Ventilatory Threshold

Ventilatory threshold corresponds well with the concept of the lactate threshold or a rise in blood lactic acid concentration greater than 1 mmol·L^{-1} above rest. Typically, this ranges around 2 mM blood lactate in most people. Meyer et al.[1] have termed this phenomenon as the aerobic threshold. VT is obtained by using the "V-Slope Method," in which $\dot{V}O_2$ (L·min^{-1}) is plotted on the *X*-axis of a graph and $\dot{V}CO_2$ (L·min^{-1}) is plotted on the *Y*-axis. The underlying phenomenon surrounding the VT is that, at some point during an increasing exercise workload, the rise in blood lactate concentrations leads to an overproportional increase in CO_2 as related to oxygen uptake due to bicarbonate buffering of proton accumulation associated with lactate dissociation. When this occurs, there is a deflection in the line obtained from the previous graphing method. This can also be obtained by examining the first rise in the ventilatory equivalent for O_2 ($\dot{V}E/\dot{V}O_2$) without a concomitant rise in the ventilatory equivalent for CO_2 ($\dot{V}E/\dot{V}CO_2$).

Respiratory Compensation Point

The respiratory compensation point corresponds well with the concept of anaerobic threshold or the onset of blood lactic acid accumulation (OBLA). It is this point that is strongly associated with 10-km running pace and 40-km cycling time trial pace. It is also useful for delineating interval-training efforts. The underlying phenomenon is an accumulation of lactic acid equal to or greater than 4 mmol·L^{-1}, leading to the inability to buffer lactic acid via the use of the bicarbonate system. From a respiratory point of view, RCP represents the onset of exercise-induced hyperventilation due to inadequate bicarbonate buffering, thus causing an overproportional increase in ventilation ($\dot{V}E$) as related to $\dot{V}CO_2$. A synonymous term, albeit confusing, to describe this term is ventilatory threshold 2 (VT_2).

Putting It Together

The benefits of using VT and RCP as a training aid are many. When collected simultaneously, both can be used to establish basic training zones associated with heart rate, running pace, cycling power output, etc. A synopsis of these training zones is provided below.

Zone 1: The initiation of exercise through VT
  Intensity: Very low to low.
  Perceived Exertion: 6–11 on 6–20 and 1–4 on 1–10 Borg scales
  Energy Source: Fat and fat/carbohydrate
  Functional Pace: Warm-up, cool-down, baseline and recovery, light aerobic activity
Zone 2: VT through RCP
  Intensity: Moderate intensity cardiovascular through high-intensity cardiovascular
  Perceived Exertion: 12–16 on 6–20 and 5–8 on 1–10 Borg scales
  Energy Source: Carbohydrate/fat through glycolysis
  Functional Pace: Race pace
Zone 3: RCP and above
  Intensity: High-intensity cardiovascular through high-intensity cardiovascular
  Perceived Exertion: 17–20 on 6–20 and 9–10 on 1–10 Borg scales
  Energy Source: Muscle glycogen
  Functional Pace: Anaerobic threshold, interval training, attack, and breakaway pace

References

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2. Lucia A, Earnest C, Arribas C. The Tour de France: a physiological review. Scand J Med Sci Sports. 2003;13(5):275–283.

Quick Review

- Ventilation is in part controlled by the partial pressure of carbon dioxide, partial pressure of oxygen, and acidity, all of which are related to metabolism. As a result, ventilation is not perfectly related to oxygen consumption.
- Because the ventilatory equivalent of carbon dioxide is a relatively stable number with increasing workloads, it indicates that partial pressure of carbon dioxide is a major factor controlling ventilation.
- Because the ventilatory equivalent of oxygen is a less stable number with increasing workloads than the ventilatory equivalent of carbon dioxide, it indicates that the partial pressure of oxygen exerts less control of ventilation than the partial pressure of carbon dioxide.
- Changes in the ventilatory equivalent of carbon dioxide and ventilatory equivalent of oxygen can be used to determine ventilatory threshold, an indirect estimate of lactate threshold.

VENTILATION LIMITS

As exercise intensity increases, so does $\dot{V}E$ because of an increase in both tidal volume and breathing frequency. At high exercise intensities, tidal volume tends to plateau so that the only way to further enhance $\dot{V}E$ is by increasing frequency. Corresponding to this, the work of ventilation is intensified, which in turn results in an increased need for oxygen by the respiratory muscles. For untrained healthy adults, the oxygen cost of ventilation is 3% to 5% of total oxygen intake ($\dot{V}O_2$) during moderate exercise, and increases to 8% to 10% of total oxygen intake at $\dot{V}O_{2peak}$.[1] As with other muscles, as exercise intensity increases, venous blood leaving the respiratory muscles shows increased oxygen desaturation, indicating an increase in a-v O_2 difference.[11]

The diaphragm is a highly oxidative muscle and therefore is fatigue resistant. Because of the diaphragm's resistance to fatigue during low- to moderate-intensity exercise in healthy adults at sea level, respiratory muscle fatigue does not appear to limit exercise performance.[7]

However, respiratory muscle fatigue does occur with some disease states, such as obstructive lung disease, and may occur at higher exercise intensities in healthy people.

Diaphragm force output in trained and untrained individuals is not decreased during exhaustive exercise at intensities less than 80% of $\dot{V}O_{2peak}$. However, during exercise at intensities greater than 80% to 85% of $\dot{V}O_{2peak}$ continued to exhaustion, diaphragm force output significantly decreases.[3,10] Fatigue of the diaphragm does not necessarily mean that the ability to ventilate the lungs is compromised, because some fatigue does not mean that the diaphragm cannot perform most of its ventilatory function. Additionally, if diaphragm fatigue is present, a greater portion of muscular work performed for ventilation may be assumed by accessory ventilatory muscles.

Box 6-6 DID YOU KNOW?

Diaphragm Training with Nonrespiratory Maneuvers

The diaphragm does undergo training-induced adaptations because of the need for increased pulmonary ventilation during physical activity, as well as because of the increased work of breathing with diseases such as chronic obstructive lung disease (COPD). However, the diaphragm also undergoes training-induced adaptations due to nonrespiratory maneuvers, such as physical labor and weight training exercises. During weight training exercises, such as sit-ups, bicep curls, bench press, and dead lifts, the diaphragm and abdominal musculature are recruited to help stabilize the lumbar spine area. Contraction of the abdominal musculature during weight training exercises results in an increase in intra-abdominal pressure that decreases the compressive forces on the spine and helps to stabilize the spine. The increase in intra-abdominal pressure also pushes the diaphragm into the thoracic cavity, resulting in an increase in the intrathoracic pressure. If the glottis is open due to the increase in intrathoracic pressure, air will leave the lungs. However, if the glottis is closed, a Valsalva maneuver is performed and blood pressure increases, substantially increasing the force the left ventricle must develop in order to eject blood into the systemic circulation. This is why weight trainers are told not to perform the Valsalva maneuver or at least to minimize its effect. To decrease the intrathoracic pressure while performing a Valsalva maneuver, the diaphragm can be recruited. When active, the diaphragm flattens, resulting in an increase in intra-abdominal pressure and a decrease in intrathoracic pressure. If the diaphragm is recruited while performing a Valsalva maneuver, it decreases intrathoracic pressure, minimizing the effect of the Valsalva maneuver on blood pressure. Performing weight training exercises for 16 weeks significantly increases both the thickness of the diaphragm, indicating hypertrophy, and the maximal inspiratory pressure at the mouth, indicating an increase in force capabilities of the diaphragm.[1] Thus, the diaphragm not only adapts because of its recruitment during inspiration, but also because of recruitment during weight training exercises.

Reference

1. DePalo VA, Parker AL, Al-Bilbesi F, et al. Respiratory muscle strength training with nonrespiratory maneuvers. J Appl Physiol. 2004;96:731–734.

Box 6-7 APPLYING RESEARCH

Respiratory Muscle Training in Swimmers

It is controversial whether respiratory muscle training can increase vital capacity or total lung volume. If it is possible to increase these lung capacities, however, it would be advantageous to swimmers. An increase in vital capacity or total lung volume would increase the buoyancy of the swimmer. Passive drag while swimming (resistance to movement) is lower when lung volume is higher. This in part explains why a large total lung volume is beneficial for competitive swimmers. So, if respiratory muscle training would result in an increase in total lung capacity, it would be advantageous for the competitive swimmer.

Glossopharyngeal breathing (GPB) is the use of the glossopharyngeal muscles to assist in lung accommodation by pistoning or gulping small amounts of air (200 mL) into the lungs. This type of ventilation training is used by patients with neuromuscular disorders that affect respiratory muscles and can normalize tidal volumes within these patients.

GPB training performed for 6 weeks has been shown to increase vital capacity of healthy sedentary women by 3%.[1] GPB training performed for 5 weeks significantly increased vital capacity in female swimmers by 2%, but had no significant effect on vital capacity in male swimmers, although vital capacity did increase slightly.[2] The increase in vital capacity resulted in an increase in buoyancy of 0.17 and 0.37 kg in male and female swimmers, respectively. The authors speculated that GPB training resulting in an increase in vital capacity of swimmers would have a positive effect on the swimmers' maximal velocity through the water with filled or partially filled lungs.

References

1. Nygren-Bonnier M, Lindholm P, Markstrom A, et al. Effects of glossopharyngeal pistoning for lung insufflation on the vital capacity in healthy women. Am J Phys Med Rehabil. 2007a;86:290–294.
2. Nygren-Bonnier M, Gullstrand L, Klefbeck B, et al. Effects of glossopharyngeal pistoning for lung insufflation in elite swimmers. Med Sci Sports Exerc. 2007b;39:836–841.

and breathing frequency could also be increased to partially compensate for a decrease in tidal volume. If respiratory muscle fatigue does occur, it raises the question of whether respiratory muscles undergo training adaptations.

Research has shown that the respiratory muscles can, in fact, undergo adaptations to physical training. For example, the oxidative capacity of respiratory muscle increases due to endurance training.[16] The extra work of breathing required in those with chronic obstructive pulmonary disease (COPD), which increases airway resistance, also stimulates increased oxidative capacity of the respiratory muscles.[5] However, the concentration of glycolytic enzymes in respiratory muscles changes little with physical training. The improved oxidative capacity in the diaphragm of endurance-trained athletes allows that muscle to avoid indications of fatigue until reaching levels of $\dot{V}_E$ that are higher than those of healthy sedentary individuals.[2] Although the diaphragm is largely a respiratory muscle, it is also recruited during nonrespiratory maneuvers,[6] such as physical labor or weight training activities (see Box 6-6). In response to being recruited during nonrespiratory maneuvers, the diaphragm does hypertrophy, as indicated by an increase in diaphragm thickness and force capabilities.[6] So, it appears that respiratory muscles, like any other muscle, can undergo adaptations to physical training. One unique application of respiratory muscle training is used by swimmers and is described in Box 6-7.

CASE STUDY

Scenario

You are the coach for a Division I men's cross-country team at a university located at sea level. You are traveling to a competition at a university with a course located at 2,300 m in altitude.

Questions

How do you expect performance to be affected?

Options

Endurance events, lasting longer than 2 minutes, are highly reliant on oxygen delivery to tissue. At altitudes above sea level, partial pressure of O_2 is lower. This directly impacts the O_2 saturation of hemoglobin and oxygen transport. As Po_2 decreases, there is a decrease in the amount of O_2 bound to hemoglobin. Thus, the capacity to transport oxygen to exercising muscles is reduced and maximal oxygen consumption is reduced. Even with acclimatization, endurance performances will be hindered.

When exposed to low Po_2 at altitude, the body's response is to produce additional red blood cells to compensate for the desaturation of hemoglobin. Thus, allowing athletes sufficient time to acclimatize to the altitude may help performance. However, at altitude, training intensities will be hindered; thus, you may want your athletes to follow the model of "live high and train low." This means that athletes should live and sleep at altitude to allow enhanced red blood cell production, but to return to sea level to train to allow maintained training intensities. In addition, hypoxic masks have been used to enhance metabolic and respiratory function with training while at sea level. However, these types of training are not at your disposal. So you decide to let your athletes know this will be a difficult meet and they should just do their best.

Scenario

You are a road cycling coach and have seen advertisements for devices that are supposed to train the muscles of inspiration. Several of your athletes have asked you if these devices work or might improve their cycling performance.

Options

You perform a literature search and find several articles related to endurance performance and use of inspiratory muscle training. These devices make it more difficult to inspire resulting in the muscles of inspiration needing to develop more force in order to perform their function. Over a period of time this results in possibly increased strength and endurance of these muscles and so less fatigue during an endurance event. This potentially results in greater pulmonary ventilation and so oxygen supply to working muscles.

Several studies do show improved performance after inspiratory muscle training in trained athletes. Mean power during 6 minutes of rowing is increased 2.7%,[1] whereas 20-, 25- and 40-km time trial performance is improved by 2.7% to 4.6%.[2,3] However, other studies also show no significant improvement in performance. It does seem that inspiratory muscle training increases strength and endurance on the inspiratory muscles. For example, training increases the inspiratory muscle pressure approximately 26% in rowers.[1] It also appears that inspiratory muscle training results in increased performance with as little as 4–6 weeks of training. With your research and the apparent lack of negative side effects, you decide to advise your athletes to try inspiratory muscle training for a short period of time.

References

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2. Johnson MA, Sharpe GR, Brown PI. Inspiratory muscle training improves cycling time-trial performance and anaerobic work capacity but not critical power. Eur J Appl Physiol. 2007;101:761–770.
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Quick Review

- In healthy individuals at submaximal workloads, respiratory muscle fatigue does not occur; however, at exercise intensities above 80% of $\dot{V}O_{2peak}$, the diaphragm can show indications of some fatigue.
- The respiratory muscles, including the diaphragm, do adapt to training.

CHAPTER SUMMARY

The respiratory and circulatory systems work together to supply tissues with oxygen and to expel carbon dioxide from the body. Pulmonary ventilation and pulmonary diffusion are referred to as pulmonary respiration because these two processes occur at the alveoli in the lungs. Cellular respiration refers to the use of oxygen in aerobic metabolism and production of carbon dioxide by tissue. Partial pressure gradients between the air and blood (pulmonary respiration) and blood and tissue (cellular respiration) determine the direction and rate of the exchange of oxygen and carbon dioxide during respiration. In addition to gas exchange at the lungs, the pulmonary system also humidifies, filters, and warms air to protect the alveoli and respiratory membrane from damage. During inspiration, contraction of the inspiratory muscles, of which the diaphragm is the most important, causes a decrease in intrapulmonic pressure, resulting in air rushing into the lungs. Relaxation of the inspiratory muscles causes an increase in intrapulmonic pressure, resulting in expiration. During physical activity, expiratory muscles contract, thus aiding expiration.

During light to moderate physical activity, pulmonary ventilation is increased due to both an increase in tidal volume and frequency of breathing. At higher intensities of physical activity, tidal volume plateaus so that the only way to increase pulmonary ventilation is to increase frequency of breathing.

The majority of oxygen is transported bound to hemoglobin, whereas the majority of carbon dioxide is transported as bicarbonate. The transport of oxygen and carbon dioxide, however, is affected by increased acidity, increased temperature, and increases in partial pressure during physical activity, so that more oxygen is delivered to active tissue. This is in large part due to shifting of the oxyhemoglobin disassociation curve so that hemoglobin decreases its affinity for oxygen at working tissue.

In order to meet the body's needs, pulmonary ventilation at rest and during physical activity is controlled by the respiratory center located in the medulla and pons. The respiratory center receives input from many sources, including peripheral chemoreceptors, central chemoreceptors, higher brain centers, muscle proprioceptors, and muscle chemoreceptors. This input allows pulmonary ventilation to change to meet the needs of oxygen delivery to tissue and carbon dioxide removal. The major determinants of pulmonary ventilation are hydrogen ion concentration, oxygen partial pressure, and carbon dioxide partial pressure, which are monitored closely by chemoreceptors so that pulmonary ventilation changes to meet the metabolic needs of the body. However, as workload increases, there are several points at which there are alterations in pulmonary ventilation relative to the amount of oxygen and carbon dioxide exchanged at the lungs. These changes in pulmonary ventilation are related to performing workloads that are above lactate threshold.

Respiratory muscles can adapt to physical training, resulting in less respiratory muscle fatigue during physical activity. If it were not for the respiratory system's remarkable ability to match pulmonary ventilation with the metabolic needs of the body at rest and during physical activity and its ability to adapt to training, our ability to perform both aerobic and anaerobic exercise would be compromised.

REVIEW QUESTIONS

Fill-in-the-Blank

1. ________ are saclike structures attached to the respiratory bronchioles and are surrounded by capillaries, where gas exchange takes place.
2. If intrapulmonic pressure is ____ than atmospheric pressure, air will move into the lungs or _____ will occur; if intrapulmonic pressure is ____ than atmospheric pressure, air will move out of the lungs or _____ will occur.
3. The partial pressure of oxygen in the alveoli is _____ than the partial pressure of oxygen in the blood, which allows oxygen to diffuse into the blood. However, the partial pressure of oxygen in muscle is _____ than the partial pressure of oxygen in the blood, which allows oxygen to diffuse into the muscle.
4. When oxygen is bound to hemoglobin, ____________ is formed, whereas hemoglobin not bound to oxygen is termed ____________.
5. The ____________ is the workload at which $\dot{V}_E$ increases disproportionately relative to $\dot{V}O_2$ and changes from proportional increases in $\dot{V}_E$ relative to workload to disproportionate increases in $\dot{V}_E$ relative to workload.