Doctor's Training V02 Interpretation

What is a Good Test - What to Look For

1. Review age, gender, weight, and limitations noted in Tech Test Report (May explain poor test results)
2. Evaluating the Quality of the Test: The most important issue to Evaluate! Everything Follows from this!
    a. Predicted MHR vs Peak Heart Rate #44 #46 #36 #34 #15 #40 #11
        i. Predicted Maximal Heart Rate (220-age) vs Peak Heart Rate noted on Results (Should be within +/- 10 beats) If it is below this, then look at the following...
            (1) Review the V02 vs Time Chart (#1) on test. Look carefully at the slope off the Heart Rate. (Goal - an ever increasing gradual slope reaching within 10-15 beats of PMHR with in 10-15 mins) (Submaximal Test 3+ mins to 85% PMHR)
            (2) Review the V02 vs Time Chart (#1) on test. Look to see if 02 was eclipsed by C02. If not, clearly the patient's pulmonary system was not fully tested. Further the RER should be over 1.15 to verify AT (C02 eclipse of 02). RER is Ratio between C02 produced in metabolism and 02 used
            (3) If there is not a True AT (C02 Eclipsing 02) with a <10-12 bpm of PMHR, then it is most likely due to too little effort during testing. (Except Submaximal Clients)
            (4) Failure to reach PMHR by >15 bpm may be a sign of poor effort, testing, or patient's health limitations. Review the RER
                (a) May be a sign of Heart or Pulmonary limitations (Dx. CHF-COPD-Etc)
                (b) May be a sign of Musculoskeletal Issues
                (c) It could be a "Submaximal" test, but low or poor testing is not the same
            (5) Submaximal Testing would be 85% of Predicted MHR.
                (a) Example 60 yr old. 220-60 = 160, then 160 x .85 = 136 hr (<24 bmp)
                (b) Submaximal Testing can last 3+ mins. To reach 85% PMHR
                (c) Possible Tech deferred to Submaximal Test for a reason
                (d) Unnecessary Submaximal Testing can provide inaccurate data and Interpretation, resulting in inaccurate health classification, reccommendations and work out parameters.
                (e) Maximal Testing can be very risky with the wrong person
            (6) Chronotropic incompetence (CI), broadly defined as the inability of the heart to increase its rate commensurate with increased activity or demand, is common in patients with cardiovascular disease, produces exercise intolerance which impairs quality-of-life, and is an independent predictor of major adverse cardiovascular events and overall mortality
            (7) Review Meds for Beta Blockers
3. Review the RER
    a. This measures the effort the patient exerted during the test.
    b. RER should be between .70 and .80 prior to exercise, although some patients may start higher, like 1.0, often because of some pre-test anxiety
    c. A Peak RER of 1.10 or greater is considered a standard of high effort, and suggest that the peak V02 obtained in the test provides an accurate estimate of the patient's true maximal functioning capacity (Cardiopulmonary Exercise Testing - Daniel E Foreman ...)
    d. In patients who fail to reach an RER >105, we look for other indicators of a maximal effort or advanced disease

patterns, such as early flattening of the V02 even when RER is lower than 1.1

e. We generally disregard the peak V02 when RER is <1.00. Notable our own analysis demonstrated that even peak V02 values associated with very low RERs (<1.0) remained prognostic, albeit, much less reliable than v02 values associated with higher RER values

4. Review the Data Sheet on V02 Peak

a. review the data to make sure that the increase in VO2 is relatively consistent throughout the test and there are not big jumps or drop-offs that might indicate an air leak or other problems. Traditionally, the peak VO2 is selected as the highest value obtained during the last minute of exercise.

b. When evaluating peak VO2 , we compare the measured value to the predicted value derived from the Wasserman equations based on the patient's age and gender. The reason for this is that a pVO2 of 14.5ml/kg/min may be relatively normal for an 80 year-old woman while a peak VO2 of 25 ml/kg/min may represent a significant limitation for a 23-year-old former athlete. In general, we usually use the following classification

c. Wasserman's Equation for Predicted V02

i. [50.72 - (0.372 x age)] x weight (Sedentary Men)

ii. [22.78 - (0.17 x age)] x (weight + 43) Sedentary Women

(1) Normal: measured peak VO2 80% or greater of predicted

(2) Mild limitation: measured peak VO2 60% to 79% predicted

(3) Moderate limitation: measured peak VO2 40% to 69% predicted

(4) Severe limitation: measured peak VO2 <40% predicted

b. When looking at the peak VO , it is important to take the patient's body habitus into account. Since the peakVO is measured per kilogram of body weight, morbidly obese patients can have a falsely low peak VO .Lavie et al 2013 found when peak VO2 falls below 14 ml/kg/min in obese patients, a closer consideration should be made regarding their body habitus .

c. V02 Peak Vs V02 Max

d. V02 Max or Peak is the "MOST" relevant piece of information regarding interpretation

2. Measure the VE/VCO2 slope

Practical Guide to Interpretation of Cardiopulmonary Exercise Testing

Oxford Respiratory Medicine Library (William Kinnear / John Blakey)

11/27/19

1. **Key CPEX Measurements**

a. The volume of air breath in and out (minute ventilation (VE)

b. The maximum volume of oxygen extracted and used by the body in one minute (Oxygen Uptake: V02)

c. The Volume of Carbon Dioxide produced (VC02)

d. Heart Rate (HR)

e. Oxygen Saturation (Sp02)

2. **Pointers to the Presence of Heart Disease**
    a. Reduced Exercise Capacity (Low V02 Max)
    b. Heart Rate Rises Rapidly (No HR Reserve at Peak Exercise)
    c. Impaired Ventricular Stroke (Minimal rise in 02 Pulse) (Left Ventricle)
    d. Low Cardiac Output, with Poor Delivery of O2 to Muscles (Anaerobic Threshold Occurs Early)
    e. Exercise Not Limited by Breathing (VE does not Reach Predicted Levels)
    f. Inefficient Ventilation, with High Ratio of Dead Space Volume (Vd (Dead Space Volume) to Tital Volume VT) High Ventilatory Equivalents
3. **Pointers to the Presence of Lung Disease**
    a. Reduced Exercise Capacity (Low V02 Max)
    b. Exercise Not limited by Cardiac Output (HR Does Not Reach Predicted)
    c. Exercise Limited by Breathing (No Reserve in VE at Peak Exercise)
    d. Exercise Often Stops as Soon as Muscles Become Anaerobic
        i. VE cannot increase in response to C02 production
    e. Inefficient Ventilation, with High Ratio of Vd to Vt (High Ventilatory Equivalents)
    f. Limited Rise in Tital Volume VT
    g. Impaired Inability to Increase Ventilation in Response to Acidaemia (No RCP - Respiratory Compensation Point.
    h. Desaturation
4. **Things to Do First Before Testing**
    a. Detailed History
    b. Meticulous Clinical Examination
    c. A Few Basic Tests - Chest xray, ECG, Haemoglobin (Hb) Concentration, and Renal Function)
    d. Spirometry - FEV1 for MMV

8 Parameters of Testing

1. **Oxygen Uptake (V02)**: volume of oxygen (V02) utilized in metabolism by the body, primarily in mitochondria. It is the difference between inhaled and exhale 02, fractions are expressed in ml/min or standardized for weight as ml kg/min
    a. Equation Predicted V02 Max = 50 - (0.4 x age in years)
        i. Wasserman's Equation for Predicted V02 (Copied from previous page)
            (1) [50.72 - (0.372 x age)] x weight (Sedentary Men)
            (2) [22.78 - (0.17 x age)] x (weight + 43) Sedentary Women
    b. Normal 80% Predicted - means that it is very unlikely that the subject has clinically significant pathology affecting their heart or lungs
    c. V02 Peak vs. V02 Max
    d. **What Causes a Low V02 Max?** (Pg20)
        i. A V02 Max of less then 20 ml kg/min is low, less than 15 ml kg/min is moderate impairment, and less than 10 ml kg/min is severe impairment
    e. **V02 Max and Mortality**
        i. Lower V02 Max is associated with earlier death in the general population without diagnosed major illnesses.
        ii. V02 Max is closely related to risk of death or major complications in cardiac and lung surgery, and is associated to

varying degrees with poor outcomes in other major surgeries.

iii. Lower V02 max is associated with poorer outcome in chronic cardiopulmonary diseases

2. **Heart Rate**

a. Normal subjects will reach 80% or more of their predicted maximum heart rate (HR): Cardiac output is what determines their exercise capacity (PMHR = 220 - age)

b. Unfit subjects and patients with heart disease will also reach 80% of predicted maximum heart rate (HR), but their maximum oxygen uptake (V02 Max) will be low.

c. If the maximum heart rate is low, either the subject didn't try very hard or something other then the heart is limiting exercise.

d. **Heart Rate Reserve**

i. **Low HR Reserve**: An HR >80% predicted at peak exercise is often referred to as " low HR Reserve". The possibility of increasing HR any further is pretty limited

ii. Unfit subjects will push up their HR pretty quickly, as with those with impaired Left Ventricular function, in whom the only way to get more cardiac imput is to increase heart rate. This is also seen in those with pulmonary vascular disease in whom the Right Ventricular function is impaired. (All these subjects will have a low V02 Max)

iii. **High HR Reserve**

(1) The HR will not reach 80% predicted (High HR Reserve)

(a) If the subjects stops early

(b) If the test stopped too early

(c) Or test was limited by something other than cardiac function, such as, musculoskeletal issues, Lung Disease, or peripheral vascular disease

iv. Chronotropic Insufficiency = is a fancy way to say that the HR doesn't rise normally.

(1) Since cardiac output is stroke volume X HR, the ability of the heart to increase its output (and hence the volume of 02 it transports from the lungs) will be compromised. Drugs such as Beta Blockers may be the culprit or Sinoatrial node dysfunctions. This pattern is not particularly common, but worth looking out for

v. **High Heart Rate**

(1) Not uncommon to see a high HR in an anxious subject, often this settles down in the early phase of exercise testing

(2) A persistence tachycardia may indicate poor ventricular function, since stroke volume cannot increase. So the only way of increasing cardiac output is by increasing HR

3. **02 Pulse**

a. **Intro:**

i. 02 Pulse can be used as an indirect indicator of cardiac stroke volume.

ii. A normal subject should achieve an 02 of more than 10 ml / beat at peak exercise

iii. A plateau in the 02 pulse at a low value implies limited cardiac output. Because of either heart disease or disorders of the pulmonary circulation

iv. Don't over-interpret a low 02 pulse, particularly if the maximum oxygen uptake (V02 Max) is normal

v. Subjects should reach 80% of Predicted 02 Pulse

b. **What is 02 Pulse?** Is simply oxygen uptake (V02) divided by heart rate (HR)

c. **What does the 02 Pulse measure?**

i. 02 Pulse is the amount of oxygen (02) taken up by the lungs into the blood with each heart beat.

ii. If there is more blood flowing thru the lungs, then more 02 will be taken up.

iii. Cardiac output is the product of HR and stroke volume, so V02 is related to cardiac output by the equation

(1) Cardiac output = stroke volume x HR = V02

(2) 02 Pulse = V02 / HR = Stroke Volume

d. **Equation for Normal Pulse Rate**

i. Divide the Predicted V02 Max by the Predicted Maximal Heart Rate

(1) Predicted V02 max / PMHR = Predicted 02 Pulse

e. **Low 02 Pulse**

i. In heart disease, stroke volume may not increase at all. The only way to increasecardiac output is by speeding up the heart. In this case the 02 Pulse (i.e. stroke volume) will remain much the same throughout the test

ii. If the subject develops cardiac ischaemia , stroke volume will be suddenly impaired.

iii. If the 02 pulse reaches a plateau , suspect impairment of cardiac output (due to heart disease or pulmonary vascular disease), particularly if the peak value is less then 10 ml/beat

f. **02 Pulse and Athletes**

i. Page 31

ii. Fick Equation (pg 31)

4. **Ventilation**

a. **Key Points**

i. Minute Ventilation (VE) increases during a cardiopulmonary test.

ii. VE does not normally limit exercise

iii. If VE reaches 80% of predicted, this implies there is something wrong with the lungs

iv. Tital Volume (VT) Should increase in early part of CPEX

v. SPIROMETRY - We need to pretest for: FEV1, which can give us MVV, and Tidal Volume

b. **Ventilation**

i. The sum of the volume of all breaths in one minute

ii. Product of Frequency and Depth

iii. Normal resting breathing is about 12 breathers per minute

iv. Anxious subjects may hyperventilate at beginning of test, but will level out

c. **Predicted Values**

i. Theoretically maximum minute ventilation (VEmax) is usually estimated from the subject's forced expired volume in 1 s (FEV1) Spirometry. The most accurate simple formula seems to be: VEmax = (FEV1 x 20) + 20. With FEV1 in liters per min.

(1) the Maximum Voluntary Ventilation (MVV) could be done also thru spirometry, but it highly dependent upon the motivation of the subject and can lead to hypocapnia or can provoke bronchoconstriction.

d. **Ventilation Reserve**

i. Ventilation reserve is the same concept as HR reserve.: If VEmax is >80% of predicted value, then this is called low Ventilation reserve. (there is little possibility of increasing ventilation any further.)

ii. In normal subjects. As well as those with heart disease, cardiac output limits exercise. There is usually sufficient reserve in ventilation that does not reach 80% of the predicted value.

e. **Ventilation Limitation**

i. In lung disease, the subject will stop because of ventilation limitation. From a cardiac point of view, it is as if the patient stopped before maximum capacity was reached, so the HR will be < then 80%

ii. In clinical experience, a CPEX does not often reveal significant lung disease which could not have been anticipated from

the tests you do normally do before CPEX testing. (Spirometry and ?)

iii. Athletes can reach their predicted ventilation because of their training in which they can increase their cardiac output

iv. Nevertheless, it is important to be able to recognize the sorts of abnormalities seen when the lungs are a factor limiting exercise. Especially useful prior to thoracic surgery or pulmonary rehab. Programs

f. **VT (Tidal Volume)**

i. is the lung volume representing the normal volume of air displaced between normal inhalation and exhalation when extra effort is not applied. In a healthy, young human adult, tidal volume is approximately 500 mL per inspiration or 7 mL/kg of body mass..

ii. A high Minute ventilation is the tidal volume times the respiratory rate, usually, 500 mL × 12 breaths/min = 6000 mL/min. Increasing respiratory rate or tidal volume will increase minute ventilation.

iii. In a normal subject, VT increases during low intensity exercise. As the load gets more severe, further increases in ventilation are achieved by increasing the rate.

iv. Failure to increase VT during Testing implies the presence of Lung Disease

v. CPEX has a measurement of VT/VE , which we do not have

g. **Dysfunctional Breathing**

i. Looking at VE/Time plots can give us some clues that a subjects breathlessness is a problem of perception, rather than indicating a physiological problem with the heart or lungs. Rather erratic ventilation implies dysfunctional breathing, which is probably a better term than "hyperventilation syndrome". Look at this more under C02 output

(1) chart has a wide scatter

5. **Carbon Dioxide Output**

a. **Key Points**

i. Carbon Dioxide Increases during exercise

ii. C02 is produced by burning fuel

iii. C02 is also a by-product of buffering lactic acid

iv. Exhaled C02 comes from Alveolar Ventilation

b. **What is C02 Output?**

i. Carbon dioxide output (VC02) is the volume of C02 exhaled, expressed in ml/min

c. **How is VC02 Measured?**

i. The amount of oxygen taken in by the body (V02) is calculated by looking at how much oxygen (02) is left in expired air. Working out VC02 is just as simple there is no C02 in inspired air, so looking at the concentration in expired air and multiplying by the minute ventilation (VE) yields VC02 in ml/min.

ii. C02 comes from burning fuel in 02 (Aerobic Metabolism) and from buffering the H+ from lactic acid (generated from Anaerobic metabolism)

d. **Ventilation and VC02**

i. The link between alveolar ventilation and VC02 is pretty tight: more alveolar ventilation means more VC02.

(1) Lots of ventilation without much VC02 implies the lungs are not working

(2) One way of looking at this is to plot VE against VC02, but it is probable easier to assess the efficiency of ventilation by looking at ventilatory equivalents

e. **Alveolar Ventilation**

(1) There are 2 types of Dead Space (Vd).

(a) Anatomical Dead Space: means the volume of the conducting airways, which **can't** participate in gas exchange

because they aren't alveoli.

(b) Physiological Vd is the volume of the lung which does not participate in gas exchange, either because it is anatomical dead space (Vd) or because the alveoli aren't perfused.

(c) During exercise, VD declines a bit as more lung units are recruited. Vt increases, as we've seen, so Vd/Vt falls.

f. **Acidaemia and Ventilation**

i. Extreme exercise will result in anaerobic metabolism, and the production of lactic acid will eventually swamp the buffering mechanisms. When this happens more acid appears in the blood: this is called acidaemia.

ii. Acidaemia stimulates ventilation, as for example in patients with diabetic ketoacidosis or renal failure. This hyperventilation in response to acidosis can often be seen on the VE/VC02 plot, and the point at which it starts is called " Respiratory Compensation Point (RCP). (RCP: beyond which there is respiratory compensation point for acidaemia)

g. **Hypercapnia and Hypocapnia**

i. Carbon dioxide diffuses pretty quickly out of the blood into the alveolar gas,> Unlike 02, which is much less soluble, it is not particularly affected by processes such as fibrosis, which is the thickening of the alveolar wall.

ii. The arterial C02 level (PaC02) is the driving pressure, which determines how fast C02 flows out into alveolar gas – if a subject hyperventilates and lowers their PaC02, then the driving pressure is lower and less C02 will be exhaled. This explains the change in Slope beyond the RCP.

iii. IF PaC02 is high, then the driving pressure is high, and more C02 will be exhaled for a given level of ventilation. In practice, the possibility of hypercapnic respiratory failure is likely to have been picked up earlier on in the diagnostic process, prior to testing

h. **VE/VC02 and Mortality**

i. Curiously, several studies have shown that VE/VC02 is a surprisingly good predictor of subsequent morbidity and mortality, especially in patients with heart failure. A high VE/VC02 implies there is a lot ventilation wasted on dead space, possibly because of poor perfusion of some areas in the lungs, which are unable therefore to participate in gas exchange.

ii. Another explanation for High VE/VC02 might be a low PaC02, hence reducing the driving pressure to get C02 from the blood out into the alveolar gas. Patients with severe heart failure sometimes have a low PaC02, probably because they hyperventilate in order to try and keep their arterial 02 level (Pa02) up. A low PaC02 is associated with the development of Cheyne-Stokes respiration, which is a very poor prognostic sign in heart failure, particularly if seen in wakefulness.

6. **Respiratory Exchange Ratio**

a. **Key Points**

i. The respiratory Exchange Ratio (RER) is the Ratio of Carbon Dioxide output (VC02) / Oxygen Uptake (V02)

ii. Beyond the anaerobic threshold (AT), the RER increases above 1.0; VC02 rises more steeply, reflecting the production of Carbon Dioxide (C02) from the buffering of lactic acid, whereas V02 – by definition – cannot increase

iii. Subjects with dysfunctional breathing have erratic RER Traces

b. **What is RER?**

i. RER is the VC02 divided by the V02

ii. RER = VC02/V02

c. **Why is RER Measured?**

i. It gives a way of determining the AT

ii. It shows if the subject is hyperventilating

d. **What Should the RER be?**

i. If VC02 and V02 are plotted against time during a cardiopulmonary exercise test, VC02 is slightly less than V02 durning the first part of test. Example, the RER (VC02/V02) is less than 1.0 At the end of the test, VC02 is greater then V02, so the RER will be greater than 1.0

ii. A plot of RER against time shows the same thing, but is simpler than trying to see if the V02 line is above or below the VC02 line.

iii. **Physiology**

(1) There are reasons why the RER is less than 1.0. Firstly, the cells may produce slightly less C02 than 02 they consume, particularly if they are metabolizing fat rather then glucose. (This is the respiratory quotient of cells, which is sometimes confused with the RER of the whole person.)

(2) Secondly, some of the C02 dissolves in water and becomes part of the bicarbonate (HC03), buffering pool of the body. If some of the carbon in the C02 produced by the cells in the muscle were labeled, some of it would ultimately show up as HC03 excreted in the kidneys. There is no simular alternative for 02

(3) The buffering capacity of the HC03 system is much greater than that available for 02. So any change in C02 has a much slower effect on the levels in the blood than is the case for 02. The slower the kinetics also make the RER a bit lower during a CPEX

iv. **RER and the AT**

(1) Beyond the AT, the subject starts to exhale more 02 (produced from the buffering lactic acid by HC03). At the AT the two lines cross, at this point, VC02 and V02 are the same, so the RER must be 1.0.

v. **Hyperventilation**

(1) Hyperventilation is not unusual at the beginning of a test. Many times subjects are a bit anxious

(2) Hyperventilation causes increased washout of C02 from the alveoli. On the other hand, increased ventilation cannot get any more 02 into the body, because 02 is poorly soluble and the haemoglobin (Hb) in red blood cells is already fully Saturated. As a result , the RER is greater than 1.0

(3) In this context, hyperventilation is “Alveolar “ hyperventilation: If lung disease has led to a very high dead space (Vd), hyperventilation may be necessary to get the C02 out, but the RER will be normal.

(4) Review Dysfunctional Breathing (pg. 48)

7. **Anaerobic Threshold**

a. **Key Points**

i. Beyond the Anaerobic Threshold (AT), anaerobic processes supplement aerobic metabolism, with production of lactic acid

ii. Lactic acid is buffered by bicarbonate (HC03) to produce more carbon dioxide (C02)

iii. The AT should occur when the oxygen uptake (V02) is >40% of the predicted maximum oxygen uptake (V02 max)

iv. A low AT is caused by impaired oxygen (02) delivery to muscles, usually because of heart disease or peripheral vascular disease.

b. **What is the AT**

i. In this book the AT is the point beyond which work is done by anaerobic as well as aerobic metabolism. It is important to remember that aerobic metabolism continues beyond the anaerobic threshold, but that it is supplemented by anaerobic processes. Although there is considerable debate, for simplicity in this book it is assumed that anaerobic metabolism generates lactic acid in the muscles

ii. The AT referred to is that detected by analysis of exhaled gases, i.e. it is a respiratory AT, rather than one determined by measurement of lactic acid in the blood or analysis of changes in the muscles themselves

c. **Why Does the AT Matter?**

i. While the scientist debate, athletes are well aware that there is a threshold below which the intensity of exercise can be sustained for fairly long periods, whereas above this threshold more intense exercise incurs a "debt" which must be repaid.

ii. As the external load increases progressively, during a CPEX, as long as the subject is cycling at the correct speed, the work done gets steadily greater until they stop. Oxygen uptake (V02), however tails off toward the peak of exercise. So how does the work done continue to increase? Well aerobic metabolism must be supplemented by anaerobic processes.

d. **HC03 - Buffering of Lactic Acid**

i. Initially the Hydrogen ions (H+) from the lactic acid produced in muscle is buffered by HC03, producing water (H20) and carbon dioxide (C02) / H+ +HC02 - H20 +C02

ii. The water is easy to get rid of, but where does the C02 go? It is eliminated by increasing ventilation, of course.

e. **The V-slope Method of Detecting the AT**

i. The VC02 is plotted gainst the V02, there is an inflexion point at the AT beyond which the slope is steeper, as the VC02 increases more than V02. This is called the V-slope

f. **Uncertainty about the AT**

i. There is one more method of determining the AT, using ventilatory equivalents. One of the problems of having several different methods is that they may not agree. So there can be some uncertainty about where to place the AT. Indeed, in some subjects it may be impossible to decide where to put it, particularly if the CPEX was very brief.

g. **What is a Normal AT?**

i. The AT should be greater than 40% of Predicted Maximum Oxygen Uptake (V02 Max), in trained athletes this could be higher, even up to 80%

h. **What Causes a Low AT?**

i. Anaerobic metabolism occrs when the circulation is not able to deliver enough oxygen (02) to meet the metabolic needs of the tissue. This may occur at lower than normal exercise intensityduring a CPEX for 3 main reasons:

(1) The cardiac output is low

(2) The blood vessels to the legs are obstructed

(3) The peripheral oxygen saturation (SP02) of arterial blood is low

(4) See Clinical Scenario pg. 54

8. **Ventilatory Equivalents**

a. **Key Points**

i. The ventilatory equivalents for oxygen (VEq02) are the amounts of ventilation (ml/min) divided by how much oxygen (02) is taken in (ml/min)

ii. VEq02 fall during the initial part of the CPEX , as ventilation and perfusion become more even throughout the lungs.

iii. Beyond the AT, VEq02 rise as ventilation increases (stimulated by carbon dioxide output (VC02) without any increase in oxygen uptake (V02)

b. **What are Ventilatory Equivalents?**

i. The VEq02 is simply the minute ventilation (VE) divided by the amount of 02 taken up. In other words, how many milliliters of air went in and out of the lungs to get a milliliter of 02 in.

ii. It can be thought of as an index of how well the lungs work. Lots of air in and out without much 02 taken up sounds bad

iii. Ventilatory Equivalents (VEq) are one of those odd things that have no units. VE is in ml/min, and so is V02: ml/min divided by ml/min leaves nothing. Hence a unit-less index

c. **Why Does VEq Matter?**

i. In the first part of a CPEX, the VEq02 may gradually fall. The lowest point of the VEq is where the lungs are working their best. How many milliliters of ventilation per milliliter of oxygen (02) in at this nadir gives some idea of how good the lungs are

ii. VEq02 falls as cardiac output increases and ventilation (V) perfusion (Q) matching becomes more even (Physiology Pg 58)

d. Why does

9. **Disorders from Research**

a. Respiratory acidosis is a condition that occurs when the lungs can't remove enough of the carbon dioxide (CO2) produced by the body. ... This is also called respiratory failure or ventilatory failure. Normally, the lungs take in oxygen and exhale CO2. Oxygen passes from the lungs into the blood.

i. COPD

10. **Pathologies and Disorders**

a. Heart Pathologies

i. Cardiovascular disease

(1) Heart Disease in General on a V02 Test (Wasserman pg 114-15)

(a) In nearly all heart defects, the increase in heart rate as a function of V02 is steeper than normal.

(i) usually d/t reduced stroke volume

(b) Heart rate response could be inappropriately low because of:

(i) Beta Blockers

(ii) Cardiomyopathies, whose sinoatrial node fails to respond appropriately for low stroke volume

(iii) Patients with Heart Block = Heart block is an abnormal heart rhythm where the heart beats too slowly (bradycardia). In this condition, the electrical signals that tell the heart to contract are partially or totally blocked between the upper chambers (atria) and the lower chambers (ventricles).

(c) Patients with Heart Disease may develop metabolic acidosis at low work rates, this may become chronic and evident at rest

(2) Coronary Artery Disease

(a) It will usually cause peak V02 to be reduced.

(b) They may or may not experience chest pain

(c) When the exercise induced increase in myocardial oxygen supply requirement is not met, by myocardial oxygen supply, myocardial ischemia may result in ST segment and T wave changes in an ECG, and ventricular ectopic beats may develop with increasing frequency as the work rate is increased.

ii. Silent Ischimia

b. Respiratory Pathologies

i. COPD (Asthma - Chronic Bronchitis - Emphysema)

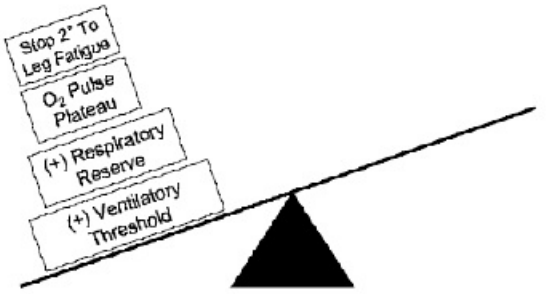
ii. Pulmonary Hypertension

c. PAD

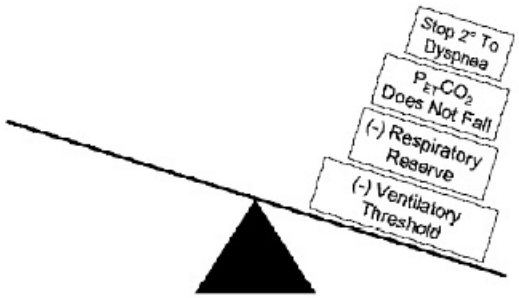
i. Wasserman pg 114

ii.

d. Myopathies



Cardiac Limitation



Ventilatory Limitation