

True Health Center

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Westside Family Acupuncture

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Cardiorespiratory Health and Fitness Report

08/15/23

Patient: John Smith (Age: 49) Test Type: Cardiorespiratory Exercise Test (V02) Treadmill

Purpose of Test: Health Assessment - Risk Assessment - Baseline

Test Date: 08/04/23

Test Results

Cardiorespiratory Test Results V02 36.9 - Fair ml/O2 (121.4% Predict) (94.6% Healthy) Test Duration: 8 min 00s / (AeT to AT: not reached)						Predicted V02 Healthy Good V02 V02 Plateau RER	30.4 ml/kg/min 39 No .68
O2 Pulse Normal Predicted (PV02/PMHR)		Heart Rate Reserve Normal		Chronotropic Response / Heart Rate Recovery		Ventilatory Ratio Ve/V02 to VC02 Abnormal	
Predicted	17.8 ml/beats	PMHR	171 bpm	Expected	> 15 beats	Ventilatory Ratio	1 to 1.66
Actual	21.5 ml/beats	Resting HR	77 bpm	Peak HR	156	Ve/V02 to	
% Predict	120.9 %	Peak HR	156 bpm	1 min < HR	143	Ve/VC02	
Plateau?	No	Expect Reserve	0-15 %	Result	13		
Interp.	Normal	Actual HRR	8.8 %	Interp.	Low Norm	Expected Ratio	
		Interp.	Normal	Heart Resp.	Normal		

RER CVo2 / Vo2 Not Compensating		Ventilatory Reserve Very Low - Abnormal		Respiratory Compensation Ve/V02 to Ve/VC02	Ve /V02	Ve /VC02	Diff Ve/	%
Lowest RER	.56	Spirometry MVV	78.5 /min	Start of Test	29.86	49.23	19.37	39.35 %
Highest RER	.68	Actual MVV	74.0 /min	AeT - Min 5:15	18.05	30.92	12.87	41.62 %
Time	8 min	VR %	5.73 %	Total Avg. Ve	21.28	35.32	14.03	39.73 %
Compensation	Abnormal	Normal % VR	25-50%	Ratio of Average	=====	=====	1 to 1.65	160 %
		Interpretation	Very Low *	Expected			1 to 1.20	

Impression - V02 Score measure oxygen utilization throughout the body. O2 Utilization is dependent upon cardiac and pulmonary function, cardiovascular system, perfusion rates, muscles, and mitochondrial numbers and health

- Mr. Smith is a 49 yrs old male. His main complaint is dyspnea. He was tested using a Korr Medical Gas Analyzer, used for testing Cardiorespiratory Health and Aerobic Fitness.
 - Patient's Score of O2 Utilization (V02) was **Fair** by standards from Cooper Clinic, 85% or greater of predicted is considered normal (non pathological). Patient scored 94.6%
 - O2 Pulse (indicator of Stroke Volume) is 120% of Predicted / 85% of Predicted is considered normal or above expected
 - Heart Rate Reserve should range from 0-15%, patient had a 8.8 %, which is normal. Heart rate response to exercise (Chronotropic Response to Exercise) was normal. His heart rate increased as expected to the workload.
 - Ventilatory Efficiency / Compensation : The RER, the Ventilatory Ratio between Ve/V02 to VC02 is Abnormal, showing an increased ventilation of 39.73% above normal for removing CO2 from the body. He was unable to remove CO2 effectively. He had a very low Respiratory Reserve of 5.73 %, which would be expected from poor compensation
 - He had an Aerobic heart rate capacity of 94 beats, in which he used 79 beats. His Heart Rate Reserve was 8.8 %. He was unable to use the full capacity of his heart rate due to poor Ventilatory compensation. Test ended d/t shortness of breath
- Summary - Pulmonary Limitation - It is quite possible this limitation was due to hyperventilation. This is a common cause. But it appears more likely the patient has a mild to moderate lung disease, more likely restrictive.

Recommendations: Patient should follow the exercise recommendations we have provided below for 12 weeks and then retest. Further, patient should consult with Pulmonologist for testing and assessment to determine if any pulmonary issues exist.

V02 Score	36.9 - Fair	B
Heart Health	Heart Rate Response to Workload: Normal	A
	Heart Rate Reserve: 8.8 % - Normal	A
	Heart - Stroke Volume: 120 % of Predicted	A+
	Heart Rate Recovery: 13 beats	C
	Functional Health : 21% above	A
	Cooper Cardiorespiratory Health : 94%	B
Respiratory Health	RER - VC02 / V02 - .56 to .68	D
	Ratio of Ve/V02 to Ve/VC02 : 1 to 1.6	D
	40% > than normal ventilatory requirement for Ve/VC02	D
	Ventilatory Reserve - 5.73% Very Low	D
Assessing Surgical Risk Level According to V02 Peak	In general, a peak VO2 greater than 20 ml/kg/min is associated with a low risk for perioperative morbidity and mortality after non-cardiac surgery. The two parameters that should be taken into consideration are the maximum oxygen consumption (VO2max) and the efficiency slope (VE/VC02). (NIH) VE/VC02 Efficiency Slope - Normal <34	D V02 Max Score - 36.9 Ve/VC02 Score is 33 <u>Pulmonary Assessment Recommended Prior to Surgery</u>

Resting Metabolic Rate

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Insulin Resistance Risk Level (Hyperinsulinemia)

Insulin Resistance Signs and Symptoms	<input type="checkbox"/> Cravings and Hunger	<input type="checkbox"/> Darken Skin	Risk Level
	<input type="checkbox"/> Obesity	<input type="checkbox"/> Fatty Liver	
	<input type="checkbox"/> Fatigue	<input type="checkbox"/> Hypertension	
	<input type="checkbox"/> Dyslipidemia	<input type="checkbox"/> Pre-diabetes / Diabetes	
	<input type="checkbox"/> PCOS	<input type="checkbox"/> Heart Disease	

Modern Disease Risk Level

Metabolic and Cardiovascular Risk Assessment Levels	Metabolic Disorders	Cardiorespiratory Risks	Risk Level
	<input type="checkbox"/> Fatty Liver <input type="checkbox"/> Hypertension (Stage) <input type="checkbox"/> Pre-Diab. / Diabetes A1C <input type="checkbox"/> Heart Disease CAC <input type="checkbox"/> Obesity / Kidney GFR	<input type="checkbox"/> Hypertension <input type="checkbox"/> COPD <input type="checkbox"/> Dyslipidemia <input type="checkbox"/> Heart Attack / Stroke <input type="checkbox"/> Cardiovascular Dx	Metabolic - Low CardioResp. - Low

Exercise Prescription

Heart Rate - METS - Work Zones - V02 Test		
Maffetone's "180 Formula" 180 minus age = 131 bpm Your Aerobic Target Area 131 bpm	Aerobic Capacity Recommended HR: 116 - 140 bpm 65% of PMHR : 101 bpm 65% V02 36.3 / 6.8 METS Recommended 5 - 6.8 METS 3x wk	Anaerobic Capacity Recommended HR : 140 to 156 bpm 85% of Max HR : 132 bpm 85% of V02: 31.36 / 8.9 METS Recommended 8-9 METS 2x wk
Aerobic Exercise 150 mins. week (Recommended minimum ACSM)		Anaerobic Exercise 2x wk. 2-3 mins per Exercise

Information / Definitions

Predicted V02 Max Equation (PV02): 1st Predicted : Wasserman :Equation V02 Max 50 - (0.4 x age in years) - 2nd Predicted: "Good"

Cooper Table

Normal:VO2 : 85% or greater of predicted, meaning there appears to be nothing pathological, but does not mean healthy. 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. Cardiac output is what determines or limits exercise capacity in normal individuals.

- **Physical Work Load** : The ability to perform physical work is an important determinant of quality-of-life¹ and is enabled by an increase in oxygen uptake (VO₂).² During maximal aerobic exercise in healthy humans, VO₂ increases approximately 4-fold.² This is achieved by 2.2-fold increase in heart rate (HR), a 0.3-fold increase in stroke volume, and a 1.5-fold increase arteriovenous oxygen difference.² Thus, the increase in HR is the strongest contributor to the ability to perform sustained aerobic exercise.³ It is therefore not surprising that CI can be the primary cause or a significant contributor to severe, symptomatic exercise intolerance.
- **Mean Arterial Pressure** = $1/3*(SBP) + 2/3*(DBP)$ - MAP indication of global perfusion pressure necessary for organ perfusion and oxygen delivery. What is a Normal Mean Arterial Pressure? A normal MAP is between 70 and 100 mmHg. If the MAP drops below 60 mmHg, there is a concern there won't be enough pressure to perfuse vital organs including the brain. If the MAP is above 100 mmHg, the patient may be experiencing a high artery pressure / Shear Pressure

Heart Rate Predicted: $220 - \text{Age} = X$

- **O2 Pulse - Equation for Predicted Max O2 Pulse** : Predicted V02 divided by Predicted Maximum Heart Rate (PV02/PMHR)
 - Normal > 85% Predicted with no plateau / Abnormal <85% / <10 ml/beat High Risk (Cardio. Exercise Background etc. Herdy)
 - Minimal rise in O2 Pulse / Left Ventricle can be used as an indirect indicator of cardiac stroke volume A normal subject should achieve an O₂-pulse of more than 10ml/beat at peak exercise A plateau in the O₂-pulse at a low value implies limited cardiac output, either because of heart disease or disorders of the pulmonary circulation Don't over-interpret a low O₂-pulse, particularly if the VO₂max is normal. (Oxford Medicine Online)
- Heart Rate Reserve : Normal Subjects will reach 85% to 100% of their Predicted Max Heart Rate, or 0% to 15% Heart Rate Reserve High Heart Rate Reserves indicate some limiting factor(s).
 - 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
- **Chronotropic Insufficiency** : Heart Rate does not Rise Normally. Possible Beta Blockers or Sinoatrial node dysfunction.. Chronotropic incompetence is the inability of the heart to increase its rate according to the body's demand, mostly with increased activity. Chronotropic incompetence can reduce exercise tolerance and impair quality of life. It is an independent predictor of adverse cardiovascular events and all-cause mortality [1]. Though increase in stroke volume and oxygen extraction increases with exercise, the predominant contribution to increase in oxygen consumption (VO₂) is by an increase in heart rate. A reduced exercise heart rate is cited as the reason for age related decline in aerobic work performance [2]. Though the intrinsic rate of the sinoatrial node is around 100/min, resting heart rate is generally in the range of 60-80/min due the influence of the parasympathetic nervous system. Increased resting heart rate would mean either increased sympathetic activity or reduced parasympathetic tone. Increased resting heart rate has been associated with increased cardiovascular death, coronary artery disease and sudden cardiac death. Chronotropic incompetence may manifest as failure to achieve the predicted maximal heart rate, inadequate submaximal heart rate or as heart rate instability during exercise. Chronotropic incompetence may be noted in sick sinus syndrome, atrioventricular block, coronary artery disease and heart failure [3]. Heart rate recovery after exercise is due to withdrawal of sympathetic activity. Attenuated heart rate recovery after exercise has been shown to be a predictor of mortality independent of angiographic severity in coronary artery disease. This was from a study of 2,935 consecutive patients who underwent symptom limited exercise testing for suspected coronary artery disease and then had a coronary angiogram within 90 days. Follow up period was 6 years. A cut off value of 12 beats/min or less during the first minute after exercise has been given. In those undergoing stress echocardiography, cutoff was 18 beats/min or less [4]. Reactivation of vagal activity is an important mechanism

for heart rate recovery after exercise. It is blunted in patients with chronic heart failure while it is accelerated in athletes [5]. Chronotropic incompetence can be quantified by the chronotropic index. First the age predicted maximal heart rate is estimated using the Astrand's formula as $220 - \text{age in years}$ [6]. Then the chronotropic index is calculated as follows [7]:

Chronotropic index = (peak heart rate – resting heart rate)/(age-predicted maximal heart rate – resting heart rate)

- **Ventilatory Reserve:** Normal Ventilatory Reserve should be > 20%, and is typically between 30-50%
 - Breathing reserve (VE/MVV): represents the ratio between maximal ventilation during exercise (VE) and maximum voluntary ventilation (MVV) at rest, both variables in L/min. Equations to predict MVV can be used (forced expiratory volume in the first second – FEV1 x 40), although it can be measured directly on pre-test spirometry. Lung Mechanics are not a limiting factor in healthy individuals.
- **Ve/VO2:** The VE/VO2 reflects the ventilatory need for a certain O2 consumption level, being, thus, an index of ventilatory efficiency. Patients with inadequate ratio between pulmonary ventilation and pulmonary perfusion (increased physiological dead space) ventilate inefficiently and have high VE/VO2 values (pulmonary disease and HF). Peak values above 50 have been useful to diagnose patients suspected of having mitochondrial myopathy
- **Ve/VCO2:** VE/VCO2 Ventilatory efficiency, as indicated by the increment in minute ventilation (VE) relative to CO2 production (VCO2), reflects right ventricular-pulmonary vascular (RV-PV) function during exercise. The VE/VCO2 slope reflects the severity and prognosis of patients with HF, pulmonary hypertension, HCM, COPD and restrictive pulmonary disease. Slope Values >34 are considered abnormal. Normal < 30 - Poor >34 Pathological Significance >39
 - With anaerobic metabolism, lactic acid generated, is neutralized by bicarbonate (HCO3), resulting in increased CO2 production. Hence VCO2 rises more than VO2 in anaerobic exercise. This further burdens the ventilatory system, which must eliminate this excess CO2
 - Results: Cardiopulmonary morbidity and mortality rates were 23% (51 patients) and 2.2% (5 patients). The 25 patients with respiratory complications had a significantly higher VE/VCO2 slope than those without complications (34.8 vs 30.9, p=0.001). Peak VO2 was not associated with respiratory complications. Logistic regression and bootstrap analyses showed that, after adjusting for other baseline and perioperative variables, the strongest predictor of respiratory complications was VE/VCO2 slope (regression coefficient, 0.09; bootstrap frequency, 89%; p=0.004). Patients with a VE/VCO2 slope exceeding 35 had a higher incidence of respiratory complications (22% vs 7.6%, p=0.004) and mortality (7.2% vs. 0.6%, p=0.01).

Conclusions: VE/VCO2 slope is a better predictor of respiratory complications than peak VO2. This inexpensive and operator-independent variable should be considered in the clinical practice to refine operability selection criteria.

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- **Heart Rate Recovery:** Superior > 20 beats in 1st min // Excellent > 18-20 beats in 1st min // Good >15 beats in 1st min Fair > 12 // Poor < 12 beats in 1st min.
 - Low Heart Rate Recovery Associated Risk with Mortality, New Onset Atrial Fibrillation Middle Age Adults, Diabetics, SMI - Silent Myocardial Ischemia, Increased Risk for Hypertension in Men, General Ischemia, Heart Disease - increased risk 3 - 5 % a year
- **Aerobic Capacity:** Cardiorespiratory Fitness and Health (CRFH) is directly related to the integrated function of numerous systems, and it is thus considered a reflection of total body health. CRFH (V02 Max). It is the most important correlate to health. It is the strongest predictor of cardiovascular disease and all cause mortality. V2 Max Testing is the single most important test for health assessment, prevention, and improvement. Everyone capable of testing should be tested. Moderate to high levels of CRF and improvement in CRF are associated with a lower risk of mortality from all-causes and CVD in both men and women regardless of age, smoking status, body composition, other risk factors, method of CRF assessment, and study design. CRF appears to attenuate the higher risk of death associated with obesity although it is not yet clear whether CRF completely eliminates mortality risk in obese individuals

- VO₂ is an index of the body's efficiency at producing work. It is expressed in milliliters of oxygen consumed per minute, and adjusted for body weight in kilograms: ml/kg/min. There are many factors that can influence VO₂max, e.g. heredity, training, age, gender, and body composition. Generally, VO₂max declines with age (about 2% per year after age 30) and males typically have a greater oxygen consumption value than females. Nevertheless, the trend is that a higher VO₂max allows one to produce more energy, thereby performing more work. With this in mind, VO₂max is the "gold standard" measure of overall fitness (Cooper)

Respiratory Metabolism and Disorders

- **Ventilatory Impairment**
 - **Obstructive Impairment**
 - Obstructive lung diseases are characterized by an obstruction in the air passages, with slow and shallow **exhalation**.
 - Obstruction can occur when inflammation and swelling cause the airways to become narrowed or blocked, **making it difficult to expel air from the lungs**. This results in an abnormally high volume of air being left in the lungs (i.e., increased residual volume). This leads to trapped air and lung hyperinflation—changes that contribute to worsening respiratory symptoms.
 - The following lung diseases are categorized as obstructive:
 - Chronic obstructive pulmonary disease (COPD), Chronic bronchitis, Asthma, Bronchiectasis, Bronchiolitis, Cystic fibrosis
 - **Restrictive Impairment**
 - In contrast to obstructive lung diseases, restrictive conditions are defined by difficulty filling the lungs with air during inhalation. Restrictive lung diseases are characterized by a reduced total lung capacity.
 - Restrictive lung diseases can be due to either intrinsic, extrinsic, or neurological factors.
 - Intrinsic Restrictive Lung Diseases. Intrinsic restrictive disorders are those that occur due to restriction in the lungs (often a "stiffening") and include:
 - Pneumonia, Pneumoconioses, Acute respiratory distress syndrome (ARDS), Eosinophilic pneumonia, Tuberculosis, Sarcoidosis, Pulmonary fibrosis and idiopathic pulmonary fibrosis, Lobectomy and pneumonectomy (lung cancer surgery), Extrinsic Restrictive Lung Diseases
 - **Extrinsic restrictive disorders refer to those that originate outside of the lungs. These include impairment caused by:**
 - Scoliosis, Obesity, Pleural effusion, Malignant tumors, Ascites, Pleurisy, Rib fractures,
 - **Neurological Restrictive Lung Diseases**. Neurological restrictive disorders are those caused by disorders of the central nervous system that interfere with movements necessary to draw air into the lungs.
 - Among the most common: Paralysis of the diaphragm, Guillain-Barré syndrome, asthenia gravis, Muscular dystrophy, Amyotrophic lateral sclerosis (ALS or Lou Gehrig's Disease)
- **Hypercapnia** - Hypercapnia, also called hypercarbia, is when you have too much carbon dioxide (CO₂) in your blood. Your body creates CO₂ when your cells make energy. Your red blood cells carry it from your organs and tissues to your lungs, where you breathe it out.
 - The etiology can be extensive, but it can be helpful to divide the potential causes into three groups: decreased minute ventilation, increased physiologic dead space, increased carbon dioxide production.
 - **The first group** is anything that causes decreased minute ventilation (respiratory rate x tidal volume). The central respiratory center in the medulla takes feedback from multiple inputs and integrates them into a respiratory drive, which functions to control our minute ventilation. Anything that affects the central respiratory center can affect the minute ventilation. Notable etiologies include overdose of sedative medications (narcotics, benzodiazepines, tricyclic antidepressants, etc.), stroke, and hypothermia. Although the medulla functions to control the respiratory drive, many peripheral nerves and respiratory muscles are needed to perform respirations. Decreased respiratory neuromuscular function can decrease minute ventilation. Notable etiologies include Guillain-Barre, myasthenia gravis, amyotrophic lateral sclerosis, myositis, multiple sclerosis, phrenic nerve injury, tetanus, botulism, organophosphates, and ciguatera. Deformity of the thoracic cage can impact tidal volumes, therefore decreasing minute ventilation.

- **The second group** is anything that increases physiologic dead space (part of the lung that does not participate in gas exchange); this is ventilation without perfusion. This condition can be due to pulmonary capillary compression (positive pressure ventilation) or the destruction of pulmonary capillaries (pulmonary vasculitides, COPD, asthma, interstitial lung disease). A large pulmonary embolism can also cause significant dead space.
- **The third group** is anything that increases CO₂ production. It is more likely that this group only partially contributes to hypercapnia and is not commonly the primary cause but can occur in conditions that increase metabolic rate, sepsis, thyrotoxicosis, or fever.
- Hypercapnia commonly causes respiratory acidosis. CO₂ combines with H₂O to form H₂CO₃, which dissociates into H⁺ and HCO₃⁻. This buffer equation is in constant flux. Giving patients with hypercapnia supplemental bicarbonate will worsen their condition if not adequately ventilating. The supplemental bicarbonate will push the acid/base buffer equation towards increased CO₂ production; however, if the patient's ventilation is inadequate, the equation will move back towards more H⁺ production, worsening the acidosis. With respiratory acidosis, the kidneys try to compensate by increasing H⁺ secretion, raising the HCO₃⁻ concentration, assuming the patient has adequate kidney function. In acute respiratory acidosis, the serum HCO₃⁻ increases 1 mEq/L for every 10 mmHg elevation in PaCO₂. If PaCO₂ remains elevated for three to five days despite compensatory mechanisms, it is considered chronic respiratory acidosis. In this state, the serum HCO₃⁻ increases from 3.5 to 5 mEq/L for every 10 mmHg elevation in PaCO₂
- This is estimated from the ventilatory equivalent of carbon dioxide, which is the ratio of minute ventilation to carbon dioxide production, VE/VCO₂. A normal value is around 25-30, and increases in the ratio reflect impairment of V/Q mismatch, either from respiratory causes or from impaired cardiac function
 - A higher VE/VCO₂ ratio describes a greater ventilatory requirement for eliminating the CO₂ produced by aerobic metabolism and defines a reduced ventilatory efficiency. The reduced ventilatory efficiency is therefore caused by an increase in physiological dead space and a reduced PaCO₂ set-point
 - This supports the hypothesis that pulmonary vascular resistance is a determinant of the VE/VCO₂ slope.
 - That the volume of oxygen consumed (VO₂) by the body is equal to the difference between the volumes of inspired and expired oxygen. 2. That the volume of carbon dioxide produced (VCO₂) by the body is equal to the difference between the volumes of expired and inspired carbon dioxide.
- **Ventilation/perfusion mismatching**
 - The ratio of ventilation (V) to perfusion (Q) is decisive for the quality of the gas exchange in the lungs. Pronounced ventilation/perfusion mismatch (V/Q) occurs in pulmonary disease, pulmonary vascular disorders and heart failure

24-H MAP Categories	24-H MAP Thresholds, mm Hg
Normotension	<90
Elevated BP	90 to <92
Stage-1 HT	92 to <96
Stage-2 HT	≥96