1) HEART FACTS AND TRIVIA

What's in a Name?

The existence of the heart was well known to the Greeks, who gave it the name Kardia, still surviving in modern words such as cardiac and tachycardia. Aristotle believed that the heart was the seat of the soul and the center of man. Romans modified Kardia to Cor, the latter word still surviving in "cordial greetings". The old Teutonic word herton was also derived from Cor and gives us heart via the medieval heorte.

Where is it Located?

Dumb question right? Well if you answered left chest, you're wrong! The heart is situated almost dead center in the middle of the chest nested between the two lungs. However, the apex or tip of the heart is shifted towards the left chest wall and hits against the ribs during contraction. Consequently, the rhythm is best detected on the left side, just below the pectoralis.

How Big is it?

It is generally about the size of your fist. This is not really very big when you think about the job it does. In some animals, such as horses, the heart size to body size ratio is much greater. This helps explain why horses are such great endurance athletes! The heart is also bigger in champion endurance athletes, due to genetics and training. (***see subcategory-(b) below***). The average untrained heart can pump about 15 to 20 liters of blood per minute at max. Large, elite athletes may have a maximal cardiac output of nearly 40 liters / min. This is a huge flow moving through a pump the size of your fist! To get some perspective on these output rates, go to your kitchen sink and turn on the water full blast. Now find a milk jug or something that will give you a measure of volume. I bet you find that your faucet does not flow as fast as the heart can pump.

In a sense, the heart is really two linked pumps, the left heart and the right. Both sides pump the same amount of blood, but to different locations at different pressures. The right side pump (right ventricle) pumps oxygen-depleted blood that has returned from the body to the lungs for reoxygenation. This is a short trip and requires little pressure development, so the right ventricle is rather thin walled, like a fireplace bellows. The left side (left ventricle) is the real workhorse, pumping oxygenated blood that has returned from the lungs (the right and left side of the heart are thus connected) to the entire body. That means moving blood through an incredible maze of blood vessels from the top of the head to the toes! Consequently it must develop more pressure each beat (about 120mm Hg at rest). The left heart muscle is thicker as a result, just as your bicep would become thicker if you had to lift heavy weights with it all day.

How Does it Pump Blood?

Classically, we have been taught that the heart squeezes blood through the aorta by decreasing the external circumference of the heart. This view is supported by the fact that during heart surgery (with the chest cracked open), the heart does pump in this manner. However, under normal conditions, the heart operates within the thoracic cavity in a closed, fluid-filled volume. There is now growing evidence to indicate that during exercise, the heart performs more like a piston or a vacuum pump, with little change in external circumference. As we learn more about the dynamics of heart function, it is evident that this model is critical to the efficiency of the heart as a pump. More recent models of heart performance indicate that the heart takes advantage of vacuum effects and fluid inertia as heart rate increases during exercise. One reason why artificial hearts have performed so poorly is that they have tried to use a design based on erroneous assumptions about how the human heart pumps. The classical view of heart pumping mechanics will die slowly, due to its pervasiveness. However, it seems reasonable to say that the heart performs more like a vacuum pump than like a hand squeezing the juice out of a lemon. When the heart pumps, the ventricular wall's outer diameter changes little, while the internal diameter dramatically decreases as blood is ejected from the ventricle.

What Controls the Heart Rate?

Now this is a tough question to answer without using a little physiology lingo. Unlike skeletal muscle, which is under voluntary control, the heart is an involuntary muscle. Most of us cannot just tell our heart to slow down or speed up (biofeedback training not withstanding). The beating frequency (heart rate) is controlled by the balance of stimulation coming from the sympathetic and parasympathetic branches of the Autonomic Nervous System. Both nervous inputs to the heart converge on a small area of tissue in the right atrium called the Sino-atrial (SA) node. Parasympathetic (rest and recover) stimulation tends to slow down the rate, while sympathetic (fight or flight) input increases the rate (and the force of contraction). Normally, there is a balance between the two inputs leaning toward the parasympathetic side. However, even without any nervous input, the heart will beat automatically due to some unique features of its membrane physiology. This intrinsic rate is quite slow however (about 20 bpm). A purely parasympathetic stimulation will result in a heart rate of about 30. So the average untrained person has a resting heart rate of about 70 as a result of some constant sympathetic stimulation. With training, the balance between parasympathetic and sympathetic stimulation tends to shift in favour of the parasympathetic, resulting in a slower resting heart rate. Elite endurance athletes may have resting HRs of 35 to 40. Values of 28 have been reported!

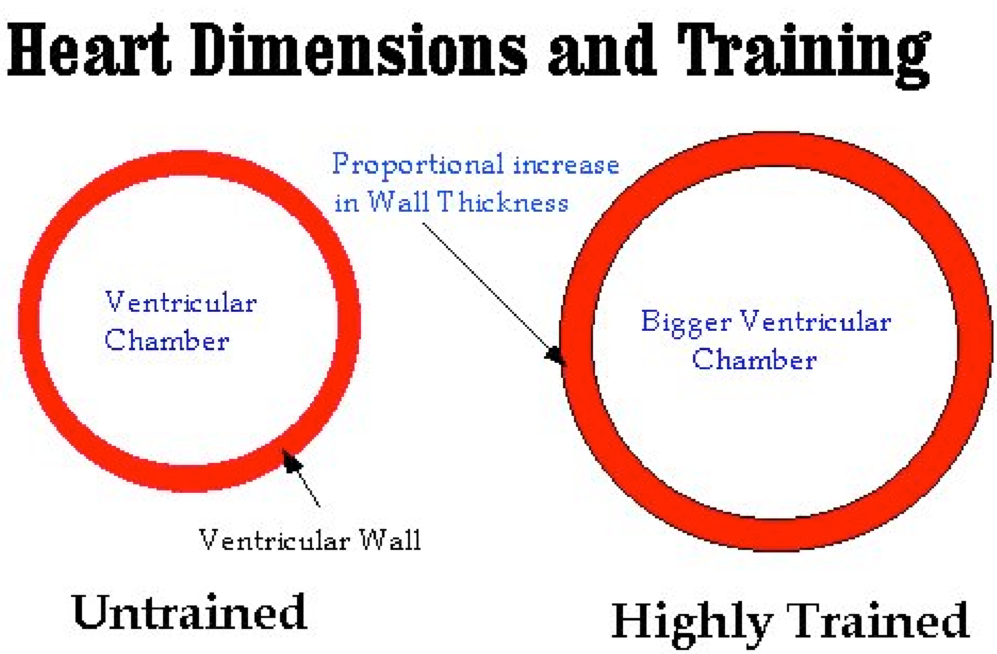
The initiation of activity results first in a withdrawal of the parasympathetic stimulation (up to a heart rate of about 100) followed by an increase in sympathetic stimulation with more intense activity up to the maximum heart rate ***(see subcategory-(d) below).*** A number of studies have demonstrated that maximal heart rate actually tends to DECREASE with high volumes of endurance training. The average of a number of studies is about a 7 beat reduction in maximal heart rate after training compared to the untrained state. Anecdotally, it also appears that even in athletes, periods of very high volume can transiently cause a reduction in the maximal heart rate, or perhaps more correctly a reduction in the capacity of the sympathetic nervous system to maximally mobilize the heart rate. We have tested junior XC skiers before and after a 10 day training camp filled with a lot of training volume. On average, the team showed a slight reduction in VO2 max despite being very fit, and their maximal heart rate during a VO2 max test was perhaps 4 beats per minute lower. The athletes were very fit, but could not fully mobilize; they lacked that last gear. After a few days of relative rest, they were back to normal.

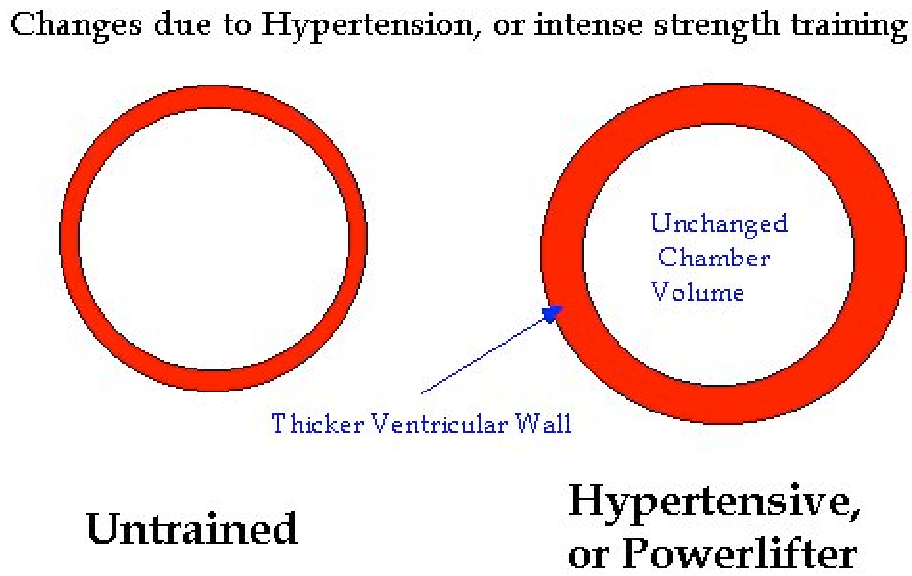
Will Training Make My Maximal Heart Rate Increase?

The answer to this question has just been answered. No, the maximum heart rate is not increased by training! As we get older, our maximum heart rate decreases. The major difference in the endurance trained heart is a bigger stroke volume. The trained heart gets bigger and pumps more blood each beat. So, that small reduction in maximal heart rate is more than compensated for by an increase in stroke volume.

i) More About How the Heart Adapts to Training

More accurately, the End Diastolic Volume (EDV) increases in the trained heart. EDV is the volume of blood in the left ventricle just prior to the initiation of contraction. End Systolic Volume (ESV) is the residual volume remaining immediately after contraction. Ejection fraction is the ratio (EDV-ESV)/EDV. The Frank-Starling Law tells us that if more blood enters the heart, more will be ejected. I will spare you the reasons why that is true. After training, the heart operates on a more efficient portion of the length-tension curve. The Increased End Diastolic Volume and resulting increased stroke volume is accomplished three ways. **First, the decreased heart rate increases ventricular filling time. Second, the ventricle increases in size** through what is termed *eccentric hypertrophy*. The volume of the ventricular chambers (lumen) increases due to longitudinal sarcomere addition. This adaptation is in contrast to the ventricular wall thickening or concentric hypertrophy without increased lumen volume that is observed in hypertensive patients, or in people who train intensely with weights. There is a small increase in ventricular wall thickness in the endurance trained heart. This balances the increased wall tension associated with operating at an increased diameter (**Law of Laplace**). Graphic representation below.





Finally, the EDV increases after training due to an **increase in blood volume**. More blood volume results in greater venous return of blood to the heart at any given peipheral capacitance. Blood volume/kg bodyweight is about 15% higher than untrained. This adaptation is quite rapid, and helps explain why VO2 max is significantly increased after only 1 week of training in previously sedentary subjects. This blood volume expansion is also rapidly lost (3-7 days) with inactivity. The increased blood volume is due to both an increase in blood plasma and an increase in red blood cells. However, the plasma volume change is slightly greater so that blood hematocrit is slightly reduced with training (exercise pseudoanemia).

ii) Maximal Oxygen Consumption - The VO2 max

If you walk into the locker room of a bunch of American Football players, bragging rights are reserved for the man with the heaviest bench press. Similarly, talk to a group of endurance athletes that are "in the know", and conversation will eventually turn to "What is your VO2 max?" A high maximal oxygen consumption is indeed one of the hallmark characteristics of great endurance performers in running, cycling, rowing and cross-country skiing, so it must be pretty important. What is it and how is it measured?

VO2 max defined

VO2 max is the maximum volume of oxygen that by the body can consume during intense, whole-body exercise, while breathing air at sea

level. This volume is expressed as a rate, either liters per minute (L/min) or millilitres per kg bodyweight per minute (ml/kg/min). Because oxygen consumption is linearly related to energy expenditure, when we measure oxygen consumption, we are indirectly measuring an individual's maximal capacity to do work aerobically.

Why is his bigger than mine?

To rephrase, we might start by asking "what are the physiological determinants of VO2 max?" Every cell consumes oxygen in order to convert food energy to usable ATP for cellular work. However, it is muscle that has the greatest range in oxygen consumption. At rest, muscle uses little energy. However, muscle cells that are contracting have high demands for ATP. So it follows that they will consume more oxygen during exercise. The sum total of billions of cells throughout the body consuming oxygen, and generating carbon dioxide, can be measured at the breath using a combination of ventilation volume-measuring and O2/CO2-sensing equipment. The figure below (fig 2.1), borrowed from Prof. Frank Katch, summarizes this process of moving O2 to the muscle and delivering CO2 back to the lungs.

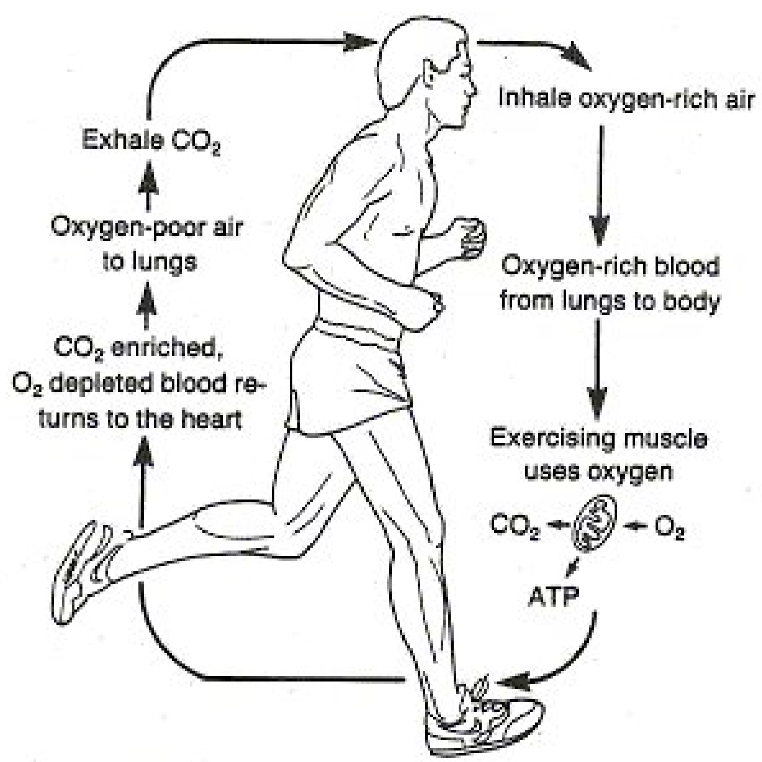


Figure 2.1 The pathways by which oxygen is transported from atmospheric air to the active muscles.

So, if we measure a greater consumption of oxygen during exercise, we know that the working muscle is working at a higher intensity. To receive this oxygen and use it to make ATP for muscle contraction, our muscle fibers are absolutely dependent on 2 things: 1) an external delivery system to bring oxygen from the atmosphere to the working muscle cells, and 2) mitochondria to carry out the process of aerobic energy transfer. Endurance athletes are characterized by both a very good cardiovascular system, and well developed oxidative capacity in their skeletal muscles. We need a big and efficient pump to deliver oxygen rich blood to the muscles, and we need mitochondria-rich muscles to use the oxygen and support high rates of exercise. Which variable is the limiting factor in VO2 max -- oxygen delivery or oxygen utilization? This is a central question that has created considerable debate among exercise physiologists over the years, but for most. the jury is now out.

In the well-trained, oxygen delivery limits VO2 max

Several experiments of different types support the concept that, **in trained individuals, it is oxygen delivery, not oxygen utilization** that limits VO2 max. By performing exercise with one leg and directly measuring muscle oxygen consumption of a small mass of muscle (using arterial catheterization) it has been shown that the capacity of skeletal muscle to use oxygen exceeds the heart's capacity for delivery. Thus although the average male has about 30 to 35 kg of muscle, only a portion of this muscle can be well perfused with blood at any one time. The heart can't deliver a high blood flow to all skeletal muscle, and still maintain adequate blood pressure. This limitation is analogous to the water pressure in your house. If you turn all the faucets on while trying to take a shower, the shower pressure will be inadequate because there is not enough driving pressure. Without getting in too deep on the hemodynamics, it seems that blood pressure is a centrally controlled variable; the body will not "open the valves" to more muscle than can be perfused without compromising central pressure, and blood flow to the brain. The bigger the pumping capacity of the heart, the more muscle can be perfused while maintaining all-important blood pressure.

As further evidence for a delivery limitation, long-term endurance training can result in a 300% increase in muscle oxidative capacity, but only about a 15 to 25% increase in VO2 max. VO2 max can be altered artificially by changing the oxygen concentration in the air. VO2 max also increases in previously untrained subjects before a change in skeletal muscle aerobic capacity occurs. All of these observations demonstrate that VO2 max can be dissociated from skeletal muscle characteristics.

Stroke volume, in contrast, is linearly related to VO2 max. Training results in an increase in stroke volume and therefore, an increase in maximal cardiac output. Greater capacity for oxygen delivery is the result. More muscle can be supplied with oxygen simultaneously while still maintaining necessary blood pressure levels.

In the untrained, skeletal muscle capacity can be limiting

Now, having convinced you that heart performance dictates VO2 max, it is important to also explain the contributing, or accepting, role of muscle oxidative capacity. Measured directly, Oxygen consumption = Cardiac output x arterial-venous oxygen difference (a-v O2 diff). As the oxygen rich blood passes through the capillary network of a working skeletal muscle, oxygen diffuses out of the capillaries and to the mitochondria (following the concentration gradient). The higher the oxygen consumption rate by the mitochondria, the greater the oxygen **extraction**, and the higher the a-v O2 difference at any given blood flow rate. Delivery is the limiting factor because even the best-trained muscle cannot use oxygen that isn't delivered. But, if the blood is delivered to muscles that are poorly trained for endurance, VO2 max will be lower despite a high delivery capacity. When we perform VO2 max tests on untrained persons, we often see that they stop at a time point in the test when their VO2 max seems to still be on the way up. The problem is that they just do not have the aerobic capacity in their working muscles and become fatigued locally prior to fully exploiting their cardiovascular capacity. In contrast, when we test athletes, they will usually show a nice flattening out of VO2 despite increasing intensity towards the end of the test. Heart rate peaks out, VO2 maxes out, and even though some of the best trained can hold out at VO2 max for several minutes, max is max and they eventually hit a wall due to the accumulation of protons and other changes at the muscular level that inhibit muscular force production and bring on exhaustion.

How is VO2 max measured?

In order to determine an athlete's true maximal aerobic capacity, exercise conditions must be created that maximally stress the blood delivery capacity of the heart.

A physical test that meets this requirement must:

• Employ at least 50% of the total muscle mass. Activities which meet this requirement include running, cycling, and rowing. The most common laboratory method is the treadmill running test. A motorized treadmill with variable speed and variable incline is employed.

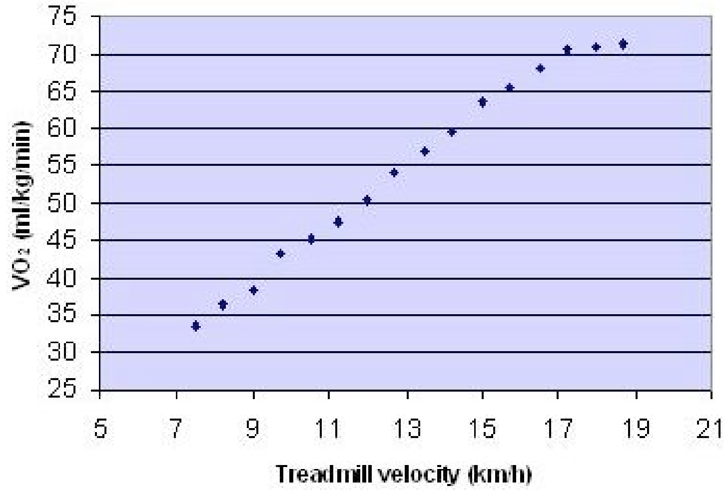
• Be independent of strength, speed, body size, and skill. The exception to this rule is specialized tests for swimmers, rowers, skaters, etc.

• Be of sufficient duration for cardiovascular responses to be maximized. Generally, maximal tests using continuous exercise protocols are completed in 6 to 12 minutes.

• Be performed by someone who is highly motivated! VO2 max tests are very tough, but they don't last too long.

If we use a treadmill test as an example, here is what will happen. You will go to a good laboratory at a University fitness program, performance testing lab, or hospital wellness center. After a medical exam, and after being hooked up to an ECG machine to monitor cardiac electrical activity, you might start the test by walking on the treadmill at low speed and zero grade. If your fitness level is quite high, the test might be initiated at a running speed. Then, depending on the exact protocol, speed or inclination (or both) of the treadmill will increase at regular intervals (30 sec to 2 minutes). While running, you will be breathing through a 2-way valve system. Air will come in from the room, but will be expired through sensors that measure both volume and oxygen concentration. Using these values and some math, your oxygen uptake will be calculated by a computer at each stage. With each increase in speed or incline, more muscle mass will be employed at a greater intensity. Oxygen consumption will increase linearly with increasing workload. However, at some point, an increase in intensity will not result in an "appropriate" increase in oxygen consumption. Ideally, the oxygen consumption will completely flatten out despite ever-increasing workload. This is the true indication of achieving VO2 max.

In the figure below, we see the results of actual test on a well trained runner performed in our lab with the treadmill incline a constant 5% and velocity increased 0.75km/h each minute. Even well trained athletes cannot stay at their VO2 max very long due to concurrent skeletal muscle fatigue. Other indications of max VO2 are extreme hyperventilation, and a heart rate of very near 220 minus age that does not increase further with increased workload.



The value you are given by the test administrator will be in one of two forms. The first is called your **absolute VO2 max**. This value will be in liters/min and will probably be between 3.0 and 6.0 liters/min if you're a man and it will be between 2.5 and 4.5 l/min if you're a woman. This absolute value does not take into account differences in body size, so a second way of expressing VO2 max is common. This is called your **relative VO2 max**. It will be expressed in milliliters per min per kg bodyweight (ml/min/kg). So if your absolute VO2 max was 4.0 liters/min and you weighed 75 kg, then your relative VO2 max would be 4000 divided by 75, or 53.3 ml/min/kg. In general, absolute VO2 max favors the large endurance athlete, while relative VO2 tends to be higher in smaller athletes. ***(see subcategory-(iii) below).***

For comparison, the average maximal oxygen consumption of an untrained male in his mid 30's is about 40-45 ml/min/kg, and decreases with age. The same person who undergoes a regular endurance exercise program might increase to 50-55 ml/min/kg. A champion male masters runner age 50 will probably have a value of over 60 ml/min/kg. An Olympic champion 10,000 meter runner will probably have a VO2 max over 80 ml/min/kg! **What about females?** ***(see subcategory-(iv) below).*** The underlying physiology is the same, however specific differences result in lower population values for VO2 max in untrained, trained and champion females when compared to men at a similar relative capacity.

Genetics play a big role

I grew up being told that I could do anything and be anything I set my mind to. I think that was nice of my mother to encourage me that way. However, the biological reality is that there is a significant genetic component to most of the underlying physical qualities that limit just how “Citius, altius, fortius” we can be with training. VO2 max is no exception. The reality is that if an adult male with a natural, untrained VO2 max of 45 ml/min/kg trains optimally for 5 years, they ***might*** see their VO2 max climb to around 60-65 ml/min/kg. This is a huge improvement. But, alas, the best runners have a VO2 max of 75 to 85 ml/kg. So our hard training normal guy is still going to come up way short against the likes of these aerobic beasts. If they were to stop training for a year, their VO2 max might fall to about where the average guy’s topped out after years of optimal training. How unfair is that? The bottom line is that Olympic champions are born with unique genetic potential that is transformed into performance capacity with years of hard training. Recent studies focusing on the genetics of exercise adaptation have also demonstrated that not only is our starting point genetically determined, but our adaptability to training (how much we improve) is also quite variable and genetically influenced. While the typical person will show a substantial increase in VO2 max with 6 months of exercise, carefully controlled research studies have shown that a small percentage of people will hardly show an increase in VO2 max at all.

One more thing. Just to put things in perspective, the VO2 max of a typical 500kg thoroughbred horse is about 75 liters/min or 150 ml/min/kg! So compared to a horse, even an Olympic endurance champion human comes out looking like a couch potato.

iii) The Impact of Body Dimensions on Endurance Performance

So you want to build a great endurance athlete? Well we know the heart is important, as well as the composition of the working skeletal muscles. What is the effect of the size of the athlete?

Form Follows Function: The "optimal" physical dimensions of an endurance athlete are critically dependent on the specific demands of the sport. What is the resistance that must be overcome? If it is gravity, such as in running or road cycling in the mountains, then a high aerobic capacity relative to bodyweight is most important. If the primary resistance is air (time trial cyclist) or water, then absolute aerobic capacity is most important because bodyweight is supported during the activity.

We will assume for now that skeletal muscle characteristics (i.e. lactate threshold) are identical.

Let's put some numbers to this. Start with an elite road cyclist: 5'7" (1.70m) bodyweight 140 lbs (63.6 kg), absolute VO2 max 5.0 liters/min, (79 ml/kg/min). If we create a **geometrically similar and qualitatively identical athlete** that is 12% taller, he will be 6'3" (1.9 m). His cross sections (bones, muscles, heart) will all be related as the square of 1.7 to the square of 1.9. Because of the increased heart size and, therefore, stroke volume, absolute VO2 max will increase 25% to 6.25 l/min. So, this taller version should be a faster road cyclist right? Wrong. His volume (weight) will increase as a cubed function of height. Therefore our rider will now weigh 195 pounds (88.6 kg). His relative VO2 max will therefore decrease 9% to 71 ml/min/kg. This is still quite high, but not high enough to win major road races. Our big rider will suffer trying to stay with his smaller version in the hills. However, if he learns to row, his absolute increase in aerobic capacity will serve him well because the penalty for carrying around the extra mass will be less severe (there will be some, due to the increased wetted surface area [drag] of his boat).

In fact, these two hypothetical athletes are representative of the physical dimensions and capacities of elite road cyclists and rowers respectively. The men's U.S Olympic rowing team (35 members) averaged 6 feet 4 inches (1.92m) and 194 lbs (89 kg) with an absolute VO2 of 6.25 l/min (data supplied by Fred Hagerman PhD at Ohio University). I do not have exact figures for road cyclists but typical elite class riders have VO2 max values in the mid to upper 70s. And, they are usually smallish, weighing in at 140-150 pounds.

There are some exceptions. Certainly 5 time Tour de France winner Miguel Indirain was one. He was big for a world class climber at ~6 feet tall and 170 lbs, yet he was a great mountain stage performer. Undoubtedly, one explanation for his dominance is that his heart size is disproportionately large relative to his physical dimensions, even for a well trained endurance athlete. That would help explain his resting heart rate of 28 bpm!

And, perhaps the best example of the difference between absolute and relative oxygen consumption comes from the well-known story of 6-time Tour winner Lance Armstrong. His pre-cancer body was more robust and muscular than his post cancer physique. However, he regained his aerobic capacity so that with about 5kg less body mass, his climbing capacity became much better in keeping with his higher weight adjusted maximal oxygen consumption (about 82 ml/kg/min).

iv) Gender Differences in Endurance Performance & Training

This article is long overdue and I apologize to those who were interested in the topic. To bring up the issue of gender differences in physical performance may suggest sexism, but that is not my intention. Historically, there is no doubt that sport has been a center of faulty assumptions and sexism where female athletes are concerned. Social issues, and misunderstanding about female physical and medical limitations (or the presumption of limitations) conspired to slow the development of female performance for many years (the marathon for women was only added to the Olympic schedule in 1984!), but those times are gone, at least among young athletes. Among masters athletes, we still see greatly reduced participation by the older female age groups. This participation difference will no doubt diminish over the next couple of decades. As a result, performances by the oldest females will probably improve more rapidly than those of the oldest males, as this new generation of well trained young female athletes moves into age-group competition, and are joined by more and more talented "late bloomers."

"Old" social norms and habits are still having negative consequences on participation and performance by older (50 +) females. Modern female athletes have repeatedly demonstrated these norms ("women are not built to run long distances" blah- blah-blah) are totally bogus. Currently, teenage daughters are encouraging their formally sedentary mothers and even grandmothers to take up exercise. This transfer of knowledge and norms UPSTREAM is the reverse of what we traditionally see in males (Dad teaching his boy all he knows). However, this is a transitional period for women in sport, so the knowledge transfer across generations is helping to speed the development of women's masters sport.

Having said all that, there ARE some physiological differences between the sexes that impact performance in females independent of age. Some years ago, when the marathon was first becoming a competitive event for women, the rapid improvement in female times led some to predict that female performances would soon equal those of men in the marathon. This has not happened, and it won't. The current world record for women is 2:21, compared to 2:06:50 for the men, a difference in speed of about 10%. This same 10% gap is present across the distance running performance spectrum The reason for the performance gap is not that women don't train as hard as men. There are some important physiological differences between the sexes that can't be overlooked or overcome. I want to point out the most important.

Where relevant, I will try to do so in terms of the BIG THREE Performance adaptations that I have discussed on the MAPP.

The Maximal Oxygen Consumption

The "typical" young untrained male will have an absolute VO2 max of 3.5 liters/min, while the typical same-age female will be about 2 liters/min. This is a 43% difference! Where does it come from? Well first, much of the difference is due to the fact that males are bigger, on average, than females. We humans are all (sort of) geometrically similar, so heart size scales in proportion to lean body size . If we divide VO2 by bodyweight, the difference is diminished (45 ml/min/kg vs 38 ml/min/kg) to 15 to 20%, but not eliminated. What is the source of this remaining difference?

If we compare average bodyfat in males and females, we find part of the answer. Young untrained women average about 25% bodyfat compared to 15% in young men. So, if we factor out body composition differences by dividing VO2 by lean body mass (Bodyweight minus estimated fat weight) the difference in maximal O2 consumption decreases to perhaps 7-10%. Keep in mind though that this is only a meaningful exercise on paper. *A female athlete cannot expect to improve her performance by reducing her bodyfat down to the sub 7% levels that are often observed in elite males. The health consequences for the female are too severe!*

To find an explanation for the remaining 10% difference we must go back to the key limitation on VO2 max, oxygen delivery. On average females have a lower blood hemoglobin content than males, up to 10% lower. Finally, there is some evidence, that the female heart is slightly smaller relative to body size than the male heart. Recent ECG and echocardiographic studies also suggest that the young female heart exhibits less enlargement in response to either endurance or resistance training than the male heart (George et al, 1995) This may be due to differences in androgen receptor density in the female heart. A smaller heart would be expected to be a less effective pump.

Slightly lower oxygen carrying capacity of the blood (lower hemoglobin levels) plus a somewhat smaller or less adaptive heart are sufficient to account for the gender differences in maximal oxygen consumption that are independent of body size and fat percentage.

It is worth noting here the results of a 1993 study by Spina et al. Their data suggested that in previously sedentary older men and women (60 to 65 years old) who trained for 9 months to a year, both men and women increased their VO2 max by the same amount (an average of 20%). **However, the mechanism of improvement was different.** The men improved primarily by increasing maximal cardiac output due to higher stroke volume. This is just the pattern of response I have previously described ***(see subcategory-(ii) above).*** However, the older women did not demonstrate any increase in cardiac performance, but rather increased oxygen consumption by improving oxygen extraction by the working muscles, due to greater capillarization and more mitochondria. This data supports previous studies in 60+ year old women that show no cardiac hypertrophy in response to endurance training.

To summarize, their is a growing body of data suggesting that females demonstrate a somewhat different pattern of cardiac adaptation to exercise, which may become more dissimilar with age. They also generally have a lower hemoglobin level by several percent. The net effect is a small but significant difference in maximal oxygen consumption, even among similarly trained males and females, and after scaling for differences in size and body composition.

It is important to make note of the fact that these differences are "on average". In reality, there are **many** women with significantly higher VO2 max values than average men. However, if we look at the "best of the best", the differences persist. Using XC skiing as an example from here in Norway, the highest reliable values for VO2 max recorded in national team XC skiers are about 90 ml/min/kg. The very best Norwegian woman has been measured at 77 ml/min/kg, a 17% difference. So, while this woman will outperform 99.9% of all men, she will not out-perform the national team level males.

The Lactate Threshold

Now we come to the second component of endurance performance, the lactate threshold. As a review, this is the exercise intensity at which lactic acid begins to accumulate in the blood stream at levels significantly above "baseline" values. This intensity sets a (slightly fuzzy) boundary between that exercise intensity which can be sustained for long periods (over one hour) versus those which lead to fatigue in minutes. We have already discussed the fact that changes in the lactate threshold are due to adaptations that occur in the exercising muscle. We call these *peripheral* adaptations (Changes in cardiovascular performance are called *central adaptations*).

The question here is, do women demonstrate a different pattern or capacity for peripheral adaptations then men? As best as I can tell, the answer is NO.

First, Female skeletal muscle is not distinguishable from male skeletal muscle. Second, within some margin of error, the fiber type distribution (percentage of slow versus fast fibers) is not different in the male and female population. Third, male and female skeletal muscle responds similarly to endurance exercise. Finally, elite female endurance athletes have similar lactate threshold values compared to men when expressed as a percentage of their VO2 max. Elite women perform at the same high percentage of their maximal oxygen consumption as their male counterparts.

Some years ago it was proposed by some that women would actually perform **better** at ultra-endurance type activities. This theory has been disproved both in the laboratory and in practice as a performance difference persists in the ultramarathon events. Some of you may balk and recall a recent Running Times article that suggested women had an edge in the really long events. They discussed a study in which a group of male and female runners who were matched for marathon time were raced head to head in the Comrades marathon, a 90k race. The women won by 54 minutes, suggesting a female edge in longer events. The problem with this study is that when you match men and women for performance, the women are relatively better runners and probably have a higher slow twitch fiber percentage. This advantage becomes bigger in an ultradistance event.

The fact remains that the performance gap between male and female record holders in the really long running races 50k to 6 days is actually more on the order of 15 to 20%, instead of the 10% difference for the standard distances. Part of this larger gap may be to lower participation, and the fact that the most talented females have not yet tested themselves over the ultradistances. But at elite level, I don't think the gap will disappear.

Efficiency

The third component of endurance performance is **efficiency** which of course has different constraints, depending on the sport. The research information comparing the efficiency of female and male athletes is both sparse and inconclusive. In running, for example females have been found to be more, less, and equally efficient compared to males depending upon the specific study. Some of this confusion comes down to how the differences in bodyweight and bodyfat were accounted for.

After looking over some of the research comparing running economy between genders, I started to go into a couple of studies, but it all starts to become a scaling and factoring game, which I like, but you probably can do without it. So, I decided to just summarize things this way. Currently, I would argue that any inherent economy differences in male and female runners are smaller than the individual variation in running economy that is observed among runners, independent of gender. I would support that argument by suggesting that the differences in VO2 max observed between elite males and females are sufficient to explain the "10% gap" without other factors being involved. If more data comes to my attention to dispute this, I will share it.

Now, if we look at efficiency/economy differences in other sports, things mostly boil down to body shape/anthropometric differences. In situations like running or cycling, these may actually favor females in general, due to narrower upper bodies for a given total body mass, and potentially less wind or water drag. As I have shown for rowing , differences in VO2 max alone are sufficient to explain the gender performance gap in rowing. I am not aware of any research studies to support or dispute this, but it seems that there are no differences in rowing efficiency among male and female rowers of similar relative ability.

Fat metabolism differences?

Back in the 70s, a theory got started that said "Since women have more fat stores, they will be better at utilizing fat during endurance performance when glycogen stores are depleted." One of the supporting pillars of the theory was that it had been noticed by one female runner/author how "fresh" many female runners looked as they crossed the finish line! Well, this shaky theory was crushed under the harsh light of science. Back in 1979, Costill and colleagues compared males and females who were equally trained during a 60 minute treadmill run. There were no differences in any measures of fat metabolism. These guys even took some muscle out of the runners' legs and tested it in a test tube. Still no difference! This is an often repeated finding among similarly trained males and females. There is no gender difference in the ability of men and women to burn fat!

Are Men Sweatier than Women?

On an absolute basis, and per kg bodyweight, women have lower sweat rates than men. However, because of their higher body surface area to volume ratio, they dissipate heat equally well. Men have an advantage in evaporative cooling, but women have an advantage in radiant cooling, so they come out even.

Summary So Far

Of the three critical components of endurance performance, the only one that is clearly and consistently depressed in females is the maximal oxygen consumption. Even after accounting for differences in bodyweight and body fat percentage, a gap of roughly 10 - 15% remains. Now I want to talk about some other comparisons that don't fit so cleanly into the Performance Model for endurance performance.

Muscle Strength and Power

Although maximal muscular strength and anaerobic power has little to do with pure endurance performance, there are many events which can be classified as "power-endurance" events. These events ranging from 2 to about 8 minutes require some combination of aerobic and anaerobic capacity. For this reason, I think it is important to also consider this "anaerobic" component of the performance package. When we talk about anaerobic capacity, the critical determinant is muscle mass. Females, on average, have less total muscle mass than males. As a result, maximal strength measures as well as maximal power measures (power = force/time) are reduced. Gross measures of upper body strength suggest an average 40-50% difference between the sexes, compared to a 30% difference in lower body strength. What about power? Maud and Schultz compared 52 men and 50 women, all about 21 years old using a maximal power test on a bicycle ergometer. Peak power was about 60% lower for the females when comparing absolute values. But, the men were heavier. Peak power per kg bodyweight was more similar, 9.3 watts/kg vs 7.9 watts/kg for the women, an 18% difference. Finally, when power outputs were adjusted for fat-free mass, the values were 10.4 watts/kg and 9.9 respectively. This 5% difference was not statistically different. Numerous other studies using different techniques have demonstrated that when you just look at muscle **quality**, male and female muscle is not different. Within the accuracy of current comparative techniques, it appears that the strength and power differences between the sexes are a function of muscle **quantity** only. Biomechanical differences probably play a role in some situations, but this will be very sport specific.

Should Men and Women Train Together?

OK, now we move onto something a little different. I think there are two reasons for making this gender comparison. First, I think it is useful to understand that at the elite levels, male and female performance differences are physiological in origin, not a function of differences in training, desire etc. (The one caveat to this is among the oldest athletes. Here, I think the gender performance gap is probably still wider than it will ultimately be, due to differences in participation and training intensity among the oldest age groups.) The second reason is a very practical one. Men and women live together, work together, and often train together, either as husband and wife, as friends, or as part of a training group. So, if we are going to train together, I think it is pretty important that we understand each other as athletes. Athletes are not just bodies. They have brains too! No, really, they do.

Psychological Differences?

I have examined briefly some physiological gender differences. Now I want to move into the psychological realm. Oh boy, now I am really treading on thin ice, but I'm safely and happily married, and living pretty far away from most everyone I know here in Norway, so I am going to proceed. **As a broad generalization**, here are a few things I have noticed, read, experienced etc. that I think are important regarding males and females training together. Again, let me repeat. THESE ARE GENERALIZATIONS. For every point I will make here, I myself have seen just the opposite behavior on occasion.

The Numbers Game

If you walk into a fitness center, teeming with men and women huffing and puffing on all manner of computerized exercise machines, take a look at the men first. As a rule, they will be staring directly at the computer screen, calculating, extrapolating, comparing the numbers with their previous efforts, or with measured glances, to the guy on the next machine. **Males are number guys.**

Now take a look at the women. In my experience, most are using one of several dissociation strategies. They are either listening to music, reading a book or magazine, or simply covering the entire computer screen with a towel. Some use all three methods in combination. The bottom line, is they are NOT paying attention to all the blinking lights! In fact, when I have had the nerve to inquire about this, most tell me that they hate all the numbers, clocks, bells, and whistles.

Now, most of these folk aren't athletes, but I think the tendency remains among the competitive set. On the sports lists that I lurk on via email, and the messages I get from you guys, it is mostly the men that are getting caught up with heart rate, time, power output etc. Men seem to need to **quantify** their training in as much detail as possible. Have you noticed how men are more likely to keep training logs then the women? Meanwhile, I would argue that women are more sensitive to qualitative, internal, measures of training effectiveness. Which method is better? Neither. We can definitely learn from each other. Sometimes the numbers are helpful for getting us over specific hurdles. They also help us to see small changes in performance and evaluate the effectiveness of our training. On the flip side, a more qualitative approach helps to take some of the internal pressure off sometimes. If the odometer, speedometer, or HR monitor rules our heads, then we men often find ourselves "competing" every workout. This is a sure-fire prescription for become stale and overtrained.

The Sociology of the Training Group

I have not read "Men are from Mars, Women are from Venus", but I have read some similar stuff. Basically, I have to go along with the idea that men and women communicate differently. Men tend to be more hierarchical, while the women develop better horizontal lines of communication. What the heck does this have to do with performance? This is my slant. Men who were active in sports as youth are very familiar with the pecking order mentality. In sports, some get picked first, others get picked last. You have first team and second team etc. "It's not personal. He is just a little faster than you Charlie." Watch a bunch of guys in competitive practice situation. My most recent personal experience is in rowing. On the water, we would do daily battle against each other in our singles. Sweating, grimacing, taunting, yelling, winning, losing. Then we get to the dock, get out of the boat and say "Great workout. See ya tomorrow." On the water each day a hierarchy was established and defended, then dissolved as soon as practice was over. Another example came to me from a coxwain for the men's lightweight national team who was now coaching collegiate rowers. He made the point that the national team athletes had the ability to turn everything on in practice. Then, as soon as practice was over, they forgot about it. No internalization, no dwelling on successes or failures during times when nothing can be done about them!

In my experience, this separation of competition within and outside of the training environment CAN be more difficult among female athletes. The same qualities that often make them more effective communicators and empathizers, also can lead to personalizing the physical battles of daily training. In the extreme it can splinter a team. I have observed it (from a safe distance) in rowing among masters women. Most of those women were not competitive athletes in their youth. Perhaps this made a difference. Battles for seats in the boat waged on the water and coaching decisions that resulted were not forgotten or accepted when practice was over. The women didn't seem to know how to communicate under these new conditions. Disaster!

Competitive training situations are generally good, I think. It helps to train with others who share your goals. However, everyone has their own optimal amount of competitive stimulation. Even among world class athletes, some thrive in an aggressive team environment, and others don't. Here in Norway, I know the coach of last year's top female XC skier in Norway, Marit Mickelsplass. She rose to new levels on the international circuit (Top 3 in the world) this year after leaving the national team, and training on her own. The problem was that the stress of daily training in the aggressive team atmosphere was too much **for her**. Psychological stress led to physical stress and overtraining. Thanks to a good coach who understood the link between psychology and physiology, the problem was solved. I guess my view is that there are often going to be subtle differences in the approach that a coach is going to need to take with female athletes versus male athletes. Failure to understand these differences can impact performance.

Can Women and Men handle the same training Volume?

Here, we return to some physiology. Talking with elite level coaches leads me to believe that there are small but important differences in the recovery capacity of male and females, at least when pushed to the extremes of elite level training. Again I will go to evidence from world class XC skiers here in Norway. It appears that the best women perform optimally at a training volume that is perhaps 10 -15% lower than that observed in the best men. Increasing the volume in the women does not improve results, and often leads to overtraining. The general consensus is that the difference lies in the higher average testosterone levels of males. Remember, testosterone is an anabolic hormone. This means it is critical for tissue growth and repair. Anecdotally, I have been told that only one of the Norwegian female national team skiers has been able to maintain the average yearly training volume (measured in hours) that is maintained by the entire Russian female team.

The difference appears to be steroid use, but of course this is only a rumor. At any rate, I think we should be aware that there is probably a small gender difference in recovery capacity from hard or high volume training, in addition to the individual variation that is observed.

You and your Significant Other as training partners

I married a woman who loves exercise. Heck, that is one of the reasons I was so attracted to her! We focus on different sports, but sometimes we workout together, either running, cycling or XC skiing, depending on the season here in Norway. What we learned pretty quickly was that we couldn't do the same type of workout together effectively. If we run at the same speed, I can be comfortable just under my lactate threshold. Meanwhile she is teetering on the edge of disaster as she runs at or above hers. I have a good run, she is miserable. The same thing happens on a bicycle. What is the solution? Well one is we can just not ever train together. Neither one of us like that idea. So we compromise. Sometimes I train alone. These workouts are usually hard interval sessions or lactate threshold workouts. Then, when we train together, I am running or cycling at a good steady state aerobic pace, and she is doing a tempo run or lactate threshold session on the bike. The bottom line is that we had to understand where we were both at physiologically and make the adjustments necessary to allow us both to profit from our joint training sessions, and not become frustrated with each other.

Even small differences in the performance capacity of you and your partner can be problematic if they are not recognized. The slower partner who always works a little harder to keep up can be at risk of overtraining, or just not achieving the goals of the workout. This may be the man, or the woman. Either way, it can be avoided by taking the time to evaluate the performance difference and make adjustments in the training schedule. One thing is likely. You probably should not train together all the time. Find time for common workouts, but make sure that there remain training sessions where there are no compromises being made. If this means training alone, then do it. *It doesn't mean I don't enjoy your company, dear!*

Final Words

OK, I think that pretty well hits the main points, from my current vantage point. The bottom line is that there IS a physiological explanation for the gender performance gap observed in endurance (and power) sports. Keep in mind that the best women can still beat 99+% of the men. However, if you ask me

"When will women run as fast as men." I will answer, "just as soon as they have the same VO2 max as men. Grete Waitz probably said it better, *"As long as women are women, I don't think they will surpass men."*

I have read at least one very good and quite popular running book, "The Lore of Running" that has tried to explain the gender performance differences in terms of some unmeasured but imagined difference in muscle quality. He has been forced to assume this angle, despite absolutely contrary data, because of his even more unsupported theory that VO2 max is actually not limited at all by cardiac performance. To be honest, this view takes about as much denial of the available data as that of the tobacco industry denying that smoking is bad for you! Enjoy this otherwise wonderful book, but don't read the physiology chapters.

I realize I have skipped over a tremendous area of difference related to the impact of the menstrual cycle and pregnancy on training on performance in females. However, I think there are a lot of excellent resources by much more qualified people available for women athletes with questions on this topic.

As for the presumed psychological differences, I think they are real, but I realize that there are many exceptions. So, please don't flood my mailbox with white hot flame-mail! At least my wife still loves me.

b) Myocardial Adaptations to Training

The heart, in cellular composition, structure, and mechanics, is an absolute marvel of "biological engineering". Even among human couch potatoes, it is an astoundingly well equipped endurance muscle. It has an incredibly dense network of capillaries (over 2000 capillaries per cubic millimeter!) designed to provide reliable delivery of oxygen to the working muscle with a minimum diffusion distance to intracellular mitochondria. The individual heart cells (myocytes) are densely packed with mitochondria. About 25-30% of the human heart cell volume consists of mitochondria. In contrast, mitochondria make up less than 5% of the untrained skeletal muscle cell volume. The specific biochemistry of the muscle cells is designed to minimize lactate production even at very high workloads (H isoform of lactate dehydrogenase for you scientists). The heart can metabolize fat, lactate, and blood glucose with equal effectiveness.

So, how can endurance training improve a muscle that is already superbly designed and equipped to perform constant work? The answer is fairly simple. **IT GETS BIGGER! (OK, it's slightly more complicated than that. *See subcategory-(iii) below***). Endurance trained hearts do not beat faster at maximum. They do not beat more powerfully, gram for gram. They also do not change significantly in terms of mitochondrial or capillary density. The distinction between the athlete's heart and the sendentary heart is the **larger stroke volume of the trained heart**. This improvement is critical to improved endurance performance. Why? The heart is first and foremost a pump. It pumps oxygenated blood to the body to support the production of cellular energy. During exercise, working muscles increase their cellular energy requirements up to 100X. Generating more energy (ATP) requires more oxygen delivery to the mitochondria.

The quantity of work that can be performed by the muscles is critically dependent on the volume of blood that can be delivered by the heart. A body supplied more oxygen by a bigger pump has the potential to sustain work at a greater maximal intensity. Maximal Cardiac Output = Maximal Heart Rate x Stroke Volume. **Stroke volume is the volume of blood ejected from the left ventricle each beat.** Endurance training impacts myocardial function 1) at rest, 2) during submaximal exercise, and 3) during maximal exercise.

Resting Hemodynamics and Exercise

At rest the stroke volume and resting heart rate of the average person can be remembered easily as approximately 70 ml/beat and 70 beats/minute. This gives us 70x70 or about 5 liters/minute resting cardiac output. The resting cardiac output is determined by the oxygen demand at rest, and also by the need for high blood flow to the kidneys for filtration purposes. It doesn't change appreciably with endurance training. However, the manner in which the heart delivers this resting demand does change. After 6 months of endurance training, the resting heart rate may decrease to 55 bpm. At the same time, resting stroke volume increases to about 90 ml (HR x SV stays the ~same before and after training). So a reduced resting heart rate is a hallmark of endurance training. Resting heart rate (RHR) can be much lower. In champion endurance athletes, RHR is often in the 30s and low 40s. Since resting oxygen demand still hasn't changed, this should tip you off that these athletes have extremely high resting stroke volumes! Thus, the resting heart of the athlete is more efficient. It performs the same work with fewer beats and less myocaridal energy demand. However, since some medical symptoms are also marked by a reduced resting heart rate, your physician may initially raise his/her eyebrow to your low frequency lub-dub during checkups.

Myocardial Responses to Submaximal Exercise Before & After Training

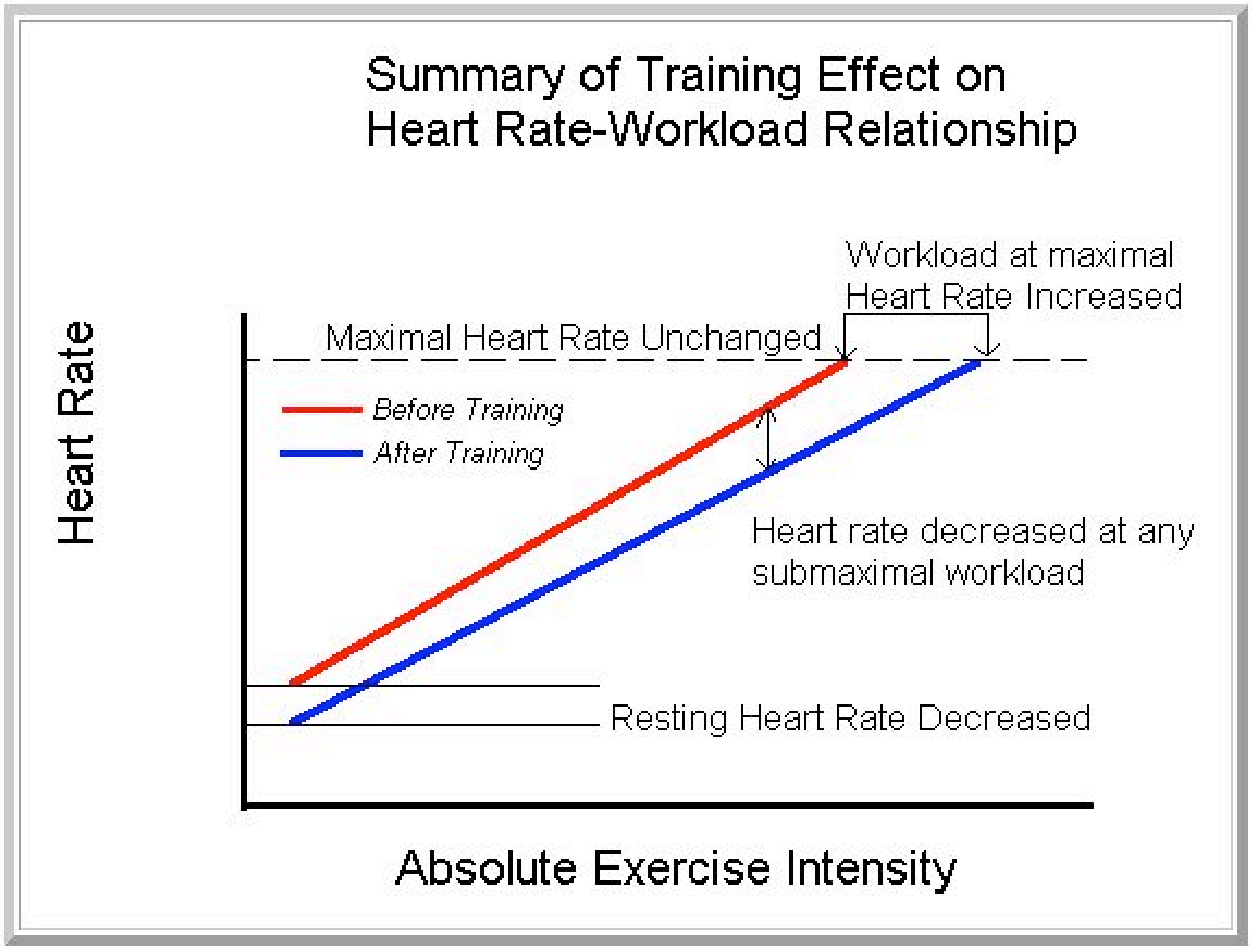
When we begin to exercise at any given intensity, more oxygen must be delivered to the working muscle. Cardiac output increases in proportion to the increased energy demand. If we measure the responses of an individual to running at 8 min mile pace before and after 3 months of regular exercise, here is what we will see. First, the metabolic cost of working at this intensity will be unchanged (assuming no improvement in running efficiency). Therefore cardiac output will be the same. However, just as during rest, the heart will deliver more blood each beat. Therefore heart rate at this and any submaximal exercise intensity will be reduced. If we use the analogy of a car engine. We have replaced a small motor with a larger one that achieves the same horsepower at lower rpms.

Hemodynamic Response to Maximal Exercise

There is for all of us an exercise intensity that will elicit our maximum cardiac output. Once this limit is achieved, further increases in work intensity will result in no further increase in heart rate. By definition, this is then the maximum heart rate. **The maximum heart rate in humans varies from individual to individual and decreases with age.** ***(see subcategory-(c) below).*** Therefore the only way to know precisely what a specific person's maximal heart rate is would be to do a maximal exercise test. Without such precise knowledge, we often use the formula "220 minus age" to approximate maximal heart rate. This formula will generally give results within plus or minus 10 bpm of reality. True maximal heart rate may not be achieved in some forms of exercise that do not employ a large enough muscle mass, or if the person is unfamiliar with the mode of exercise employed. For example, one person may have a true maximum heart rate of 195 achieved during uphill running, but only 191 during a cycling test, and 187 during swimming. These latter heart rates are termed peak heart rates and should be used as a basis for determining training intensity for a specific exercise mode.

The important thing to remember is: ***Maximal heart rate does not increase after training. It stays the same (or might even decrease just slightly). However, maximal stroke volume increases. Therefore maximal Cardiac***

Output increases in response to exercise. This is the primary reason for the increase in VO2 max!



So, in response to endurance exercise the heart adapts by increasing stroke volume at rest, during submaximal exercise, and during maximal exercise. There is some debate regarding whether stroke volume increases BECAUSE heart rate is decreased (increasing diastolic filling time), or because of an increase in ventricular volume due to eccentric hypertrophy of the heart muscle. Both factors probably contribute based on the available data. Both changes also rapidly revert towards normal with the cessation of training. One other important change that takes place is an **increased blood volume**. Increased blood volume helps to take advantage of the increased filling capacity of the heart and facilitates increased stroke volume. This adaption occurs fairly rapidly with training, but is also the first adaptation lost if we stop training for several days!

c) Aging and Cardiovascular Function

World records in endurance sports are not accomplished at age 55. Why? Because one of the unavoidable consequences of aging is a decline in the maximal capacity of the cardiovascular system to pump blood and deliver oxygen while removing metabolic waste products. The components of cardiovascular pump performance are 1) the maximal heart rate that can be achieved, 2) The size and contractility of the heart muscle and 3) The compliance (stiffness) of the arterial tree. We will look briefly at what is known about aging effects on each of these variables.

Maximal Heart Rate

Young children generally have a maximal heart rate approaching 220 beats per minute. This maximal rate falls throughout life. By age 60 maximal heart rate in a group of 100 men will average about 160 beats per minute. This fall in heart rate seems to be a linear process so that maximal heart rate can be estimated by the formula **220 - AGE**. This is an ESTIMATE, however. If we actually measure the maximal heart rates of those same 100 men during a maximal exercise test we would probably see a range of heart rates between 140 and 180. There is no strong evidence to suggest that training influences the decline in maximal heart rate. This reduction appears to be due to alterations in the cardiac electrical conduction system (SA node and Bundle of His), as well as down regulation of beta-1 receptors, which decreases the heart's sensitivity to catecholamine stimulation.

Maximal Stroke Volume

The research picture regarding age effects om maximal stroke volume is far less clear. This is in part due to the technical challenges involved in making these measurements. Studies showing a decline, an increase, and no change can be found in the literature. It appears that if middle-aged and older adults continue to train intensely, stroke volume is well maintained. Heart size in older athletes has been shown to be similar to that of young athletes, and bigger than their sedentary, same-aged peers. Ultimately, maximal stroke volume appears to decrease due to a 1) decrease in training volume and 2) an increase in peripheral resistance.

The Peripheral Resistance

The blood pumped out of the heart enters the systemic arterial system. In our youth, this system of arteries is quite flexible or compliant. This is important for the performance of the heart. Compliant vessel walls stretch when blood is pumped through them, lowering the resistance that the heart must overcome to eject a volume of blood each beat. As we age, these vessels lose their elasticity. Consequently, resting blood pressure and blood pressure during exercise slowly increase as we age. Continued training appears to reduce this aging effect, but does not eliminate it. Increased peripheral resistance results in a decrease in maximal blood flow to working muscles. However, at submaximal exercise intensities, the 10-15% decrease in blood flow is compensated for by increased oxygen extraction (a-v O2 difference). This compensation is probably possible due to the increased transit time of the blood through the capillary tree.

The Big Picture

In the sedentary population, cardiovascular performance declines progressively with age. However, much of this decline is due to 1) physical inactivity and 2) increased body weight (fat). Maximal oxygen consumption declines about 10% per decade after age 25 ***(see subcategory-(ii) above).*** However, if body composition is maintained and physical activity levels are kept constant, the decline in VO2 max due to aging is only about 5% per decade. Prior to age 50, this decline may even be less, perhaps 1-2% per decade in hard training masters athletes. Ultimately, cardiovascular capacity is reduced however, due to the unavoidable decline in maximal heart rate.

d) Understanding Heart Rate and Exercise

If you are reading the MAPP, chances are you already use heart rate as a measure of your exercise intensity. The basics are that 1) heart rate serves as a measure of exercise intensity during steady state activities, and 2) If we estimate maximal heart rate as 220 minus age we can use this value to gauge the intensity of our training. That is not the whole story though. Here are a few details that can be important in your training and racing.

• **The "220 minus Age" formula is Only an Estimate.** Your actual maximal heart rate in a given activity could be 10 to even 20 beats higher or lower than the estimated value. This has important implications for judging training intensity.

• **Your Maximal Heart Rate Differs in Different Activities.** Cardiac hemodynamics and maximal sympathetic drive are influenced by 1) body position during exercise and 2) muscle mass involvement. So, a triathlete with a max heart rate during running of 180, may only hit 176 on the bike, and 171 during swimming. In this case we call the running heart rate "Maximal Heart Rate" and the highest heart rate observed in cycling and swimming, "Peak" heart rate, for that event. Knowing your peak heart rate for each discipline will help you to more accurately gauge the intensity of your training. If the activity is restricted to upper-body muscle mass, peak heart rate will generally be considerably lower than in whole body activities. Examples include kayaking and double poling during cross-country skiing. Highly trained athletes can achieve a higher percentage of true max heart rate when performing small muscle mass activities.

• **A Better Method for Gauging Exercise Intensity with Heart Rate.** For a given exercise mode, heart rate will increase linearly with exercise intensity, and therefore, oxygen consumption. However, the resting heart rate creates an offset between % of HR max and the associated % of "peak" oxygen consumption for that activity. For example, running at 65% of heart rate max corresponds to approximately 50% of VO2 max. At 87% of HR max, you are at about 77- 83% of VO2 max. Depending on your resting heart rate, heart rate and VO2 percentage finally converge at 100%. I prefer to use HEART RATE RESERVE as my training intensity guide. To do this I need to know 1) my resting heart rate, and 2) my peak heart rate for that specific activity. The first one is easy to determine. The second one may sometimes be a slight estimate. My current resting heart rate is about 36 beats/min. My peak heart rate during rowing is about 181. So my heart rate range is 181-36 or 145 beats. Now, if I want to train at 85% of my peak VO2 for rowing, I will take 85% of my heart rate reserve (0.85 x 145 = 123) and add it to my resting heart rate (123 + 36 = 159). PERCENTAGE HEART RATE RESERVE will give a better approximation of % maximal oxygen consumption than just % max heart rate. And, it is more accurate because you can adjust for changes in your resting heart rate.

• **Body Position on the Bike will Influence Heart Rate.** Let's say I am riding on an indoor bicycle trainer with my upper body parallel to the ground (Hands on the drops) at a heart rate of 145. Raising upright while continuing to cycle at the exact same workload will result in an increase in heart rate of about 5 beats per minute. Trust me I have experimented with this effect on many a winter evening! This is due to decreased venous return in the more upright position. Heart rate increases to compensate for the slightly decreased venous return and stroke volume, keeping cardiac output constant. When I return to the drops, the heart rate drops again.

• **Temperature Will Greatly Influence Heart Rate.** Above about 70 degrees Farenheight (21C), Heart rate at a standard submaximal intensity will be increased about 1 beat/min per degree F increase in temperature. Thus, a steady state run at a heart rate of 150 on a 70 degree Spring day, may have you close to maximal heart rate on a scorching 95 degree day in July, if you try to maintain the same speed. I am from Texas, so I remember these days well. The oxygen demand doesn't increase in the heat, but the thermal stress load does. As a result, your cardiovascular system must divert blood flow to the skin to enhance heat dissapation. Since you only have so much cardiac output, this means a lower maximal steady state speed in the heat, or early exhaustion. You choose. My choice is generally to avoid running in 95 degree heat.

• **Humidity Hurts Too for the Same Reasons**. A higher relative humidity will increase heart rate at a submaximal workload. Increased humidity decreases the evaportation rate of sweat. This means the body has to resort more to heat removal via increased skin blood flow. Data from Wilmore and Costill *"Physiology of Sport and Exercise"* shows a 10 beat increase in heart rate from 165 to 175 when running in 90% humidity compared to 50%. This is the difference between a morning and afternoon workout in many parts of the country.

• **What about the Time of Day?** Our bodies show diurnal (time of day) variations in many physiological responses. Within the normal range of times that you might be training, this can result in a 3-8 bpm difference in heart rate at rest, during moderate exercise, and during recovery. The differences during maximal exercise are probably smaller. Data demonstrating this effect is in the literature. However, I suspect the exact pattern of these changes can be altered by your specific exercise pattern. For example, after several years of rowing before sunrise, I am sure my diurnal response pattern was modified. I have no data to support this assumption, but I do know that I was transformed from an afternoon exerciser, to a morning guy! So, my best guess is that you should not be too surprised by small differences in heart rate response if you do your training at an unusual (for you) time of day.

• **What is Cardiovascular Drift?** If you begin a 90 minute steady state ride on your bicycle trainer at a controlled intensity, your heart rate may be 145 after 10 minutes. However, as you ride and check your heart rate every 10 minutes, you will notice a slight upward "drift". By 90 minutes, your heart rate may be 160. Why is this happening if intensity is held constant? There are two explanations. As you exercise, you sweat (dah). A portion of this lost fluid volume comes from the plasma volume. This decrease in plasma volume will diminish venous return and stroke volume. Heart rate again increases to compensate and maintain constant cardiac output. Maintaining high fluid consumption before and during the ride will help to minimize this cardiovascular drift, by replacing fluid volume.

There is also a second reason for the drift during an exhaustive exercise session. Your heart rate is controlled in large part by the "Relative" intensity of work by the muscles. So in a long hard ride, some of your motor units fatigue due to glycogen depletion. Your brain compensates by recruiting more motor units to perform the same absolute workload. There is a parallel increase in heart rate. Consequently, a ride that began at heart rate 150, can end up with you exhausted and at a heart rate of 175, 2 hours later, even if speed never changed!