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## Physiology of Exercise Medicine

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*Professionals are known for their specialized knowledge.*

Professionalism is growing steadily in the healthcare professions (1). As exercise medicine becomes popular with the public sector, society will want to be led by credible professionals. The idea of fitness instructors applying the scientific aspects of exercise physiology is scary. For the most part, it is safe to say that they are likely to know little to nothing about the musculoskeletal and psychophysiology of exercise. In fact, it is more than reasonable to conclude that most exercise medicine lessons and concepts cannot be taught while lifting weights or jogging.

But don't take my word for it. Recently, I asked an emerging leader in the personal training field about oxygen consumption, fitness, and training. Here is what he had to say about oxygen consumption. "Consuming oxygen is like using gas in a car. The faster you go the more oxygen you need." I said, "Well yes, but not all cars are built to drive fast. If something fails to work, can you take the motor apart (meaning, can you identify the physiological problem)?" Then, I asked, "Is it a requirement to drive fast to keep the car running good? He said, "Yes, that is why my clients are pushed to go all out in their sessions with me." I said, "Well, it is important to consider the shape of the car, I mean rather...the shape that the client is in, right?"

"You do know how to calculate  $VO_2$  -- right?" The trainer said, "I don't need to know anything about  $VO_2$ , and I certainly don't need to calculate it." "I said, " $VO_2$  is oxygen consumption. It is important because it is the physiological product of cardiac output and arteriovenous oxygen difference (i.e., a- $vO_2$  diff)." The trainer looked at me without saying anything. His expression was one of confusion. After a few breaths he said, "I know the heart pumps blood to the body, but what is the 'difference' thing"? I said, "The difference

between the oxygen in the arterial blood and the venous blood is how much oxygen that is consumed by the muscles."

For example, at rest an average  $\text{VO}_2$  for a 70 kg (i.e., 154 lb) person is  $\sim 250 \text{ mL} \cdot \text{min}^{-1}$  or  $.25 \text{ L} \cdot \text{min}^{-1}$ . That is the product of a cardiac output of  $5 \text{ L} \cdot \text{min}^{-1}$  times a tissue extraction (a-vO<sub>2</sub> diff) of 50 mL of  $\text{O}_2 \cdot \text{L}^{-1}$  of blood. I looked at the trainer and said, "You understand, right?" He said, "No, I don't know what you are talking about! Do I actually need to know that?" Yes, you need to know it and much more! For example, you need to know that ventilation increases with an increase in the intensity of exercise, and that it is the product of tidal volume ( $T_V$ ) and frequency of breaths (Fb). You need to know the difference between  $V_E$  and alveolar ventilation ( $V_A$ ), and the importance of a large  $T_V$  versus Fb.

This idea is similar to the efficiency of cardiac output, especially since it should be produced primarily by stroke volume (SV) rather than heart rate (HR). The higher the SV for a steady-state exercise cardiac output the lower the HR. This means that the heart is more efficient in its delivery of oxygen to the peripheral tissues. In fact, this point is supported by the double product equation, which is an excellent estimate of the work of the heart:  $\text{DP} = \text{HR} \times \text{SBP} \times .01$

In fact, as an example of the importance of DP, assume that SBP is unchanged at a submaximal steady-state, but HR is lower after 2 months of training. This means DP is lower and the heart is not working as hard as it did before training. The decreased work of the heart means that it requires less oxygen, which is understandable since myocardial oxygen consumption ( $\text{MVO}_2$ ) is correlated with DP. By lowering DP, the heart's need for oxygen is decreased:  $\text{MVO}_2 = .14(\text{DP}) - 6.3$

Similarly, if a client's  $T_V$  is high either at rest or during exercise, the lower the Fb for a given  $V_E$ . Thus, in both instances the lungs and the heart are more efficient in bringing in oxygen and in transporting oxygen to the skeletal muscles. After taking a breath, it occurred to me I was talking to myself. With the next breath I asked the trainer if he understood the relationship of the heart to a person's body size. He looked at me as though I had lost my mind. He said, "Is there actually a relationship between a person's body size and the heart!" I said, "Yes, the relationship is real. It does exist." On average, the heart weighs about 306 g in a 70 kg person. He asked, "How do you know that?" I said, "The following equations approximate the weight of an untrained person's heart. For example, the equation  $\text{HW} = (2.54 \text{ kgBW}) + 128$  can be used for men, and the equation  $\text{HW} = (2.10 \times \text{kgBW}) + 126$  can be used to estimate the weight of a woman's heart."

Notice the word "untrained" because the equation is not likely to apply with the same relative accuracy to well-trained athletes. He just stared at me! Then, I said, "The reason is that regular exercise increases the size of the heart. In aerobic athletes, the large left ventricle is a positive adaptation to the large venous return and, therefore, the large left ventricular end-diastolic volume that increases stroke volume." This adaptation at the heart level allows for a more efficient cardiac output (i.e., the volume of blood and oxygen ejected to the peripheral tissues). With increased oxygen at the muscle tissue level, more

energy in the form ATP can be generated by the electron transport system (ETS) within the mitochondria of the muscle cells.

The trainer said, "Honestly, do I need to know what you are saying to lead my clients through an exercise session?" I said, "No, not really but it would be best if you did know the 'physiology' of exercise for safety and ethical reasons." He said, "Okay, then, tell me more!" I said, "Sure, but keep in mind that I am not even touching the tip of the exercise physiology iceberg." The content of exercise physiology and all the academic courses that define an accredited exercise physiology degree is comprehensive. That is why students study the cardiorespiratory system, applied anatomy, biomechanics, sports nutrition, electrocardiography, research and statistics, and dozens of metabolic laboratory assessment techniques.

Understandably, that is why ASEP defines an exercise physiologist as someone who has a college degree in exercise physiology or who has passed the Board Certification EPC exam (2). It is incorrect and, in fact, unethical for a person with a degree in exercise science or kinesiology or one of a dozen other degree programs to refer to him- or herself as an exercise physiologist. Unfortunately, though, too many people with an art degree or an accounting degree think that all they need to do is get a personal trainer certification and, then, they can start their own fitness and health business. Imagine the chaos that would come from a host of other professions in which a person would simply get a weekend warrior pass to engage a client in the use of drugs, update an expensive business computer or perform any function that requires a specialized and engaging education.

He just looked at me. Then, the expression on his face changed. He took a deep breath and said, "Will you explain the importance of knowing the  $MVO_2$  equation you mentioned earlier?" I said, "Do you recall that DP is the product of HR and SBP? If one or both is increased either at rest or during exercise, the work of the heart goes up. This means that the heart needs more oxygen. That is not a problem as long as the heart can get the oxygen it needs. The issue is whether the coronary arteries can supply the heart with an increase in blood flow to provide the oxygen it needs to do the work that is required of it."

Let us assume that a client I have been working with complains of chest pain earlier in the day before his appointment. The fact that I understand the chest pain might be a precursor to a heart attack (i.e., myocardial infarction) is very important. In fact, let us assume that his normal resting HR and SBP are  $70 \text{ beats} \cdot \text{min}^{-1}$  and  $120 \text{ mmHg}$ , respectively, with a calculated DP of 84. But, today, his resting HR and SBP are  $140 \text{ beats} \cdot \text{min}^{-1}$  and  $180 \text{ mmHg}$ , respectively, then, his DP is 252 or 3 times higher than should be. Once again, if he has good blood flow to the heart, then, there shouldn't be a problem.

Here, we can estimate how much oxygen is needed at the heart by using the  $MVO_2$  equation:  $MVO_2 = .14(DP) - 6.3$  and demonstrate that the elevated DP correlates with an elevated  $MVO_2$ . At the original resting DP of 84,  $MVO_2$  is  $5.46 \text{ mL} \cdot 100 \text{ g LV} \cdot \text{min}^{-1}$ . But, with the increase in DP to 252, the client's  $MVO_2$  is now  $28.98 \text{ mL} \cdot 100 \text{ g LV} \cdot \text{min}^{-1}$ , which is 5.31 times higher than it should be at rest. If the coronary arteries are occluded by

disease or inflammation, then, the chances increase substantially for experiencing a heart attack.

Now, I could simply dismiss the likelihood of pathology and carry on with the notion of "No pain, no gain." What do you think? Of course, the answer is "No!" Perhaps, the following calculation will help as well. To begin with, we will determine client's oxygen pulse ( $O_2$  pulse), which is  $VO_2$  divided by HR. At rest, the client's  $VO_2$  and HR are on average for a 70 kg man approximately  $250 \text{ mL}\cdot\text{min}^{-1}$  and  $70 \text{ beats}\cdot\text{min}^{-1}$ , respectively. This means that  $O_2$  pulse is  $3.57 \text{ mL}\cdot\text{beat}^{-1}$ . Now, if we put the  $O_2$  pulse value in the stroke volume (SV, i.e., the volume of blood ejected per beat) equation to estimate SV, we will see that his SV is approximately  $72 \text{ mL}\cdot\text{min}^{-1}$ .

Then, I stopped to say, "My point is this: With the client's sensation of chest pain and his feeling of weakness, his HR is expected to be increased well above the usual resting value. For example, if it is  $120 \text{ beats}\cdot\text{min}^{-1}$ , then  $O_2$  pulse is decreased to  $2.08 \text{ mL}\cdot\text{beat}^{-1}$ . That means his SV is decreased to  $65 \text{ mL}\cdot\text{beat}^{-1}$ , given that a good estimate of the client's  $SV = 5.22 \times O_2 \text{ pulse} + 53$ . Here again, since the client's resting HR is  $120 \text{ beats}\cdot\text{min}^{-1}$  and his SV is  $65 \text{ mL}\cdot\text{beat}^{-1}$ , his cardiac output (Q) is about  $7.8 \text{ L}\cdot\text{min}^{-1}$  versus the normal average of  $5 \text{ L}\cdot\text{min}^{-1}$  for a resting untrained 70 kg man. The elevated Q is another reason for the tendency to experience chest pain, especially when the coronary arteries cannot supply the blood to the myocardium as they should."

Then, it hit me! While talking about the physiology of exercise medicine is important, it isn't the same as listening to a series of lectures, reading articles and books, or actually doing the calculations in a laboratory setting. There was no reason to continue with the 20 or more equations that help to provide additional insight into how the body responds at rest and during exercise, disease conditions, or from exercise training. Instead, I said just as we were parting ways, "If you are going to push your client's, shouldn't you understand the physiology of prescribing exercise medicine? Otherwise, you are likely to hurt or even kill someone...really!"

## Reference

1. Boone, T. (2014). *Introduction to Exercise Physiology*. Jones & Bartlett Learning.
2. *American Society of Exercise Physiologists*. (2015). <https://www.asep.org/>