Stroke Volume Responses to Exercise

Stroke volume is a hard variable to measure, yet it is profoundly important for deciphering the importance of venous return and myocardial contractility to heart function. For example, a decrease in venous return will lower ventricular filling, end-diastolic volume, and thereby lower stroke volume. For a given end diastolic volume, increasing catecholamine stimulation to the ventricular myocardium will enhance contractile function and thereby increase ejection fraction and stroke volume. More importantly, during incremental exercise, a reduction in stroke volume at near maximal exercise intensities would reveal cardiovascular dysfunction. As you will read, research of stroke volume responses to exercise have revealed different profiles for how stroke volume increases with increases in exercise intensity.

First of all, remember that stroke volume is the volume of blood pumped each heart beat. Figure 1 shows typical stroke volume values for conditions of rest, low intensity, moderate intensity and intense exercise. However, unlike the data available to talk about heart rate responses to exercise, far less data exist to profile the stroke volume response to exercise. This is mainly due to the difficulty in measuring stroke volume, which in reality is not measured, but calculated from measurements of heart rate and cardiac output, as shown in Equation 1. Despite a lack of research, the research that has been done is clear in portraying at least three different stroke volume responses to incremental exercise, and these are presented in Figure 2.

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\text{Cardiac Output (L/min)} = \text{Heart Rate (beats/min)} \times \text{Stroke Volume (L)}
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\[
\text{Stroke Volume (L)} = \frac{\text{Cardiac Output (L/min)}}{\text{Heart Rate (beats/min)}}
\]

Equation 1

Note that some subjects can reveal a classic depiction, where stroke volume increases from rest to moderate intensity exercise, and then levels off quickly despite continued increases in heart rate and exercise intensity. Other subjects, and not necessarily explained by being trained vs. untrained, have a stroke volume response that increases almost all the way to VO_{2max}. Other subjects, and these subjects are healthy and show no abnormal symptoms during intense exercise, show a drop in stroke volume as heart rate increases close to HR_{max}. 

Figure 1. Bar graph of some typical stroke volume values for heart function at different exercise intensities, and for maximal stroke volume for different individuals.
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Figure 2. Stroke volume curves for incremental exercise that represents three different responses identified by research. There is still insufficient evidence to explain the differences in these responses.

**Why/How Does Stroke Volume Increase?**

Although it is intuitive that increasing stroke volume helps to support the increasing oxygen demands of contracting muscle, added thought reveals some problems that confront heart function as exercise intensity increases. First of all, remember that heart rate increases with increasing exercise intensity, and depending on age, HRmax can require up to or in excess of 3 beats each second or 1 beat every 300 ms.

Now let’s think about this with reference to the typical ECG and cardiac cycle. Figure 3 presents the cardiac cycle for measures of ventricular volume, arterial blood pressure, left intra-ventricular blood pressure and the ECG. As you should remember, ventricular filling occurs during a subset of the cardiac cycle called diastole; comprising the time from the opening of the mitral valve (tricuspid valve for the right ventricle), to the closing of these valves. This time period is referred to as the diastolic filling time. Systolic ejection, or systole, occurs between the opening and closing of the aortic valve (pulmonary valve for the right ventricle).

Note that both diastolic filling and systolic ejection times decrease as heart rate increases. Thus, to support sustained heart function as heart rate increases and filling and ejection times decrease the myocardium of the heart must contract more forcefully and more rapidly. Thus, the obvious question I have been building to is how does the heart do this?

The myocardium does not have the neuromuscular structure of skeletal muscle. The myocardium does not have motor units, and all myocardium contracts each heart beat. As such, the myocardium must be forced to contract differently as the circulatory demands on the heart increase, such as during exercise. To begin with, let’s look at the myocardial contraction responses to changes in end diastolic volume (EDV), as shown in Figure 4. This response is known as Starling’s Law, where an increase in EDV causes increased myocardial stretch, which in turn induces a more forceful/powerful myocardial contraction. The result is a higher stroke volume. This means that as more blood is returned to the heart, more blood can be pumped by the heart without any additional regulation.
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Starling’s Law certainly improves heart function, but it is dependent on an increasing EDV. Remember that exercise demands that the heart do more work (pump more blood) in less time (higher heart rates). Thankfully, the total work output of the heart can also be improved through catecholamine hormone and neurotransmitter stimulation, which both increase during exercise. Catecholamine stimulation of the myocardium further increases the force/power of myocardial contraction, as also presented in Figure 4. This improved myocardial contraction response, for a given EDV, is referred to as increased contractility. Thanks to the combination of Starling’s Law and increased contractility, the myocardium can now function as a more effective pump at higher heart rates, better supporting the blood flow and oxygen delivery demands of exercise.

Figure 3. Changes during the cardiac cycle for the ECG, valve sounds, intraventricular volume, left atrial pressure, intraventricular pressure and systemic arterial blood pressure.
Glossary Words

stroke volume is the volume of blood pumped from the heart (typically referenced to the left ventricle) each beat.

cardiac cycle is a summary of the events that occur during one complete cycle of heart function, typically referenced to be from the start of one p-wave to the next.

ventricular volume is the volume of blood in the ventricle (typically referenced to the left ventricle) at any point in time.

arterial blood pressure is the blood pressure of the arterial blood vessels. However, this is not a constant for the arterial system, as arterial blood pressure varies in accord with the distance the artery location is from the heart, as well as for the phase of the cardiac cycle.

ventricular filling refers to blood flow into the ventricles.

diastole is the time period incorporating passive filling of the atria and ventricles.
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**diastolic filling time** is the time period of diastole.

**systolic ejection** is the ejection of blood from the ventricles.

**systole** is the period of myocardial contraction, where there is an atrial systole and ventricular systole.

**end diastolic volume (EDV)** is the volume of blood in the ventricles (typically referenced to the left ventricle) immediately prior to ventricular systole.

**Starling’s Law** refers to the increase in ventricular contractile function as end diastolic volume increases.

**contractility** is the increase in ventricular contractile function for a given end diastolic volume, which is due to catecholamine stimulation of the myocardium.