VO2Max Improvement of 96% in a Non-EliteRecreational Athlete over 24 Months

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Abstract:

VO2 max is a measure of athletic performance and is generally considered an excellent health parameter for athletic performance testing. Various methods are used to generate such a result generally using a treadmill or cycle ergometer. Improvements have been shown through training. The upper limit of such improvement has been difficult to find in the literature in part because testing often starts with individuals already at a superior level of performance. As genetics may play a significant role in an individual's VO2 max, training can only contribute a portion of the result. Humans have a range of maximal oxygen utilization with upper limits approaching 100 mL/kg. This case report demonstrates a 96% improvement over time secondary to selective intense training. The intent was to document objectively amelioration of the VO2 max using a defined protocol on standardized testing equipment. This may lead to more efficient training of individuals desiring performance improvement.

Keywords:
- cardiopulmonary stress test
- VO2 max
- exercise induced asthma
- aerobic exercise

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VO2 max is a measure of the maximal amount of oxygen that can be used at the upper limits of exercise. An increase in the VO2 max can be seen with training; however, generally there is a decline in maximal oxygen uptake capacity over time particularly after the third decade of life. This case documents an unusually large increase in VO2 max over 24 months. The training protocols used in this case report are presented that may be useful to other professionals seeking to improve their patients' performance.

CASE REPORT

A 43-year-old, nonsmoker, Caucasian male presented in July 2007 for a voluntary baseline cardiopulmonary stress test. He felt that he was "in good shape" and wanted to obtain an objective assessment to confirm his impression. Remaining physically active, he also followed an exercise regimen of mildweight training thrice a week and indoor rock climbing once a week. Though never a competitive athlete, he did well in high school track and field events but never pursued this to a higher level. Weight was stable at approximately 165lbs (75kg) at a height of 71.5inches (182cm) with a body mass index (BMI) of 22.7kg/m^2. These parameters were unchanged over the course of the study (± 1kg). Diet was pescetarian, he denied any alcohol or tobacco use and supplemented only with multi-vitamins at the beginning of the evaluation period. He was taking no medications and was under no physician's care for any medical problems. There was a possible history of exercise induced asthma which, in fact, was demonstrated through the testing but did not limit his activity.

As this was initially a personal evaluation of his health metrics, no institutional ethics or study registration/disclosures were required. The patient was tested using a bicycle ergometer protocol available at the performance laboratory. He was monitored for blood pressure, pulse, subjective intensity using a modified Borg's scale, oxygen saturation, cardiac rhythm as well as the closed loop pulmonary function testing equipment. The initial protocol involved a ramping increment of 20 W/minute increasing until exhaustion. His effort was excellent and followed the protocol assiduously. Work capacity (VO2 max) was calculated at 27.6 mL/kg with a normal anaerobic threshold. Total wattage was 299. The forced expiratory volume (FEV) 1% fell by 12% suggesting mild obstructive pulmonary disease. Blood pressure was escalated to 220/100 over the course of the testing. He reached 103% of maximal calculated heart rate.

Since the result was significantly discordant to his expectation, he elected to start a training regimen to see if he could improve his results on future testing. Following the suggestions of the consulting cardiopulmonary physician, a program was designed incorporating 20 minutes of jogging thrice a week keeping his heart rate at approximately 140 bpm. He also continued the 90 minutes of rock climbing once a week. Finally, thrice a week, he started weight lifting at a higher intensity level than previously for 20 minutes persession. No type of exercise was done 2 days consecutively. Diet remained the same.

After six months of training, a repeat testing was done on the same equipment. The ramping protocol was set at 25W per minute. There was a similar blood pressure escalation with maximal exertion. His VO2 max improved to 32.9 mL/kg.This represented a 19% improvement over baseline.

Further restructuring of the training protocol included increasing the running target heart rate to between 140 and160 bpm. Some longer runs were added as well as a few bicycle interval workouts but these were infrequent. The patient remained compliant to instructions. There was no change in the rock climbing and weight training exercises.

The third test, now 1 year later, showed a blood pressure rise to a maximum of 206/92. VO2 max was measured at 42 mL/kg; a 52% improvement. The ramping protocol was the same as was the equipment.

In an effort to improve his results further, additional training modifications were instituted. Though the total volume of exercise was unchanged, intensity was further increased. On the run days, 1 day per week was designated as an interval training day done in the following fashion: 2 minutes of high intensity (up to 104% of maximal calculated heart rate) followed by 2 minutes of recovery (less than 67% of maximal calculated heart rate) in an alternating fashion for 22 minutes per session. The other 2 days per week involved keeping the heart rate just under anaerobic threshold for the full 22 to 26 minutes of training. In this patient's situation that calculated out at 162 bpm, the weight training and rock climbing protocols were left unchanged. This continued for 6 months.

On the next test, on a Cardinal Health Encore 229 unit with Cardiosoft coupled to a Ergoline pedal ergometer on a 25 work watt per minute ramping protocol 18 months after initiation of evaluation, his VO2 max was found to beat 50.7 mL/kg/min representing a 84% improvement over baseline. Maximal blood pres-

sure was 212/95, maximal heart rate was 181 bpm or 106% of calculated maximum and work watts were 309.

Final testing was done 6 months later after continued training without a significant change in volume or intensity. Using the same protocol, the VO2 max returned a value of 54.1 mL/kg/min and work wattage at 318. Over the course of the 24 months of testing, there was a 96% improvement in VO2 max.With the arthroscope placed in a posterior portal, visualization of the subscapularis tendon and the bone bed on the lesser tuberosity was excellent. The field of view could be varied as needed by abduction and internal or external rotation of the shoulder for better exposure of the lesser tuberosity. In general, internal rotation was useful in enhancing visualization of partial tears by relaxing the intact portion of the subscapularis. For partial tears, the percentage of tendon that was torn was estimated from the superior-to-inferior dimension of the "bare footprint," or bone bed, on the lesser tuberosity from which the tendon had torn.

We have performed an anatomical study (Tehrany AM, Burkhart SS, Wirth MA, unpublished data) on 19 cadaver shoulders in which we found that the average length of the subscapularis footprint from superior to inferior was 2.5 cm (range, 1.5 to 3.0 cm). In calculating the percentage of subscapularis tendon that was torn in a partial tear, we would measure the length of the bare footprint and divide it by the length of the complete footprint (2.5 cm). For example, a partial tear with a bare footprint of 1 cm would comprise a 40% tear (1.0 cm/2.5 cm = 0.4, or 40%).

In the case of a chronic retracted subscapularis tear, the tendon edge is often located far medially, at the level of the glenoid rim, and it can be difficult to recognize. We have found it useful to employ a tendon grasper to pull on the medially retracted tissues until we can positively identify the upper border of the subscapularis (Fig 3). Another trick to finding the retracted subscapularis is to identify what we call the "comma sign." In retracted subscapularis tears, the medial sling of the biceps, composed of the superior glenohumeral ligament and a portion of the coracohumeral ligament, is torn from the humerus at the upper border of the subscapularis footprint and remains attached to the superolateral portion of the subscapularis, forming a comma-shaped arc just above the superolateral corner of the subscapularis (Fig 4).

For partial tears of the biceps, debridement or tenotomy were performed. For dislocations or subluxations of the biceps, arthroscopic tenotomy or arthoscopic tenodesis were performed. In the senior author's experience with open repair of subscapularis tears in association with dislocation of the biceps, attempts to preserve the biceps by relocating it and stabilizing it within the bicipital groove have not been successful due to redislocation of the biceps causing disruption of the subscapularis repair. Therefore, we have not attempted to relocate and stabilize the biceps arthroscopically.

With traction being exerted on the subscapularis by means of traction sutures, an arthroscopic elevator was brought in through the anterolateral portal to mobilize the anterior and posterior aspects of the subscapularis (Fig 5). In the case of isolated tears of the subscapularis, the muscle-tendon unit was generally not retracted and did not require mobilization. However, the massive combined tears that comprised all of the subscapularis tendon plus the supraspinatus and infraspinatus tendons generally required mobilization. If the tendon could not be pulled easily over the bone bed on the lesser tuberosity, it would be mobilized on its anterior, posterior, and superior aspects with an arthroscopic elevator while pulling on the tendon with traction sutures. The inferior border of the tendon was avoided to minimize the chance of neurovascular injury. By freeing all except the inferior border, traction on the tendon would effectively disrupt any adhesions inferiorly.

Next, the bone bed on the lesser tuberosity was prepared by means of a high-speed burr through an anterolateral portal (Fig 6).

To maximize tendon-to-bone contact, the bone bed was frequently medialized 5 mm by removing articular cartilage to a bleeding base of bone. After bone bed preparation, 1 of 2 fixation methods was used. For complete tears with enough elasticity to pull easily past the bone bed and with adequate visualization to see both the anterior and posterior surfaces of the tendon, a Parachute Anchor (Arthrex, Naples, FL) was placed by transtendon insertion. This implant had a biodegradable disk that compressed the tendon against the bone bed. It was particularly useful in this confined space because it obtained fixation without the need for knot tying. For tendons that reached to the bone bed but could not be overpulled beyond the bone bed, transtendon fixation was not used because it was felt that the implant would make a hole too close to the distal end of the tendon to ensure secure fixation. In these cases, standard screw-type anchors (Corkscrew, Arthrex) were placed (Fig 7), followed by suture passage through the tendon by standard suture passers or shuttle techniques (Fig 8). For large complete subscapularis tears that required mobilization, we used a "traction shuttle" repair technique, in which we passed the braided sutures from the anchor through the ten-

don by threading them through a loop on the traction suture, then pulled the traction suture through the tendon so that it "shuttled" the braided sutures through the tendon with it.

The sutures were then retrieved and brought out through an anterolateral portal, through which arthroscopic knot tying was accomplished 5,6 (Fig 9). For complete tears, 2 anchors were used, and for tears of the upper half of the tendon, 1 anchor was used (Fig10). After passage of the suture through the tendon, before knot tying, retrieval of the transtendon suture limb could be difficult because of poor visualization caused by deltoid swelling. In those cases, the suture limb was threaded through the lumen of a single-hole knot pusher which then delivered the suture into the joint where it could be easily retrieved.

After subscapularis repair was completed, patients with combined multitendon rotator cuff tears extending more posteriorly underwent subacromial smoothing, with preservation of the coracoacromial ligament, followed by arthroscopic suture anchor repair of the rest of the tear.

Postoperatively, the patients were immobilized in a padded sling for 6 weeks with the shoulder in 30° of internal rotation. External rotation beyond neutral was avoided for 6 weeks, as was any attempt at active or passive overhead motion. At 6 weeks postoperatively, overhead motion was initiated. Resisted isotonic strengthening began at 10 weeks postoperatively.

SUMMARY AND DISCUSSION

VO2 max represents the maximum ability of an individual to utilize oxygen during exertion. A high VO2 max, therefore, correlates with better endurance during activity. While VO2 max is an important component of an individual's ability to excel in sports, technique, personal psychological motivators, fatigue, nutrition, etc., may also impact on performance. Therefore, it is important to place the VO2 max result in context.

Maximal oxygen consumption varies among different sports and individuals as measured in professional athletes. For example, VO2 max is lowest in shot put on one end of the spectrum and highest in cross country skiing. It is not clear if one sport attracts a specificVO2 max class of athletes or if sport specific training generates a specific VO2 max.

Peak VO2 max is reached by age 19 years with a reduction over time.[1] Generally, the literature suggests a 5 to 10% reduction over each subsequent decade. Genetics may play a significant component of an individual's VO2 max with a contribution of 0 to 50% or more. However, identical twin studies have not all been supportive of a strong hereditary effect.[2] Furthermore, even adopted family members can also have close concordancy.[3] Summarizing Bouchard et al,[4] it would appear reasonable to accept a 25% genetic contribution.

Training has been clearly shown to improve the VO2 max. This was likewise demonstrated in this patient. Though it tends to decrease with age (generally 1% per year), aerobic activity tends to mitigate that drop. Interestingly, maximal heart rate does not drop with continued training.[5] Weight loss will increase the VO2 in an individual by definition (mL/kg/min); however, in this case report, his weight was stable. Note that this patient did not use any β-agonists, leukotriene inhibitors, or steroid therapy though he carried a diagnosis of exercise induced asthma. Intermittent supplementation admitted to included α-lipoic acid, L-acetyl carnitine, coQ10, Rhodiola rosea, and multivitamins. Their contribution, if any, is unclear to the documented improvement but should be considered as an unknown variable.

Humans have approached a VO2 max of 100 mL/kg/min. Women tend to have approximately 10 to 25% lower VO2 max than men depending on their training, perhaps related to their increase in type I fibers, as well as smaller diameter fibers over males. Thoroughbred horses may have a VO2 max of 180 mL/kg/min,[6] while sled dogs have been shown to have VO2 max in the range of 240 mL/kg/min.[7]

This interesting case demonstrated a large increase in VO2 max demonstrated objectively over time. The training protocols were described in detail; trainers and advisors may want to try them on their subjects to see if the results are reproducible. Various protocols have been promulgated in training literature; however, anecdotal improvements have often been the norm rather than carefully documented outcomes. It is imperative to follow a known metric since speed or lap times have shown to improve despite a stable VO2 max.This is likely secondary to improved muscular efficiency or mechanical aids, such as newer swimsuits and biking technology amelioration. Performance is also sensitive to other influences, such as fatigue, altitude, nutrition, and equipment. Of note, approximately four months into the training, the patient injured his knee (likely a meniscal tear unrelated to the training and it was not surgically addressed); however, this did not affect the training or results.

Improvements in VO2 max have been shown in studies using various protocols, including high intensity inter-

val training[8], 85% maximal heart rate for 20 minutes thrice a week[9], hypoxia techniques[10], and combination aerobic and weight training[11], which generally show up to 6% improvement. One research protocol increased total amount of exercise per week to 300 minutes found an increase in VO2 max approaching 50%.[12] The case reported here demonstrates an unusually high increase of 96% which suggests the need to further explore parameters resulting in increased performance.

CONFLICT OF INTEREST

None.

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