TRI TRI AGAIN: THE PHYSIOLOGY OF TRIATHLON

By

MARISSA PEARLE WHEELER

_____

A Thesis Submitted to The Honors College

In Partial Fulfillment of the Bachelors degree
With Honors in

Physiology

THE UNIVERSITY OF ARIZONA

M A Y 2 0 1 7

Approved by:

_____

Dr. Zoe Cohen

Department of Physiology

Abstract

The cardiovascular system plays an essential role in any endurance event. The Ironman Triathlon is a long distance event that is made up of a 2.4 mile swim, a 112 mile bike, and a 26.2 mile run for a total of 140.6 miles. Each segment of an Ironman Triathlon poses unique challenges to the cardiovascular system. In order to complete such a monstrous race cardiovascular adaptations occur during the chronic stress of training, and the body also makes acute adaptations during the metabolic stress of the race itself. These alterations of the heart, blood vessels, and the blood make the body more efficient at transporting oxygen and nutrients to the muscles via the circulatory system, at extracting oxygen and nutrients from red blood cells and into the tissues, and at maintaining homeostasis. The results of these changes are higher maximum heart rate, increased oxygen extraction, higher capillary bed density, higher mitochondrial density, increased VO_2 max, increased cardiac output, better thermoregulation, and non-pathological hypertrophy of the heart. While an excessive amount of long-term vigorous endurance activity can have adverse impacts on the cardiovascular system, the body is surprisingly capable of racing such a long distance with the proper training adaptations.

Dedication

It is with my deepest gratitude that I humbly dedicate this thesis to my triathlon coach and two-time Olympic alternate Doug Friman who encourages me to push beyond the limits that I set for myself. His wise words have been a constant source of inspiration and knowledge. Over the past two years he has shown me that "it is okay to fail, but it is never okay to not try."

"Life is a storm, my young friend. You will bask in the sunlight one moment, be shattered on the rocks the next. What makes you …is what you do when that storm comes. You must look into that storm and shout as you did in Rome. Do your worst, for I will do mine! Then the fates will know you as we know you."

— Alexandre Dumas, The Count of Monte Cristo

Table of Contents

Title **Page**

Tri Tri Again: The Physiology of Triathlon

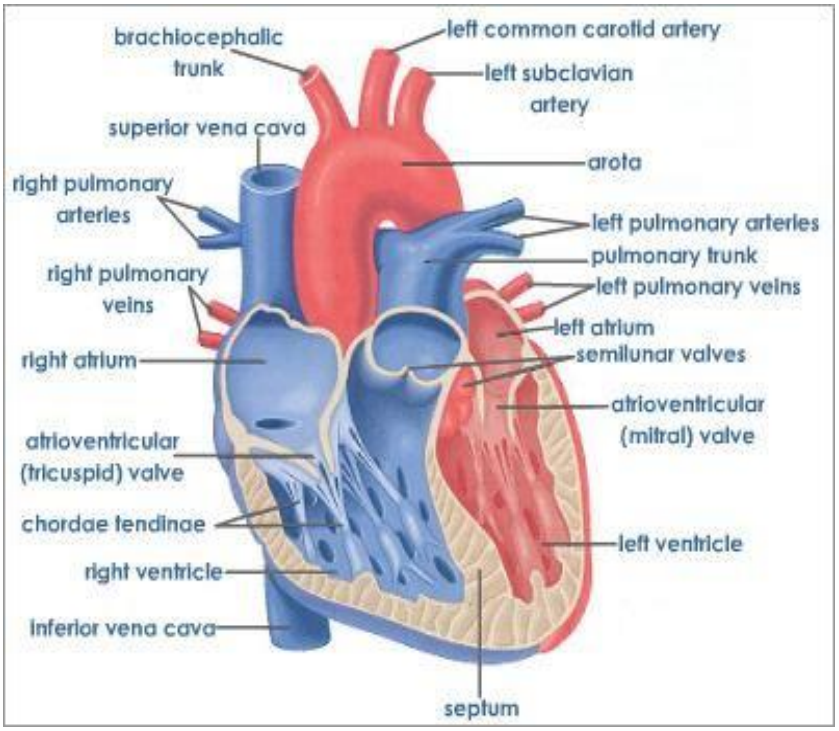
I. Introduction to the Cardiovascular System (Heart, Vessels, Blood)

The cardiovascular system is comprised of three main parts: the heart, blood vessels, and blood. Since form almost always follows function it is important to first understand the basic anatomy of the cardiovascular system.

I.a. ***The Heart***

The heart is a hollow muscular organ the size of a fist. It is located close to the midline of the body within the thoracic cavity and between the rigid bones of the sternum and vertebrae, which provide protection to the vital organ (Tortora 689-690). The heart is essential to the body because it provides pressure throughout the circulatory system by generating a high hydrostatic pressure that drives blood throughout. This is accomplished through a dual pumping action. The heart can be divided laterally by an inner partition of muscle called the septum with deoxygenated blood entering the right side of the heart and oxygenated blood leaving the left side. There are four chambers that comprise the heart which include the superior right and left atria which are low pressure chambers and receive blood, as well as the inferior left and right ventricles which are responsible for producing

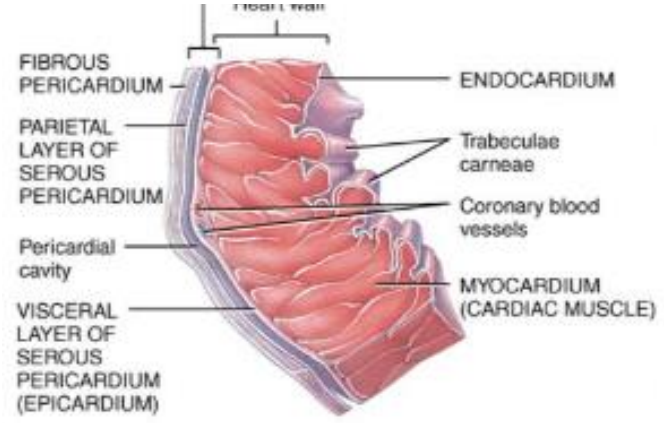
Figure 1: Normal Heart Anatomy



higher pressures and discharging blood. Blood flows in a unidirectional path first entering the right atrium then the right ventricle before being sent through the pulmonary trunk to the lungs to be oxygenated. This blood then returns via the pulmonary veins to the left atrium and finally enters the left ventricle before being pumped to the periphery via the aorta. To ensure that blood only flows in one direction there are four one-way valves: two atrioventricular (AV) valves that are between each atria and ventricle and two semilunar valves that separate the right and left ventricles from the pulmonary and aortic arteries respectively as depicted in *figure 1 (*Tortora 696-697). These valves are essential as they prevent blood from moving backwards and causing turbulence which can have negative impacts such as blood clots. Blood will always move from areas of high pressure to areas of low pressure and by contracting the various chambers, the heart can cause this flow. For instance, to propel blood out of the left ventricle the walls contract which generates pressure. With the left atrioventricular valve preventing blood from flowing back into the low pressure left atrium it is forced through the aortic semilunar valve.

The heart is a muscle that has three layers: the pericardium, myocardium, and endocardium. The pericardium is a double walled sac that encloses the heart and helps to anchor it to surrounding structures, the myocardium is the thickest muscular layer that is responsible for contraction, and finally the endocardium is the innermost layer made up of endothelium that lines the entire cardiovascular system and minimizes friction as blood passes through (Tortora 691). Since the atria are receiving

Figure 2: Layers of the Heart Wall



chambers and only have to send blood to the ventricles they are fairly thin walled in comparison to the thick muscular ventricles which must contract forcefully in order to expel blood. Furthermore, the cardiac muscle tissue is striated with up to 25% of its volume being comprised of mitochondria compared to only 2% in the skin (Keen Lecture 28: pg 233). Mitochondria are considered the power house of cells and provide energy through oxidative phosphorylation therefore, a higher density of mitochondria indicates that the heart is a highly metabolic muscle (Keen Lecture 7: pg 51).

The cardiac muscle fibers that make up the myocardium contain two distinct types of cells: cardiac myocytes and autorythmic cells. Cardiac myocytes make up 99% of the cells and are responsible for the mechanical pumping action of the heart but will only contract when stimulated. The other 1% of cells, the autorythmic cells, generate pacemaker potentials that spread through intercalated discs causing the heart to pump in a synchronized fashion (Keen Lecture 26: pg 233-234). Cardiac myocytes are laid out end to end and connected via a desmosome. A contractile bundle of myocytes is called a myofibril which is divided into compartments called sarcomeres (Tortora 303). A sarcomere is the basic contractile unit of muscle

Figure 3: Muscle Fiber Anatomy

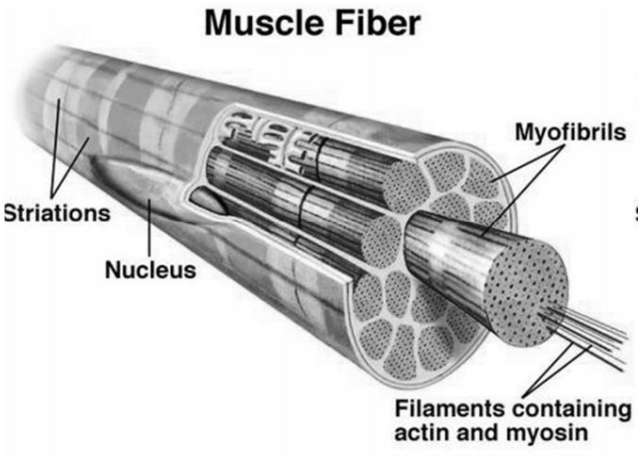
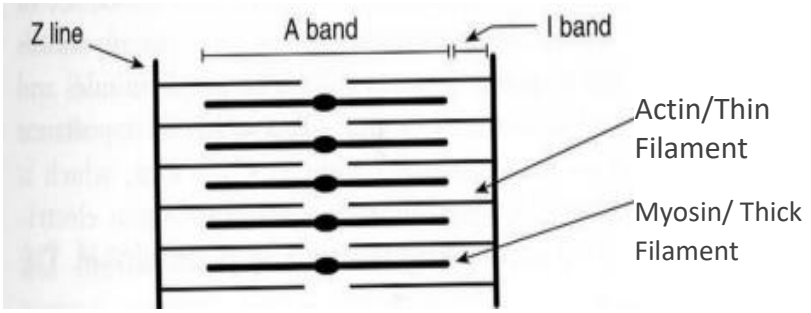


Figure 4: Sarcomere



tissue and is made of thick and think filaments that can be overlapped more or less to cause contraction. Thick filaments are made up of the contractile protein myosin and thin filaments are made up of actin (see figure 4). These filaments convert chemical energy (ATP) into mechanical energy when the myosin heads attach to a myosin binding site on the actin filament, which is called a cross-bridge, and completes a power stroke that slides the thin filament past the thick filament toward the middle of the sarcomere (Tortora 303). When this occurs the sarcomere is shortened and the heart muscle is in a contracted state. The number of cross-bridges made directly correlates to the amount of force generated. The cross-bridge cycle is regulated with troponin and tropomyosin molecules which block the myosin binding sites on actin. During contraction an action potential will travel along the sarcolemma causing it to depolarize and activate voltage-gated Ca^{2+} channels on the T-tubules. Subsequently the influx of Ca^{2+} causes the sarcoplasm reticulum to release even more Ca^{2+} into the sarcoplasm that surrounds the sarcomeres (Cohen: Structural 19-23). This process is called Calcium-Induced Calcium Release.

The Ca^{2+} then binds to troponin which will then undergo a conformational change causing tropomyosin to unblock the binding site. Therefore, calcium is a regulator of contraction as when the heart is constricting the level of calcium within the sarcoplasm is high and when the heart is in a state of relaxation the levels are low (Tortora 304).

When the heart is contracting, it is generating pressure, and this period of time is referred to as systole, whereas times of heart relaxation and filling are referred to as diastole. Cardiac output (CO) is the amount of blood pumped out of the heart in one minute and can be broken down into heart rate multiplied by the stroke volume (SV). Whereas CO is the amount of blood

pumped in one minute, the SV is the volume of blood ejected from the left ventricle per heartbeat.

SV can be calculated in terms of the amount of blood within the ventricle at the end of filling minus the amount of blood remaining in the ventricle after contraction, or end diastolic volume (EDV) minus end systolic volume (ESV). The difference between EDV and ESV is the amount ejected during contraction (Keen Lecture 29: page 237).

There are three main determinants of stroke volume: preload, afterload, and heart contractility. Preload, is the pressure produced in the left ventricle due to the amount of blood loaded into the heart prior to contraction, which is itself dependent on venous return or the rate of blood returned to the heart. This is called the "end diastolic pressure" since it is the pressure at the end of filling (Cohen: Myocardial Contractility 3-5).

Afterload is the resistance that the left ventricle must overcome in order to eject blood. This is related to the pressure in the aorta but is often times thought of as the ventricular wall stress. In this way, afterload can also be seen as the stress put on the myocardial cells to overcome the aortic pressure and eject blood (Klabunde 1). One way to visualize afterload is to think of how difficult it would be to open a car door underwater. The water would be exerting a pressure on the door that would need to be overcome in order for it to open. This is analogous to the heart having to contract with enough force that it overcomes the pressure from the aorta. The resistance stems from the blood in the vessels as well as the constriction of the vessel walls. A higher afterload means that the heart must work harder to eject blood and as a result more will remain in the ventricle after systole.

Finally, contractility is the strength of heart contraction or forcefulness of the myocardial contraction. Higher contractility results in less blood within the left ventricle following systole or

a decreased ESV. Assuming that heart rate remains constant, more contractility leads to a larger stroke volume and increased cardiac output (Keen Lecture 29).

Similar to stroke volume, there are also many factors that affect venous return. Some of these muscular pump, venous tone, and heart rate (Keen Lecture 29: pg 238). As we will see later changing any one of these factors will alter the amount of blood that is loaded into the heart prior to contraction.

Since CO is equivalent to how much blood is ejected from the LV multiplied by the number of beats per minute, heart rate plays a crucial role in cardiac performance. If SV remains constant, but the HR is decreased less blood will circulate and vice versa. Therefore, heart rate also provides a short-term mechanism for rapidly altering cardiac output. The primary regulators of heart rate are the autonomic nervous system and the catecholamines released by the adrenal medullae such as epinephrine and norepinephrine (Tortora 714). The cardiovascular center is located in the medulla oblongata which uses nerve impulse frequency to the parasympathetic and sympathetic segments of the ANS to adjust heart rate. If the sympathetic branch is stimulated norepinephrine will be released and bind to beta-adrenergic receptors of cardiac muscle fibers. This leads to an increased rate of spontaneous depolarization within the SA node and increases heart rate. On the other hand, with parasympathetic stimulation via the vagus nerves acetylcholine is released which in turn decreases the rate of spontaneous depolarization and slows heart rate (Tortora 714).

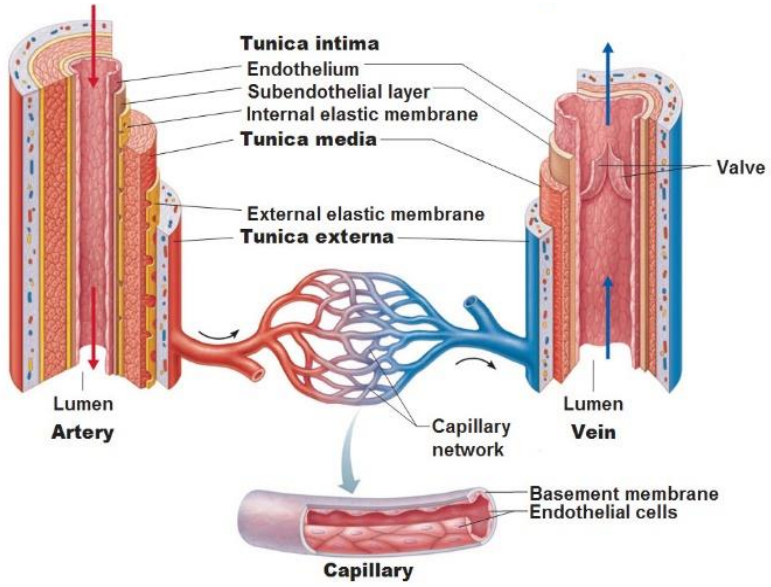
The cardiovascular center receives input from proprioceptors, chemoreceptors, baroreceptors, and the limbic system. Proprioceptors monitor body position and muscles. Similarly, chemoreceptors monitor the chemical levels of blood and responds accordingly. One example is if blood carbon dioxide levels are high. This would indicate that not enough oxygen

and carbon dioxide exchange is occurring and that cardiac output should increase which can be done rapidly by manipulating heart rate. Baroreceptors detect stretching of major blood vessels due to blood pressure. Since the aorta and carotid arteries are so essential, they have many baroreceptors to regulate the blood pressure flowing through them (Cohen: Blood Pressure 23-28). Finally, the limbic system, which plays a key role in emotions, also contributes to cardiovascular center feedback. This is why a person's heart rate spikes when they are upset or due to the anticipation of a race start (Tortora 714).

I.b. Blood Vessels

Although the heart is often the focus of the cardiovascular system, without blood vessels there would be no way to deliver oxygen and nutrients to the entire body. The body has multiple types of blood vessels which include arteries, arterioles, capillaries, venules, and veins.

Figure 5: Blood Vessel Structure



While the composition of all blood vessels follows a basic structure they do vary in accordance to their primary job. For the majority of blood vessels there are three layers which are referred to as tunics. The innermost layer is the tunica interna consisting of an endothelial lining, a basement membrane, and internal elastic lamina. The endothelial lining is in direct contact with blood traveling throughout and reduces friction. The basement layer is the second component of the tunica interna and is made of collagen fibers that create tensile strength

to keep vessels durable under stretching and elastic recoil. Lastly, the internal elastic lamina contains large holes that allow for diffusion (Tortora 730). Next, the middle blood vessel layer is the tunica media which varies greatly throughout various vessels. The tunica media is mostly smooth muscle cells and elastic fibers arranged in a circular fashion which allows for a large amount of control over the diameter of the vessel as the muscular layer contracts and relaxes (Tortora 730-731). Finally, the tunica externa covers the outside of blood vessels and helps to anchor them to surrounding tissues.

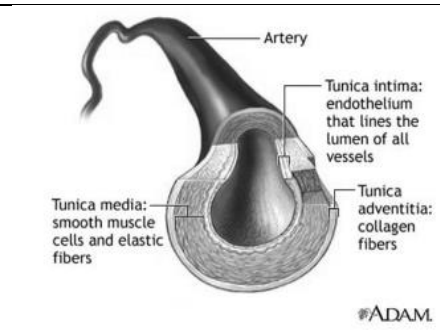
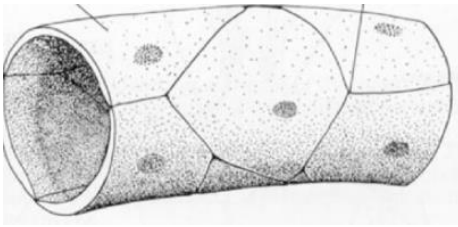
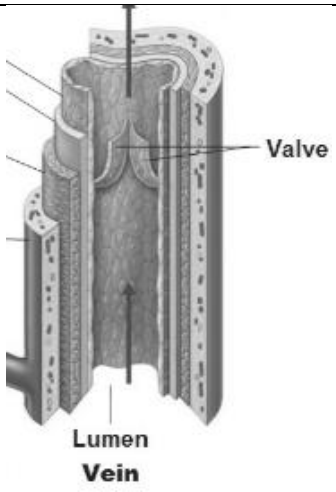
Arteries rapidly carry blood away from the heart and contain all three of the layers discussed, with a very thick tunica media. The largest arteries are those closest to the heart, such as the aorta and pulmonary trunk. Arteries are sometimes called elastic because they have a lot of elastic fibers which enables them to stretch as blood is forcefully pumped from the heart. This results in the storage of mechanical energy and in this capacity arteries are pressure reservoirs (Cohen: Arteries and Arterioles 15). When the arteries recoil this mechanical energy can be converted back into kinetic energy that propels blood onward. The further away the arteries get from the heart, the smaller their diameter becomes as they must deal with lower blood pressure. Arterioles are microscopic arteries that lead into capillaries. Prior to each capillary, the arteriole contains a precapillary sphincter that regulates blood flow into the capillaries (Tortora 733). Arterioles are structurally designed to be resistance vessels and have a very thick layer of smooth muscle which allows them to vary their diameter. The smooth muscle is very sensitive to local hormones. High oxygen levels, sympathetic stimulation, myogenic activity, endothelin, and cold are all associated with vasoconstriction. On the other hand, high carbon dioxide levels, nitric oxide, heat, and histamine are all associated with vasodilation. Since arterioles can vary their

radii independent of other arterioles they can also help control how cardiac output is distributed throughout the body. (Cohen: Arteries and Arterioles 22-24).

The smallest type of blood vessels are capillaries, despite their size (3.5 nm diameter) capillaries have the largest cross-sectional area of all blood vessels. This stems from the incredibly large number of them and makes capillaries the site for nutrient exchange (Keen Lecture 25: pg 235). Capillaries are only wide enough for one red blood cell to squeeze through at a time, slowing blood and facilitating diffusion. Tissues that are highly aerobic like skeletal muscles and the brain will have a more extensive capillary network than those lower in metabolism such as tendons. Still, when total body metabolic needs are low blood will only flow through a small portion of the capillary network. When the demands are higher though, the precapillary sphincters will relax and allow blood to flow throughout the entire capillary bed of that specific organ (Tortora 734).

Blood returns to the heart first through venules and then into larger veins which are the volume reservoirs of the body. Unlike arteries, veins have a large radii which lessens resistance of blood flow. They also have thin walls and very little elasticity since they are low pressure vessels (Cohen: Capillaries and Veins 16). Seeing as the blood pressure is so low and gravity acts against the flow of blood back to the heart, it would be possible for blood to flow backward. To prevent this, veins contain one-way valves that only allow blood to move forward; if blood tries to move backwards it fills the leaflets of the valve and forces it closed. Another reason that blood continues to flow toward the heart is that fluids flow from high to low pressure. Despite a pressure of around 17mmHg, blood still flows toward the atria since it has an even lower pressure of 0mmHg.

Figure 6: Blood Vessel Characteristics

Vessel Type	**Structure**	**Function**	**Diagram**
Arteries	Thick elastic walls Large amounts of smooth muscle Thick tunica media	Pressure reservoir Rapid transport of blood	
Arterioles	Thick smooth muscle Contain precapillary sphincter Little elastic CT	Regulate blood flow into capillaries Resistance vessels Distribute CO	
Capillaries	Largest amount and cross-sectional area Smallest vessel No tunica media/externa Single endothelium layer	Site of exchange between blood and tissue Allows one RBC at a time to increase diffusion	
Veins	Large internal radius No internal elastic lamina Thin tunica media Contains valves Little elasticity	Return blood to heart Prevent backflow Volume reservoir Low resistance	

There are two factors that greatly impact blood flow: pressure gradients and resistance. As the pressure differences increase, blood flow also increases as it moves down its gradient from high to low but as the resistance increases blood flow is hindered and decreases (Cohen: Arteries and arterioles 5).

The blood pressure is determined primarily by cardiac output. Resistance can be impacted by the viscosity of blood since thick sludgy blood will not flow as smoothly due to the friction. It is also affected by vessel radius as was discussed earlier. If a radius is very small, less blood will be able to flow through in a given time period. Even the smallest changes in radius can majorly impact blood flow because resistance is inversely proportional to $1/\text{radius}^4$. Finally, the length of the vessel is related to resistance with longer vessels have increased resistance(Cohen: Arteries and Arterioles 1-11).

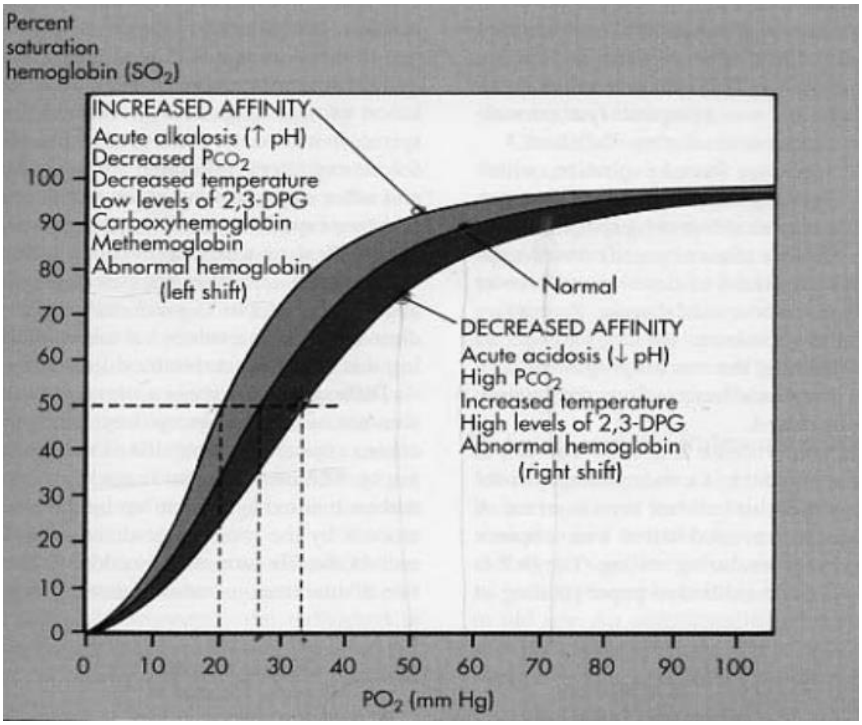
I.c. Blood

The purpose of the heart and blood vessels is to transport nutrients to the tissues and to remove waste. This is accomplished via a liquid medium which we know as blood. The functions of blood include transportation, regulation, and protection. Blood serves as a vehicle to transport oxygen from the lungs to the cells and carbon dioxide from the cells to the lungs to be removed during exhalation. Blood also helps to regulate the pH levels within the body in order to create a stable environment, termed homeostasis. This is accomplished via buffers that convert strong acids and bases into weak non-threatening ones Another way that blood helps maintain homeostasis is through temperature regulation since blood can flow through the skin to disperse excess heat. The protective function of blood comes from its ability to form clots during an injury to prevent excessive blood loss (Tortora 662). Another important protective quality comes from the white blood cells which are involved in the immune system.

Blood makes up around 8% of total body weight so males generally have 5-6 L and females generally have 4-5 L of blood. The non-cellular component of blood contains plasma and plasma proteins which comprises 55% of blood. The primary purpose of plasma is to move organic and inorganic substances as well as to redistribute heat. Plasma is 90% water but also contains proteins and some solutes. The plasma proteins are primarily produced by the liver. These proteins include albumins which maintain osmotic pressure to facilitate the exchange of fluids in capillaries, globulins which play an immunologic role by attacking viruses and bacteria, and finally fibrinogen which is vital to blood clotting (Tortora 664). The other 45% of blood is formed elements. There are three cellular components of blood that make up the formed elements: red blood cells (RBC's), white blood cells (WBC's), and platelets. RBC's transport oxygen and carbon dioxide, WBC's are involved in immunity and debris removal, and platelets coagulate to form clots and control excessive bleeding (Cohen: Blood 4).

Despite being fairly simple in structure, the RBC is highly specialized. RBC's are designed to have a large surface area by being a biconcave disc shape. Since capillaries are extremely small in diameter it is crucial that RBC's can alter their shape to squeeze through which is why they are very flexible and thin.

Figure 7: Oxygen Dissociation Curve

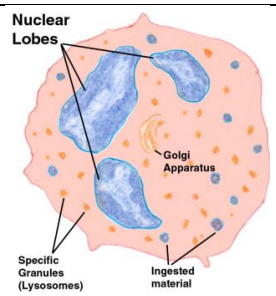
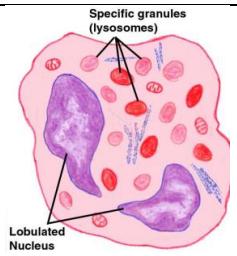
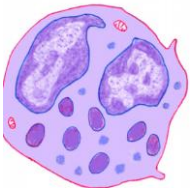
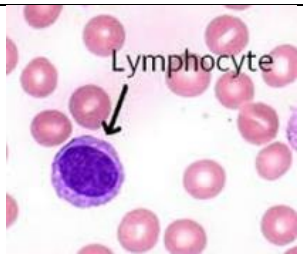
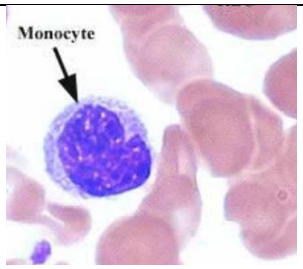


They contain no nucleus since all of their space is utilized for transport of oxygen and/or carbon dioxide. Lacking a nucleus results in a short life span of around 120 days (Cohen: Blood 18). Within the RBC are hemoglobin molecules made up of a globin that has four polypeptide chains and a heme protein with an iron ion in the middle of each heme ring. The iron allows each heme to reversibly bind one oxygen molecule allowing for a hemoglobin to bind a total of four oxygen molecules at a time. Since each RBC contains around 250 million hemoglobin molecules, each red cell can hold approximately 1 billion O_2 molecules (Tortora 668).

The oxygen dissociation curve shown in *figure 7* demonstrates that as more molecules of oxygen bind to hemoglobin the affinity for successive oxygen molecules increases. As the maximum binding limit is approached the affinity for binding decreases. The affinity at which oxygen binds to hemoglobin can also be impacted by the temperature, partial pressure of CO_2, the pH of blood, and the partial pressure of O_2 (*see figure 7)*.

Less than 1% of blood is made up of platelets and WBC's. Platelets originate from the megakaryocytes in bone marrow and are involved in adhesion, aggregation, secretion, and coagulation which are essential to the process of blood clotting. Similar to red blood cells platelets have no nucleus and therefore have a short lifespan of 8-10 days (Cohen: Blood 39). WBC's can be either granular or agranular which is a term based on how they appear after staining the cells. Granular cells include neutrophils, eosinophils, and basophils while agranular includes lymphocytes and monocytes (*see figure 8)* (Cohen: Blood 8).

Figure 8:WBC Types

WBC	**Percentage**	**Function**	**Diagram**
Neutrophil	60-70%	Phagocytosis First defenders	
Eosinophil	2-4%	Allergic conditions Destroy parasites	
Basophil	0.5-1%	Releases histamine	
Lymphocytes	20-25%	T cells destroy target cells B cells create antibodies	
Monocytes	3-8%	Become phagocytes when enter tissue	

Overall the cardiovascular system can be viewed as three basic components, a pump, some tubes, and a liquid to flow through the tubes. Although the reality is more complex than this, at its core the cardiovascular system is beautifully simple and we could not survive without it.

II. Introduction to the Ironman Triathlon "Whoever finishes first, we'll call him the Ironman"

II.a. History

It is human nature to push ourselves to physical extremes in order to see what our bodies are capable of- which turns out to be a whole lot. The body is able to adapt to incredible stress by directly altering its physiology. These changes may be small in low intensity activity, but become abundantly clear when looking at the Ironman Triathlete.

Today, the Ironman Triathlon is comprised of a 2.4 mile swim, a 112 mile bike, and a 26.2 mile run for a grand total of 140.6 miles. The average finishing time is 12 hours and 35 minutes with times ranging from 9 hours and 20 minutes to almost 17 hours, which is the time cutoff (Britt). The Ironman did not develop overnight however, and prior to these seemingly impossible distances came the original triathlon. In 1974, the idea of a swim, bike, run race-or triathlon- was used by the San Diego Track Club as fun cross training to break up monotonous marathon and 10k training regimens. One athlete who competed in these first triathlons was John Collins. In 1977, Collins used these fun races as the basis for combining three of Hawaii's endurance events: the Waikiki Rough Water Swim (2.4 miles), the Around- Oahu Bike race (112 miles), and the Honolulu Marathon (26.2 miles). This challenge, to see who the toughest athletes were: swimmers, cyclists, or runners, would be named the Ironman Triathlon (History 104).

Figure 9: Julie Moss's Iconic Crawl

It wasn't until the late 1980's that triathlon really started to grow as a sport. The global public learned about Ironman races in 1982, when ABC was permitted to film the Ironman World Championships. During this race, collegiate athlete Julie Moss was shown collapsing yards from the finish line. Although this cost her a first place finish, the footage of Moss crawling to the finish line for second place captivated audiences across the world. In 1989 twenty-five nations came together to form the International Triathlon Union (ITU) with the ultimate goal of having the sport of triathlon accepted into the Olympics. To do this, the ITU created the first triathlon world championships with the more manageable distances of a 1.5k swim, 40k bike, and 10k run. Finally, triathlon was accepted as an event in the 2000 Olympic Games (History 105). Today the sport is rapidly expanding and in 2010 an estimated 2.3 million individuals competed in various triathlon race distances in the United States, where the sport's governing body is USA Triathlon (History 109).

Figure 10: Various Triathlon Distances

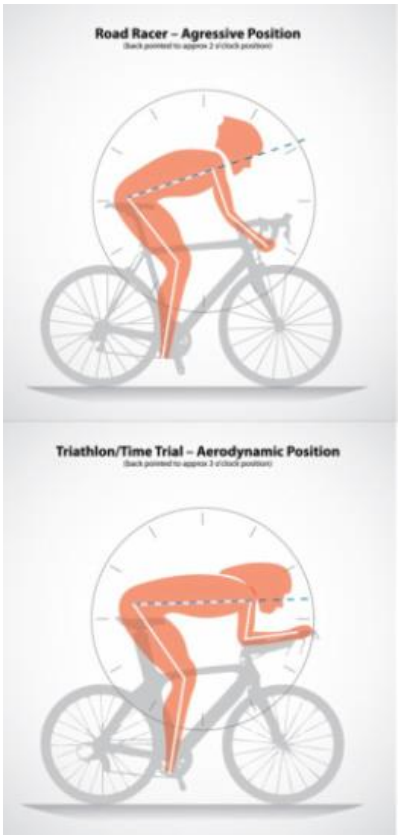
	SPRINT	OLYMPIC	HALF IRONMAN	FULL IRONMAN
Swim	½ mile	1.5K *(just short of 1 mile)*	1.2 miles	2.4 miles
Bike	12–14 miles	40K *(roughly 25 miles)*	56 miles	112 mile
Run	5K *(3.1 miles)*	10K *(6.2 miles)*	13.1 miles	26.2 miles

II.b. Overview of the Challenges within each Component: Swim, Bike, Run

Although triathlon is one sport, it can be broken down into three components: swim, bike, and run. Each discipline uses different muscles and presents its own unique set of challenges for the athlete, and the athlete's body.

During the swim portion of an Ironman, the body is in a prone position lying horizontal rather than vertical. This means that the heart does not have to fight gravity while pumping blood and can have cardiovascular implications. Every Ironman race is done in open water which can pose many challenges such as water turbulence and cold temperatures. When the water temperature is at or below 76.1 degrees Fahrenheit wetsuit use is legal. Aside from maintaining body heat, a wetsuit makes the athlete more buoyant in the water. However, wetsuits by nature fit very tight to the body and can have a compression effect that can restrict the chest cavity from expanding fully during a deep breath. Swimming can also cause oxygen deprivation to the body. This is especially true during the swim start which is often crowded and chaotic with people swimming on top of other competitors and creating lots of wake making it hard to get a breath. Despite being the first portion of an Ironman, the swim poses a multitude of challenges that the body must adapt to and overcome. The second discipline of a triathlon is cycling. After coming out of the water athletes are often dizzy and disoriented which can make starting the bike course a hurdle. Once the water from the swim dries, body temperature begins to rise creating another problem for the body to deal with. If the race weather

Figure 11: Bike Positioning



isn't too warm however, the wind from moving forward rapidly can help the body's evaporative cooling system. Another important factor on the bike is the aerodynamic position that most athletes assume to be more efficient. A triathlon style bike has aero bars that come off the front of the bike and put the athlete in a bent over position with their upper body nearly prone (*see figure 11*). Unlike swimming, cycling uses a few specific and large muscles (Quadriceps, Hamstrings, Glutes, and Calves) rather than the whole body. These muscles will also be used for the longest time since cycling accounts for approximately 50% of total race time (Britt). During the six or more hours spent on the bike nutrition and hydration are some of the biggest obstacles that an athlete faces.

After finishing the swim and bike the body must still endure a marathon distance run. Although the run may seem the simplest of the three disciplines since there is no danger of drowning or crashing, the body is extremely fatigued by the time the run begins. Just as on the bike, core temperatures remain high and avoiding hyperthermia during a warm race is vital. During the run, the body is moving slower through the air and it therefore takes longer for sweat to evaporate from the body. Unlike the swim and bike portions, the body positioning is now fully upright and the heart has to work against gravity to pump blood throughout the cardiovascular system. An additional challenge on the run is avoiding dehydration and hyponatremia from improper hydration and nutrition, since on a warm day an athlete can sweat more than one liter of fluid per hour of biking and running (Triathlete).

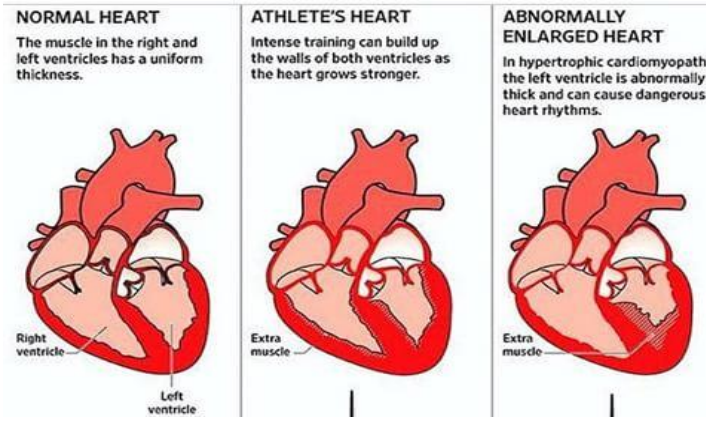
Each segment of an Ironman triathlon poses its own unique obstacles to the body, fortunately humans are able to adapt to extreme situations and with proper training the body- in particular the cardiovascular system- can rise to the task.

III. Physiological Changes That Occur During Ironman Training and Racing

III.a. The Heart

Many changes occur to the heart during chronic exercise that allow it to meet the demands of the body in an efficient manner. At the most basic level, the structure of the heart changes. The heart is a muscular pump, and like any other muscle when it is used repeatedly its structure changes.

Figure 12: Pathologic vs. Normal Cardiac Hypertrophy



When a muscle is used as in exercise, cellular damage occurs and is then repaired. If this process is done repeatedly the myofibrils will increase in thickness and amount and thus the muscle grows. The heart is also a muscle, and when frequently stressed it too will hypertrophy. The left ventricular wall of the heart is naturally the thickest since it must contract forcefully to overcome aortic pressure when expelling blood to the periphery. Therefore, with exercise induced cardiac hypertrophy the left ventricular wall increases in thickness. However, unlike pathologic cardiac hypertrophy the chamber size does not decrease and cardiac remodeling due to endurance training is a compensatory mechanism to deal with increased demand (*see figure 12).* The added muscle allows the heart to contract more forcefully than one with thinner walls and this increases cardiac performance since contractility is a factor that impacts SV.

During an endurance event such as an Ironman Triathlon, the muscles begin contracting more frequently and/or with greater force than during rest causing their demand for oxygen and

nutrients to increase. The heart counteracts this by increasing cardiac performance. At rest blood flow to muscles is approximately 1,200 ml/min and blood flow to the heart itself via pulmonary circulation is 250 ml/min (Keen 252). This demonstrates that the heart is not working very hard since the blood it requires to maintain its own oxygen demands is low. During exercise there is a redistribution of blood and blood that normally flows to less vital organ systems, such as the digestive system, is reduced. For example, blood to the muscular system will increase 20 fold to about 22,000 ml/min. The heart's blood supply will also increase and receive around 1,000 ml/min due to the fact that the muscle is working harder to pump a greater volume of blood, which then increases its own need for oxygen. On the other hand, the kidney's blood flow decreases from 1,100 ml/min to 250 ml/min (Keen 252). All in all, the total blood flow increases from 5 L/min to 25 L/min which can be seen as a dramatic increase in cardiac output by the heart (see page 4 for CO).

It can be expected that the disciplines within the Ironman that require activation of more or larger muscles will cause a greater redistribution of blood. For instance, during the swim many triathletes "save their legs" by relying more on the upper body for propulsion and keeping kicks to a minimum. The upper body has a smaller muscle mass than the lower body therefore less blood would need to be redistributed during the swim compared to the bike or run portions of an Ironman.

There are many factors that impact cardiac performance which can be evaluated in a variety of ways. As discussed in section 1a. CO is equivalent to how many times the heart beats per minute times how much blood each beat can pump, or HR times SV (*See equation 1).* The average resting male will have an approximate heart rate of 75 beats/min and a stroke volume of

Equation 1: Cardiac Output

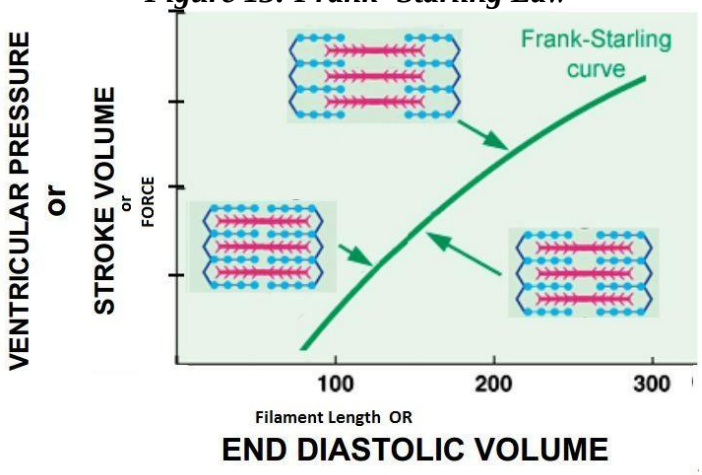
$$CO\ \left(\frac{ml}{\text{min}}\right) = HR\ \left(\frac{\cancel{beat}}{\text{min}}\right) x\ SV\ \left(\frac{ml}{\cancel{beat}}\right)$$

Equation 2: Stroke Volume
$SV = EDV - ESV$

70ml/beat, therefore the CO is around 5250ml/ min or about 5 L/min (Tortora 713). To increase CO from 5L/min to around 25L/min, as is necessary to complete a long endurance event, either SV or HR must increase. In order to change SV (*see equation 2)* the preload, afterload or contractility must be altered.

Preload impacts SV by changing how much stretching of the myocardium occurs. The relationship between cardiac sarcomere length and the tension generated by the heart is described by the Frank-Starling law. This dictates that if there is more venous return, and therefore a larger end diastolic volume, the cardiac sarcomeres will be stretched more and the recoil from this tension will propel a larger amount of blood (*See figure 12)*. This is analogous to a rubber band with the amount of stretch equating the amount of preload. If the rubber band is stretched a little it will generate tension and cause a recoil, however if the rubber band is stretched a lot the tension will be greater and therefore it will have a larger recoil. Consequently, a higher preload will produce a higher stroke volume and therefore a higher cardiac output. This is only true up to a point. If a rubber band is stretched too far it will snap; similarly if there is too much stretch the heart will not be able to form as many cross bridges and ventricular performance will begin to decline.

Figure 13: Frank- Starling Law



There are six main factors that influence preload which is directly related to how much myocardial stretching occurs. These factors are blood volume, body position, the respiratory

pump, the autonomic nervous system, the muscular pump, and heart rate (Keen 233-238). As blood volume increases the end diastolic volume will increase due to a higher venous return which stretches the heart (*see figure 13).* Blood volume can vary depending on hydration levels since a large portion of blood is comprised of water. During an Ironman Triathlon becoming dehydrated can cause blood volume to decrease which in turn lowers the venous return and decreases cardiac performance via preload. This is one reason why hydration plays such a huge role in the Ironman, especially since the race is so long.

Body position also impacts preload. If someone is lying down, the returning blood does not have to fight gravity, so there will be greater return which has a positive relationship with end diastolic volume and for that reason stroke volume. Based on this it can be inferred that during the swim, when the body is prone in the water and using the smallest muscular groups, the heart does not have to work as hard to meet metabolic demands. On the bike and run legs however, the body is in a more upright position and gravity becomes an adverse factor to returning blood.

Another factor is the intrathoracic pressure or respiratory pump. During inspiration a negative pressure is created as the diaphragm expands, helping to draw blood back toward the heart as it flows with the pressure gradient from high to low. In an Ironman ventilation rises compared to at rest due to the higher levels of arterial CO_2 (Keen 201-202). This results in a larger respiratory pump affect which further increases preload. The increased preload allows a higher stroke volume to be achieved.

Similar to the respiratory pump is the pumping action of skeletal muscle. Since the veins within the body have valves that only flow in one direction, during muscular contraction the blood vessels are compressed forcing blood toward the heart and increasing venous return. In a

race, as an athlete's muscles propel them forward it also helps to propel blood back to the right atrium of the heart.

Venous tone also plays a role in preload and is controlled by the nervous system of the body. At rest, around 60% of blood is on the venous side. If the sympathetic branch of the autonomic nervous system is stimulated as in exercise, vasoconstriction similar to the muscular pump effect will occur, creating higher venous return (Cohen: Myocardial contractility).

Figure 14: Preload Factors During an Ironman

Factor	**During Ironman**	**EDV**
Total blood volume	Likely decrease from dehydration	Decrease
Body position	Swim: prone Bike/Run: upright	S: Increase B/R: Decrease
Respiratory Pump	Larger negative pressure so increases	Increase
Muscular Pump	Increases	Increases
Autonomic NS	Venoconstriction	Increases
Heart Rate	Increases	Decreases

Finally, heart rate greatly dictates the time the heart has to fill with blood which directly impacts preload. There is an inverse relationship between heart rate and preload because if the filling time is shortened, less blood will be able to enter the right atrium before systole occurs. When heart rate is greater that ~160 beats/min, stroke volume will begin to regress due to a decreased EDV and preload (Tortora 713).

The next factor that impacts SV is afterload. As afterload increases the end systolic volume also increases because less blood is able to leave the heart during contraction. This results in a lower SV, since there is an inverse relationship between SV and ESV (*see equation 2)*. Therefore, a high afterload leads to decreased ventricular performance and can limit race performance.

As mentioned previously, afterload can also be thought of as ventricular wall stress. Wall stress (σ) can be equated to the ventricular pressure (P) multiplied by the ventricular radius (r), divided by the thickness of the wall (h) (*see equation 3).* This means that a thicker heart wall would experience less stress as it is distributed throughout more sarcomere units. A hypertrophied heart is often an indicator of a pathophysiological adaptation and results in a smaller chamber size as the wall thickness increases inwards. However, exercise can induce hypertrophy of the heart in a positive way. Therefore, with endurance training the heart wall thickness increases causing the wall stress, or afterload that individual myocardial fibers must overcome, to decrease (*see equation 3).*

Equation 3: Cardiac Output

$$\sigma \propto \frac{P \cdot r}{h}$$

Other factors that impact afterload are age and blood pressure. Older Ironman triathletes are more likely to have less compliant and stiffer blood vessels as a result of natural degeneration. This means that when the heart pumps blood out of the aortic valve, the aorta wall will not expand as easily to accept the blood which creates more resistance and increases afterload. Having high blood pressure, or hypertension, also increases afterload. The aortic valve cannot open to eject blood until the LV generates more pressure than the pressure than is in the aorta. Therefore if blood pressure is higher, the LV will have to work harder to eject blood. A few risk factors for hypertension include: obesity, old age, stress, smoking, and too much alcohol consumption. A high afterload can be viewed as a negative thing for an Ironman triathlete since it causes a decreased SV.

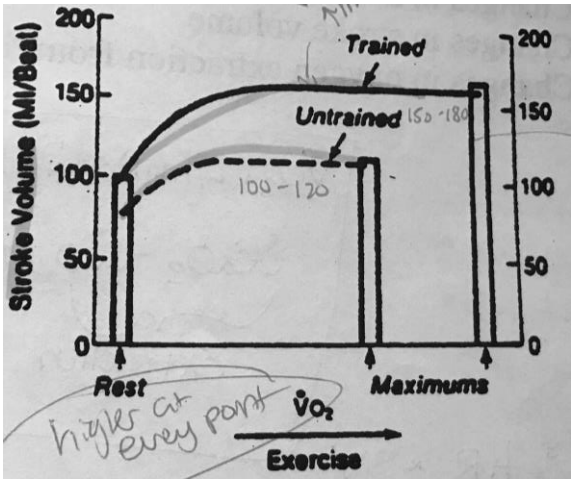
If the heart was a tube of toothpaste and blood was the toothpaste itself, preload would be equivalent to the amount of toothpaste inside the tube. It is going to be easier to push out toothpaste if the tube is filled more. Afterload is comparable to having the cap on the end of the

tube, if it is on tightly it will be very difficult to get any toothpaste out, whereas if the cap is barely on or off it becomes easier. There is still one essential factor to extracting toothpaste and that is how hard you push the tube or the forcefulness which is analogous to the contractility of the heart. Contractility is essential to maintaining a high SV and can be altered by neurological input. When SNS input increases such as at the start of a race, the adrenal medulla releases norepinephrine which then stimulates beta-adrenergic receptors. As a result, calcium is released from the sarcoplasmic reticulum as discussed earlier (page 4 of thesis). Calcium then promotes cross bridge formation and increases the forcefulness that the heart contracts with. When carbon dioxide levels within the coronary blood are high myocardial contractility will also increase in order to bring more oxygen rich blood to the heart itself so that it can continue to work efficiently (Cohen: Myocardial Contractility 7).

During the start of the race it is likely that an athlete will experience some level of stress and anticipation. The anticipatory response will activate the SNS and increase contractility of the heart causing it to expel more blood on each beat. Later on in the swim, if not enough oxygen is being taken in and there is hypoxia the baroreceptors in the coronary arteries will signal the brain to release more NE to further increase contractility. These body mechanisms help to maintain homeostasis despite the high oxygen and nutrient demands being placed on the body during an Ironman triathlon.

With training adaptations, the maximum stroke volume of an athlete increases and is higher even at rest. The average untrained person will have a maximum SV of 100-120 ml/beat

Figure 15: SV of Trained vs Untrained



whereas a trained individual will have a maximum SV closer to 150-180 ml/beat (Keen 244). This is why many athletes have a low resting heart rate- the blood the body needs at rest does not change, however the body is able to eject more blood per beat and therefore the number of beats can be reduced.

Cardiac output is more than just SV and the factors that impact it (preload, afterload, and contractility). Rather, it is the SV multiplied by the heart rate which is also a crucial player in endurance training and racing. When we exercise, increasing HR is one way the body can expel more blood to meet the body's demands. The mechanism for this also involves NE being released during SNS activation leading to a more rapid spontaneous depolarization within the SA node which increases heart rate (Keen 253). Despite training, a person's maximum heart rate usually does not change and if it does it may be slightly reduced rather than increased (Keen 244). The reduction in maximum HR may allow for a slightly longer filling time. As mentioned previously, trained individuals will have a lower resting HR due to higher SV. It is also important to note that HR generally increases linearly in response to VO_2 demands.

The start of a race sees a rapid alteration of HR as the body goes from rest to active. During the onset of exercise proprioceptors will alert the medulla oblongata of the change in muscle firing and body position which will lead to increased impulses being sent to the sympathetic nervous system in order to increase heart rate to meet the high demands of an Ironman Triathlon.

During the swim, heart rate will likely be the lowest of all three disciplines. This is dependent on the water temperature, body position, and compression of a wetsuit. A lower water temperature is common during open water Ironman swims. This will lead to a lower heart rate due to the mammalian diving response. When cold-sensitive receptors on the face, such as in the nasal cavity, signal the cold temperature to the brain, the vagus nerve can be activated which slows down the heart rate in an attempt to conserve energy. The prone body position also means that the heart does not have to work as hard to pump blood. Similarly, a wetsuit compresses the body making it easier for blood to return to the heart. A buoyant wetsuit also makes a swimmer more hydrodynamic and increases swimming efficiency by lifting the hips of the swimmer up to reduce drag. Therefore, an athlete with a wetsuit on going the same speed as a swimmer without a wetsuit will use less energy and therefore have a lower heart rate. On the other hand some athletes have inefficient swimming technique and will therefore have to work harder to move through the water, even with a wetsuit, which can spike heart rate. Altogether, it is expected that after the initial anticipatory response heart rate will be the lowest during the swim.

In contrast to the swim, the run portion of an Ironman will have the highest heart rate, and the heart will be working the hardest. This is partially due to the large muscle groups that are utilized during the run. A larger muscle group will require more oxygen and nutrients that a smaller group, thus demand is higher on the run. Furthermore, running is the most upright position in the race and the heart must fight against gravity to bring blood back to the RA. All in all, the run is likely the most stressful aspect of an Ironman in terms of demand placed on the heart.

III.b. Blood Vessels

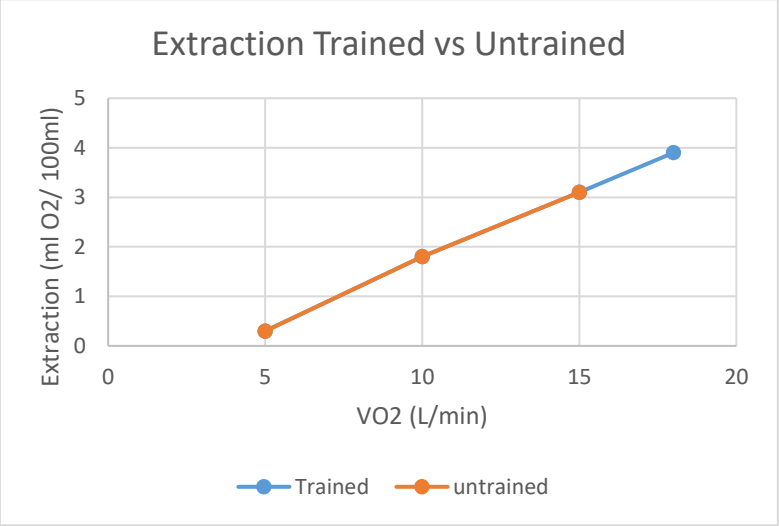
Training adaptations of the heart, as previously discussed, can allow for more blood to circulate the body during an Ironman triathlon. This is essential to meeting the oxygen-nutrient demands of an endurance event. However, circulating more blood is not useful if the body cannot efficiently extract the oxygen from it. The amount of oxygen extracted can be determined by measuring the content of oxygen in arterial blood (C_aO_2) and subtracting the amount of oxygen remaining in the mixed venous blood returning to the heart (C_vO_2) (*see equation 4*). As effort increases during a race and VO_2 increases, the extraction also increases. The amount of oxygen within the arterial blood will stay fairly constant since at rest it is already at a 98% saturation level. However, the content of oxygen that is in the returning mixed venous blood will be much lower since the body removed more oxygen as blood flowed through the capillaries (Keen 187). This higher extraction is facilitated by the partial pressure of oxygen (PO_2) within the muscle cells during exercise. When the metabolic activity of a muscle cell is high, as during a race or training, the cell consumes more oxygen. The higher the intensity of exercise, the more oxygen is consumed and the lower the PO_2 is. This creates a large gradient for diffusion of blood from the capillaries and into the muscles. The PO_2 of the blood may be around 90 (mm Hg) or higher, while that of the muscle cell may be as low as 5(mm Hg). This huge difference in PO_2 is why extraction of oxygen can increase (Keen 195).

Equation 4: Oxygen Extraction

$$Extraction\ ({}^{ml\ O_2}/_{100\ ml}) = CaO_2 - CvO_2$$

Even in untrained individuals, extraction would increase during an endurance event, however a trained individual would also be able to increase maximal extraction due to changes that occur within the body. The untrained athlete may reach an extraction of 15 (ml O_2/100ml)

Figure 16: Extraction of Trained vs Untrained



while a trained Ironman athlete may reach between 18-20 (ml O_2/ 100ml) allowing them to increase their VO_2 (L/min). This is possible due to an increase in capillary density, mitochondrial density, and aerobic enzymes that occurs with training (Keen 250). A larger capillary density allows for more oxygen diffusion into the tissues. More mitochondria within the muscle cells is also vital. Mitochondria can use many different substrates to create ATP, such as CHO and fats, however the reaction is limited by the amount of oxygen delivered to the muscles. As mitochondria concentration increases, the capacity for the mitochondria to use fatty acid metabolism over using a glycogen source increases. The effect of this is that the respiratory exchange ratio or RER decreases. RER describes how much energy, in the form of kcals, is produced per L of oxygen. Some substrates give more energy for the same amount of oxygen used during metabolism and are therefore more efficient (Keen 21-23). Through training the mitochondrial density increases allowing for the mitochondria to rely on a more efficient fuel source that requires less oxygen, which is the limiting factor in production. Therefore, the RER of an athlete is lower than that seen in an untrained person and the start of fatigue can be delayed as their cells are more capable of meeting the high metabolic needs of an endurance event (Keen 312).

Since oxygen is the limiting factor in energy production, it is essential that blood flow to muscles is high. Blood flow through a vessel (F) can be seen as pressure differences (ΔP) divided by resistance (R) as seen in *equation 5*. Resistance can be impacted by how thick the blood is, the length of a vessel, and the radius of the vessel which is dependent on how vasoconstricted or vasodilated it is. A larger person will naturally have longer vessels. Therefore an ironman athlete with minimal extra body weight will have less resistance to blood flow than a larger person. Although length plays a part, radius is the largest factor when evaluating resistance since resistance is inversely proportional to the radius to the 4th power (*see equation 6)*. This means that even slight changes in radii can have profound impacts (Cohen: Blood Vessels 9-11). During an Ironman triathlon, vasodilation of blood vessels occurs in order to increase blood flow. Many things can decrease the contraction of smooth muscle in the arteriolar walls causing vasodilation such as decreased levels of oxygen and sympathetic stimulation, increased levels of carbon dioxide or lactic acid, and finally heat (Cohen: Blood Vessels 24). During a race, muscle cells will use more oxygen leading to decreased O_2 levels and increased CO_2 levels, lactic acid will also accumulate in muscles which produce heat as a byproduct of a higher metabolism. All of these factors tell the parasympathetic system to release vasodilating factors like Endothelial-derived relaxing factor (EDRF). EDRF inhibits the flow of Ca2+ into the smooth muscle cells, thus preventing them from contracting, which results in local arteriolar vasodilation (Cohen: Blood Vessels 26).

Equation 5 & 6: Blood Flow and Resistance

$F = \Delta P/R$ $\qquad$ $R = 1/r^4$

When at rest, blood only flows through a small portion of the capillary network due to the vasoconstriction of precapillary sphincters. Similar to the systemic vascular resistance, during

exercise these sphincters relax and blood can flow throughout the entire capillary system in order to bring oxygen to the tissues (Tortora 734).

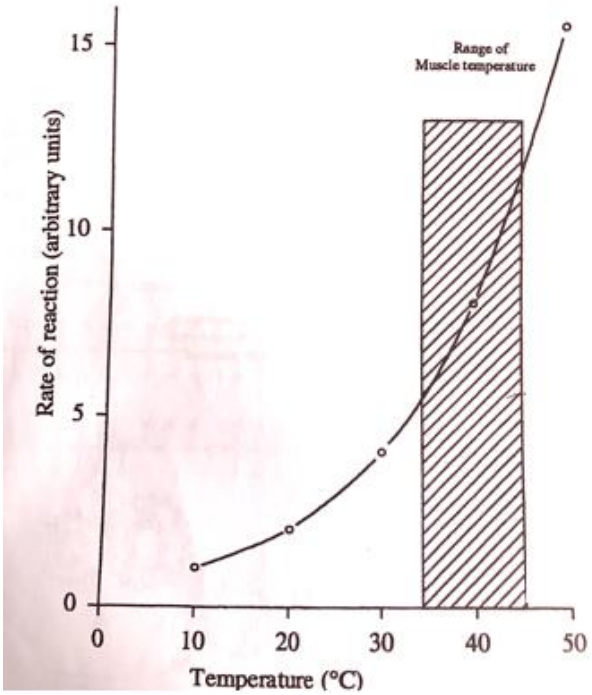
While blood flow to active muscles and other active organs are increased through vasodilation of blood vessels, as described earlier blood flow to non-essential organs is minimized. This efficient re-routing of blood takes advantage of the fact that fluids travel down the path of least resistance which can be explicitly seen in *equation 5.* Shuttling blood where it needs to go is controlled by the vasoconstriction or vasodilation of the blood vessels and is essential to completing an endurance event. Without this mechanism, blood that is desperately needed by muscles to keep contracting would also be routed towards organ systems like the digestive system.

All in all, the main difference between the average person and a trained Ironman athlete in regards to blood vessels is the density of their capillary beds, the ability to extract oxygen from blood, and the efficiency at which their body releases and reacts to parasympathetic activation.

III.c. Blood

It was mentioned in *section III.b.* that trained individuals will have a larger capillary density. If there is a larger concentration of capillaries, there is also a larger transit time for red blood cells. This means that there is more time for internal respiration, or the exchange of oxygen and carbon dioxide in the tissues. A major concern during any endurance event is maintaining a stable temperature. The exergonic process of taking energy from food to make ATP is a cellular reaction, and no reaction is 100% efficient. The human body is approximately 40% efficient at producing energy and the 60% inefficiency is seen as the release of heat (Keen 9). There is a physiologic range of temperatures between 25-45 ^{0}C in which muscles function properly. Within this range, a 10^{0}C increase in temperature results in the doubling of the rate of

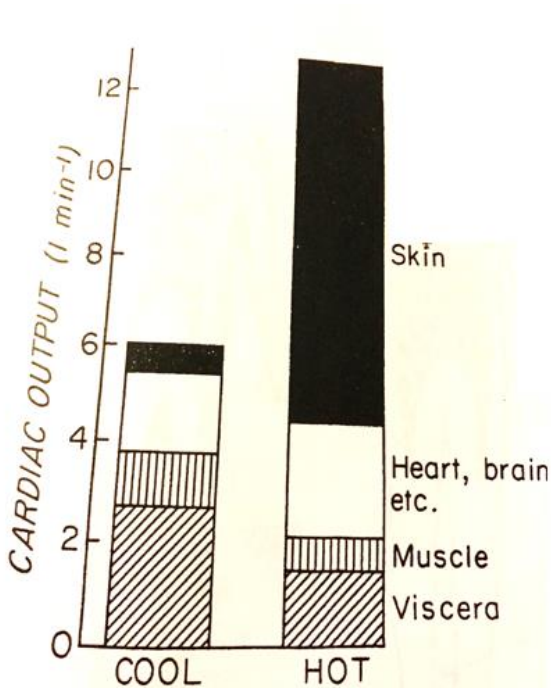
Figure 17: Q10 effect



reaction which is referred to as the Q10 effect (*see figure 17)*. This means that at the upper end of the temperature limit the rate of reactions, such as glycolysis, will be at maximal functioning. This gives a physiological basis for warming up, however at temperatures higher than the safe physiological range, proteins begin to be denatured and the body begins to shut down. In order to prevent this from happening, the body utilizes an evaporative cooling system.

During an Ironman triathlon, the core temperature of an athlete will begin to rise as heated blood from active muscles mixes with venous blood. While during the swim the core temperature is likely to stay relatively normal due to the cooling effect of the water, on the bike and especially the run temperatures are likely to rise. As the core temperature rises, the blood vessels of the skin begin to vasodilate due to an increase in parasympathetic activity. This allows 60% of blood to be shunted to the skin (Keen 299). Since the vasculature of the skin is very close to the surface, heat from

Figure 18: Thermoregulation via Blood Distribution



the blood can be released to the outside environment, given that the temperature outside is cooler than inside. This is because blood plasma can absorb and then release heat, which is a form of energy that moves from high to low concentrations. The hotter the core temperature gets, the larger the PSNS response will be and the more blood that will be shunted to the surface. Unfortunately, if temperatures get too high, the evaporative cooling system comes at the cost of blood flow to muscles. At a certain point the body will see cooling the core as more vital than exercise performance and the vasculature to the muscles and viscera will be vasoconstricted to reduce blood flow (*see Figure 18).* This competition between the skin to cool the body, and the muscles to perform during a long endurance event can greatly impact performance. For example, if the core temperature rises, more blood will be sent to the vasculature of the skin which means that less blood will be returning to the heart at any given time. This decrease the preload of the heart and therefore lowers stroke volume. To compensate for this lower SV, heart rate will rise. Still, the increase in HR can only compensate for a short period of time and eventually the cardiac output will decrease (Keen 301). While this may seem inconvenient to many athletes, in reality the body's ability to theroregulate via blood is an incredible defense mechanism that can keep an athlete going even on a warm day.

With training and heat acclimatization the body develops a decreased threshold for sweating as well as a higher rate of sweating (*see figure 19)*. This means that the sweat response will be activated earlier than in untrained athletes. This may initially seem counterintuitive, however it actually allows the body to cool itself more efficiently and postpones the need to divert blood from the muscles to the skin. Furthermore, the sweat glands themselves become more adept at reabsorbing Na^+ ions in order to maintain electrolyte balances throughout the body (Keen 306).

Figure 19: Internal Temperature and Onset of Sweating

Trained or Untrained	**Acclimatized/ Unacclimatized**	**Internal temperature at onset of Sweating (^{0}F)**
Trained	Acclimatized	~98.8
Trained	Unacclimatized	~99.5
Untrained	Unacclimatized	~99.8

Regardless of whether the temperature is warm on race day, completing an Ironman can cause an athlete to lose a lot of fluids. Since blood plasma is 90% water, dehydration can cause the plasma volume of blood to shrink resulting in thicker sludgy blood as well as a lower blood volume (Cohen: Blood 9). Both of these factors can negatively impact performance. If blood is thicker it will create more resistance and therefore decrease blood flow, therefore to maintain the same blood flow the heart will have to pump harder to overcome the resistance. A lower blood volume also implies that the blood returning to the heart may be lower and consequently preload would be decreased. A decrease in preload then leads to a lower stroke volume and therefore diminished cardiac output. In fact, it has been shown that oxygen uptake at an exercise load that could be tolerated for 5.5 minutes under normal conditions, could only be tolerated for 3.5 minutes after dehydration (Keen 308). Due to the large impact that fluids can have on blood, it is vital that athletes hydrate well during the bike and run portions of an Ironman.

Aside from helping to regulate core temperature, blood also has a vital role in maintaining stable pH levels within the body. During the first 5 minutes of an Ironman as athletes sprint into the water, the body relies on the glycolytic mechanism for fuel while the aerobic mechanism that uses mitochondrial respiration kicks in. Glycolytic processes are limited by changes in pH, rather than oxygen. When glycolysis is occurring rapidly there is no time for pyruvate, a product of the reaction, to enter the mitochondria as a substrate for aerobic

metabolism. When this occurs pyruvate is reduced to lactate in the cytoplasm of the cells. Since lactic acid inhibits actin-myosin interactions and leads to metabolic acidosis this is undesirable (Keen 14). Blood can act via buffers to convert strong acids, such as lactate, into a weak non-threatening one. Hydrogen protons are also created during metabolism, and these can generally be turned into water by attaching an oxygen. During fast metabolism though, H^+ can accumulate causing the pH to drop which can slow down an athlete. To counteract the acidity, hemoglobin binds these extra protons. In the process, the H^+ causes hemoglobin's shape to change in a way that decreases binding affinity to oxygen. Since this allows for oxygen to be unloaded in the tissues faster, this is a win-win mechanism in which the pH is controlled and oxygen is delivered rapidly (Keen 186).

A very critical property of blood is its hemoglobin which has higher affinities toward oxygen based on the conditions surrounding it (*see figure 7section I.C).* This not only makes oxygen more likely to leave the hemoglobin within the tissues during exercise, but also makes oxygen more likely to bind to hemoglobin within the lungs. When the tissues use more oxygen the partial pressure of O_2 drops and PCO_2 rises. The large gradient between the oxygen in the RBC and tissues makes the oxygen readily dissociate when it reaches the tissues. This is also facilitated by an increase in temperature due to the heat produced and a drop in pH due to lactate (Keen 186). This quality of hemoglobin allows for necessary oxygen to reach tissues during exercise. Taken altogether, the properties of blood and hemoglobin inherently allow the body to be efficient in times of high metabolic stress.

IV. Research Related to the Physiology of Triathlon

IV.a. Predictors of Race Performance

We have discussed various physiological changes that occur that allow the body to handle an Ironman Triathlon, however is it possible for certain adaptations to predict race performance? A study conducted in 2013, *Right Ventricle Best Predicts the Race Performance in Amateur IM Athletes,* tries to answer this question. Previous research has observed a correlation between a lean body type and race times, while other studies have focused on the influence that training volume and intensity have. However, these things (body type, training volume/intensity) are not the reason for faster race times but rather the mechanism to achieve physiological alterations that make the body capable of performing at a higher level. For instance, while training intensity may lead to faster times it is the physiological changes that occur do to the intensity rather than the intensity itself that cause better racing performance. In order to evaluate potential cardiac predictors of Ironman finisher times, this study used an echocardiographic exam of the left and right sides of the heart as well as anthropometric measures including BP, HR, body mass, height, percent body fat, and capillary blood properties. Lastly, a training diary that detailed training distance, duration, and speed for all three disciplines was used. The study population was comprised of 38 amateur Ironman athletes participating in the 2010 Ironman Switzerland race in Zurich (Bernheim et al. 1594).

The study found that LV myocardial mass index had a moderate correlation to race performance. The strongest predictor of race performance however was RV end-diastolic area and RV surface area (Bernheim et al. 1595-1596). The left ventricle (LV) of the heart is often seen as more important than the right ventricle since it must contract with enough force to send blood to the periphery. However, the right ventricle (RV) has also been shown to play a vital role

due to the fact that the LV can only eject what it has been given. This makes the RV a potential limiting factor in endurance events. Overall, this study reveals that RV remodeling may be an underestimated aspect of athlete heart adaptation in amateur Ironman triathletes. One mechanism for this is that the RV "exhibited a relatively greater increase in estimated end-systolic wall stress during exercise than the LV" and this stress can then lead to more extensive RV remodeling (Bernheim et al. 1597). All in all RV size was found to be an independent predictor of max O2 consumption which plays a vital role in exercise efficiency and therefore racing performance (Bernheim et al. 1594). It should be noted that there are limitations to this study as a relatively small group was evaluated, however the researchers did show comparable anthropometric data to a previously investigated larger group of IM triathletes.

IV.b. Is Ironman Distance Racing Harmful?

The reason the cardiovascular system undergoes such intense remodeling is that an Ironman Triathlon (IMT) places a great deal of stress on the body. Due to the increasing popularity of the IMT, in 2012 the Mayo Clinic conducted a literature review of IMT data in order to evaluate the potential adverse cardiovascular effects. The review found that long-term excessive endurance exercise can be harmful as it causes "pathologic structural remodeling of heart and large arteries" (O' Keefe et al. 587). One mechanism for the pathophysiologic remodeling is that an IMT generates lots of free radicals in the body, more than the buffering systems can handle, which increases oxidative stress. This repetitive cycle can stimulate an immune response by activating lymphocytes, macrophages, and mast cells. These cells secrete cytokines telling myofibroblasts to proliferate. Thus ultimately leads to the secretion of procollagen which cross-links and adds collagen to the myocardium and large arteries. This is bad as it creates a stiff myocardium and is comparable to laying down scar tissue on the heart (O' Keefe et al. 592).

The literature review also found research demonstrating that long-term vigorous aerobic endurance training is associated with rhythmic abnormalities as well as a 5x increase in prevalence of atrial fibrillation potentially as a result of chronic RA and RV dilation(O' Keefe et al. 589, 592). While the body generally bounces back fairly quickly upon completing an IMT, theoretically too many races can put a strain of the cardiovascular system.

V. Conclusion

There are many cardiovascular changes that occur in order for the body to cope with the stress of an Ironman Triathlon. Some of these alterations are a result of the chronic endurance training necessary to prepare the body, while other mechanisms occur due to the acute stress of the actual race (*see figure 20)*. Without the cardiovascular system's ability to adapt it would be physically impossible to complete an Ironman Triathlon and racing performance is highly dependent on how well the heart, blood vessels, and blood can make the modifications discussed throughout.

Figure 20: Acute and Chronic Adaptations

Chronic Endurance Training Adaptations:	**Acute Adaptations from Stress of Race:**
Exercise Induced Cardiac Hypertrophy	Redistribution of blood to muscles and/or skin
Maximum heart rate increases from 100-120 ml/bt to 150-180 ml/bt.	Higher preload-> higher stroke volume-> higher cardiac output
Oxygen extraction increases from 15 ml O2/100ml to 18-20 ml O2/100ml	Increased contractility due to anticipatory response
Capillary bed density to commonly used muscles increases	Oxygen extraction at tissues increases
Resting HR decreases due to increased SV	Hemoglobin dissociation:O_2 readily leaves RBC in tissues and attaches to RBC in lungs
Sweat response becomes more efficient	Precapillary sphincters vasodilate
Maximum Cardiac Output and VO_2 max increase	Cardiac filling time/ diastole decreases

VI. Citations

Britt, Raymond. Average Ironman Finish Times." *How Much Time Does It Take to Finish an Ironman Triathlon? Average Ironman Finish Times*. RunTri, 2016. Web. 04 Mar. 2017.

Bernheim, Alain Marcel et al. "Right Ventricle Best Predicts the Race Performance in Amateur Ironman Athletes." Medicine & Science in Sports & Exercise, vol. 45, no. 8, Aug. 2013, pp. 1593-1599.

Chang, Edward. "Triathlon Bike Shopping - Road Bike vs. Tri Bike." *MultisportED - Road to IRONMAN*. N.p., n.d. Web. 05 Apr. 2017.

Cohen, Zoe. "Arteries and Arterioles." Cardiovascular Physiology. 1 Mar. 2016. Lecture.

Cohen, Zoe. "Blood." Cardiovascular Physiology. 18 Feb. 2016. Lecture.

Cohen, Zoe. "Blood Pressure." Cardiovascular Physiology. 12 Apr. 2016. Lecture.

Cohen, Zoe. "Capillaries and Veins." Cardiovascular Physiology. 8 Mar. 2016. Lecture.

Cohen, Zoe. "Intro to CV." Cardiovascular Physiology. 19 Jan. 2016. Lecture.

Cohen, Zoe. "Myocardial Contractility." Cardiovascular Physiology. 26 Jan. 2016. Lecture

Cohen, Zoe. "Structural and Contractile Properties of Cardiac Myocytes." Cardiovascular Physiology. 31 Jan. 2016. Lecture.

"The Ironman Story." *Ironman*. N.p., n.d. Web.

Klabunde, Richard E. "Cardiac Afterload." *Cardiovascular Physiology Concepts*. N.p., 07 July 2015. Web. 05 Mar. 2017.

Keen, Douglas. *Exercise and Environmental Physiology Class Notes*. N.p.: n.p., 2015. Print. PSIO 420.

O'Keefe, James H, et al. "Potential Adverse Cardiovascular Effects from Excessive Endurance Exercise." Mayo Clinic Proceedings, vol. 87, no. 6, June 2012, pp. 587-595.

Tortora, Gerard Joseph., and Bryan Derrickson. *Principles of Anatomy and Physiology*. 14th ed. Hoboken, NJ: Wiley, 2011. Print.

VII. Appendix A

Thesis Poster created by Marissa Wheeler and Dr. Zoe Cohen: