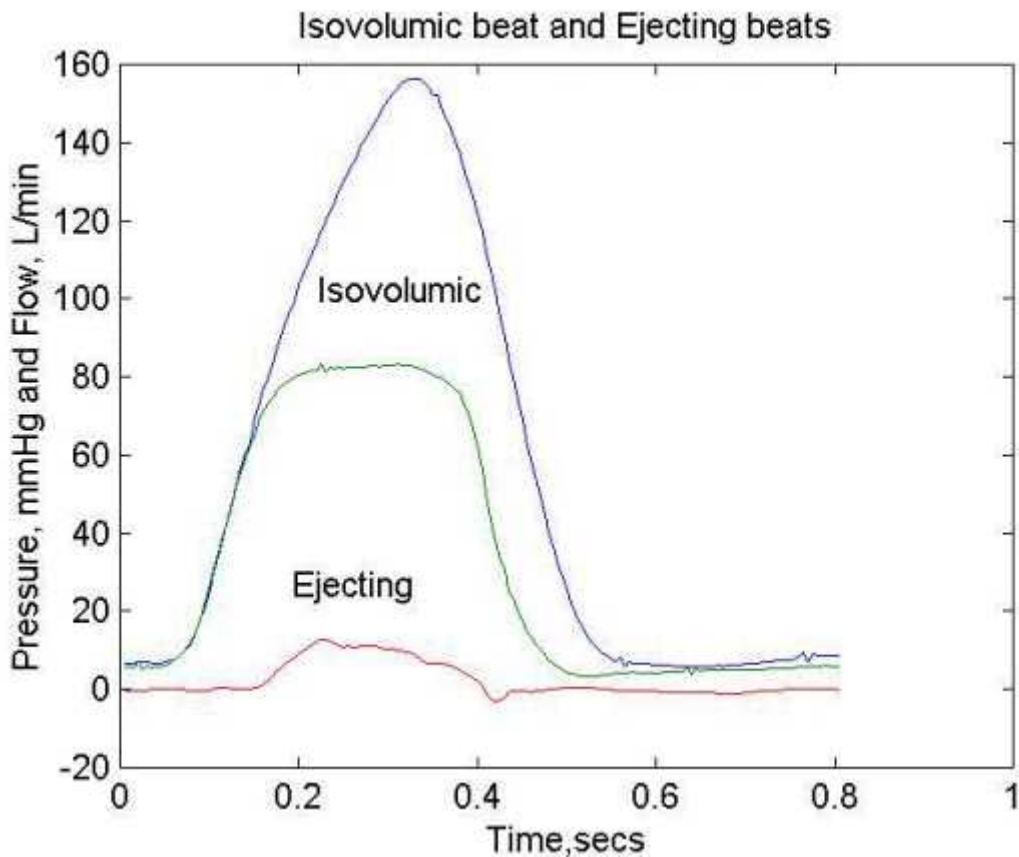


Figure 7-14b

Figure xx illustrates this better by aligning the isovolumic phases of both beats. If the beat started from the the same EDV, the isovolumic phases should be the same and deviation from the this similarity will end at the time ejection begins. This can be seen in figure xx.

Now let's apply our model to this dataset. We'll do this by calculating the elastance from the isovolumic beat by the equation:

$$\varepsilon(t) = \frac{P_{ISO}}{[EDV - V_o]}$$

V_o was estimated from an IVC occlusion experiment and was found to be 10 cc. EDV was found by measurement to be 85 cc. Thus we can calculate the elastance waveform from these data (figure xx). Using this elastance we can see

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if our model will predict LVP from the PISO now let's calculate the elastance for the ejecting beat. Here we'll use the equation from our model:

$$\varepsilon(t) = \frac{P_{ISO}}{[EDV - V_o - V_{EJ}]}$$

One can clearly see that the elastances calculated from the isovolumic and ejecting data are different. Perhaps, the elastance is altered in some way by the process of ejection, or perhaps _____???

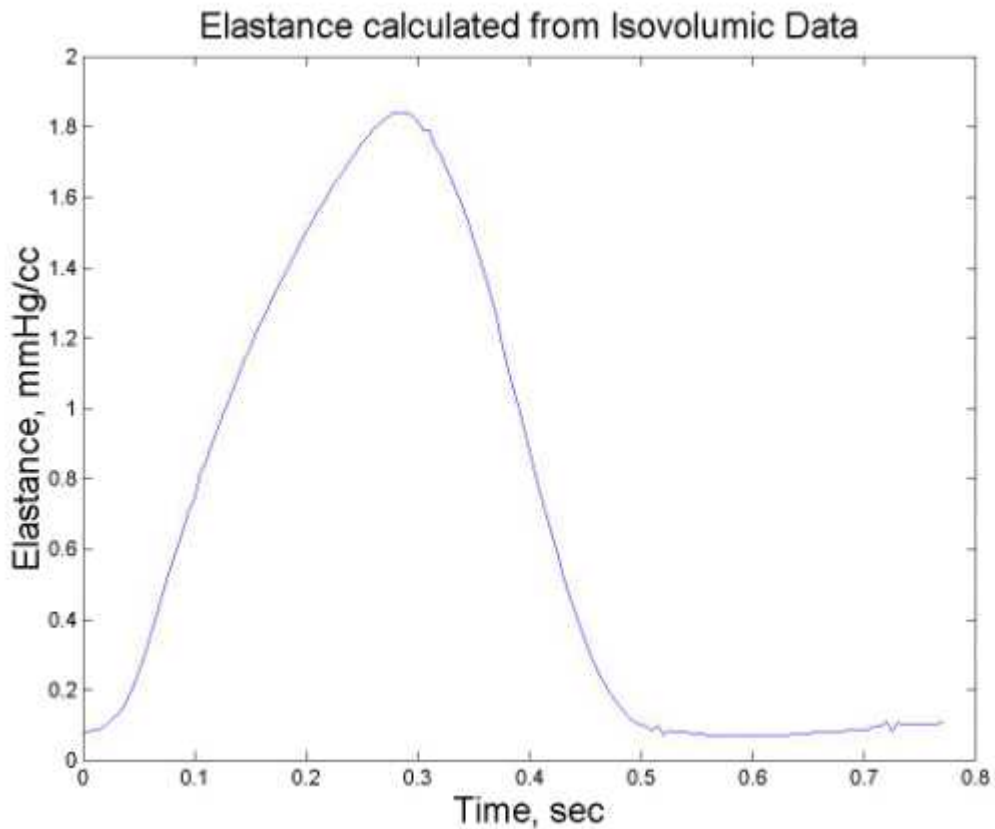


Figure 7-15

What is elastance? Elastance can be mathematically defined, as we have already seen in this chapter and we learned that it describes the relationship between volumes and pressures in the heart. Conceptually, however, elastance is more complex. It is an entity that represents a combination of temporal and spatial averages of sarcomeric forces and their respective lines of action. Each

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sarcomere produces a force in a particular direction – that is, the sarcomeric force is a vector with magnitude and direction.

To explain, as the electrical depolarization signal propagates through the myocardium, a biochemical sequence of events initiate that ultimately results in a multitude of sarcomeric force vectors. One can visualize this process as a balloon whose skin is generating forces in different directions at slightly different times. Using this visualization, one can surmise that the conversion of the instantaneous sarcomeric vectors into left ventricular pressure is going to depend, in part, on the geometry of the balloon. For example, assume that the same force is generated by the sarcomeres in a small diameter balloon and in a large diameter balloon. Does this mean that the internal pressure generated within the “ventricle” will be the same? (need to insert a figure of sphere and stress-strain (timenshenko)). The answer is no and the reason can be seen below. Pressure is defined as force per unit area.

$$P = \frac{F}{A}$$

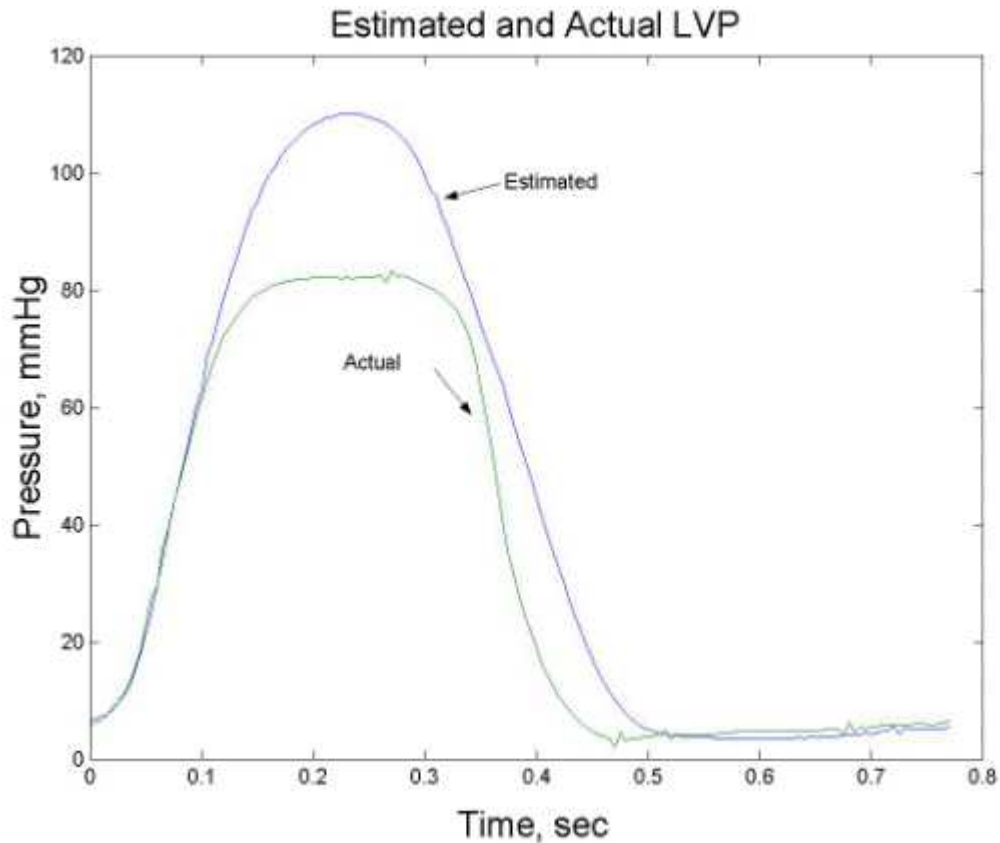
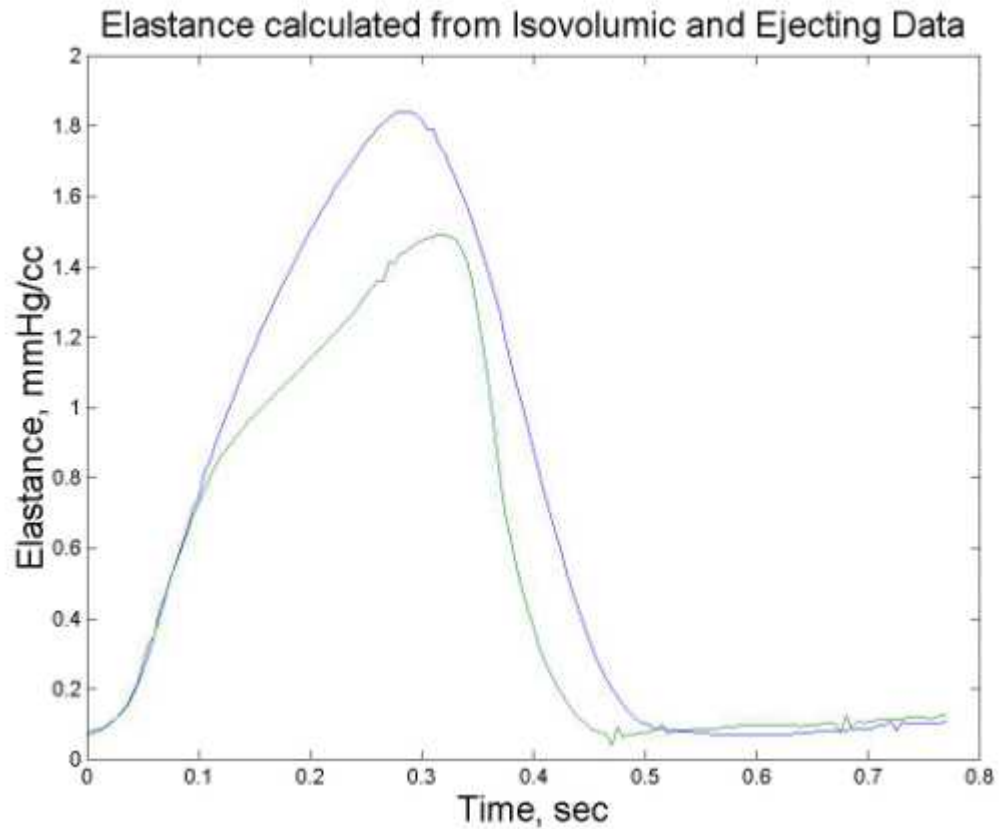


Figure 7-16

Here let A = the inner surface area of a sphere. As this gets larger we need to produce a larger force F , normal to the surface area, A to maintain the same pressure. The force, F , perpendicular to surface A , comes from the sarcomeric forces, F_s , which are tangential to surface A . Everything being equal, the larger the sphere, A becomes larger and the greater the force, F , we must apply to obtain the same pressure. However, everything doesn't remain equal. In fact, the greater the diameter of the sphere, the less sarcomeric force, F_s (tangential to the surface A) is projected perpendicular to the surface area A , resulting in even less pressure generated within the "ventricle" and it is this perpendicular force that generates "ventricular" pressure. **I don't know if this is true.** Furthermore, if we obtain the larger diameter sphere by stretching the balloon from a small diameter, we've created a larger initial tension (tangential forces) that the sarcomeric force F_s must first overcome. Offsetting this over

small ranges of stretch is the fact that sarcomeric force, F_s , increases dramatically with limited amounts of initial stretch.

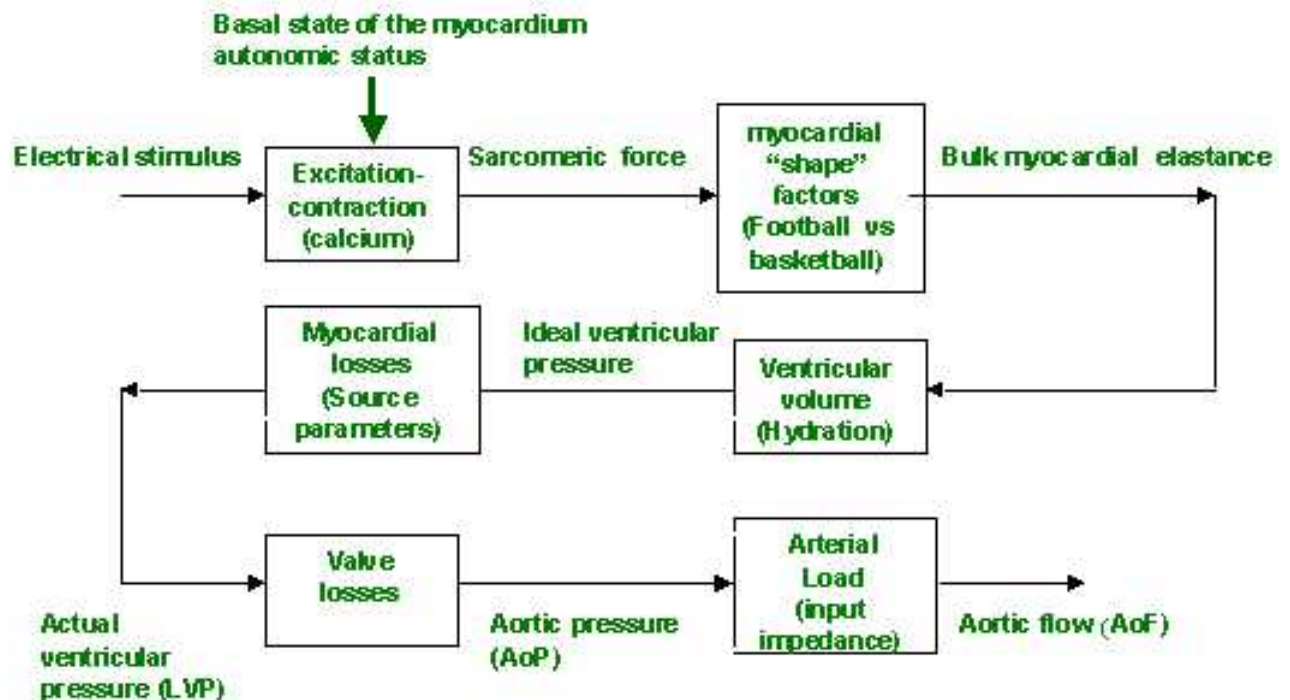


The bottom line here is that the conversion of myocardial sarcomeric force vectors into internal ventricular pressure is dependent on many factors, one of which is geometry, and it is internal ventricular pressure along with ventricular volume that we measure and use to compute a quantity termed elastance. Thus elastance is not just a material property, but depends also on other factors. The significance of this to a cardiovascular engineer is that a low elastance does not necessarily indicate a low sarcomeric force, which might indicate an ailing myocardium. Since elastance is also related to ventricular pressure, low ventricular pressures do not necessarily indicate an ailing myocardium either.

We can now begin to formulate a picture of what really constitutes cardiac function. Can we evaluate cardiac function by looking at ventricular pressures and outflows alone? It appears that we cannot if we expect to gain a complete picture of cardiac function. Figure (XX) show a block diagram of the cardiac

pumping process from beginning to end. The first block illustrates the electrical to sarcomeric force conversion process and is termed excitation-contraction coupling. The next block shows that each sarcomeric force vector sums spatially and temporally through a complex geometry shape to produce a bulk elastance. This bulk elastance operates on a ventricular volume producing an “ideal” ventricular pressure, but as shortening occurs, this ideal pressure is “degraded” to left ventricular pressure by visco-elastic properties of the myocardium. The ventricular pressure is further degraded by viscous losses through the valve and as this pressure interacts with the arterial load, a flow waveform results. Thus only measuring aortic pressure and flow sheds little light on the real culprit(s) of degraded cardiac performance.

Overview of Cardiac Function



Does the elastance waveform’s amplitude and duration vary as a function of volume (sarcomeric stretch)? Initial evidence suggests that it does. If this is,

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in fact, the case, many seeming discrepancies of the pressure volume loops can be explained ... (Add to this later with some data)