Journal of Neonatal Surgery
ISSN(Online): 2226-0439
Vol. 14, Issue 12s (2025)
https://www.jneonatalsurg.com

OPEN ACCESS

Physiological Impact of One-Month Alcohol Abstinence on Heart Rate, Oxygen Saturation, Maximal Oxygen Uptake, Pulmonary Function, and Muscle Strength in a Young Adult Male: A Case-Based Analysis

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Cite this paper as: JaeHo Yu, (2025) Physiological Impact of One-Month Alcohol Abstinence on Heart Rate, Oxygen Saturation, Maximal Oxygen Uptake, Pulmonary Function, and Muscle Strength in a Young Adult Male: A Case-Based Analysis. *Journal of Neonatal Surgery*, 14 (12s), 1165-1168.

ABSTRACT

This case study investigated the physiological effects of a four-week period of alcohol abstinence on cardiopulmonary and muscular function in a healthy young adult male with a habitual moderate drinking pattern. The subject, a 26-year-old male who engaged in regular aerobic and resistance training, underwent baseline and post-intervention measurements of resting heart rate, peripheral oxygen saturation (SpO2), maximal oxygen uptake (VO2max), pulmonary function (FVC, FEV1), and muscular strength (grip strength and leg press 1RM). Post-intervention assessments demonstrated meaningful improvements in all parameters, indicating autonomic nervous system recovery, enhanced pulmonary function, and improved muscle recovery. The findings provide preliminary evidence supporting the physiological benefits of short-term alcohol cessation, even among physically active young adults.

1. INTRODUCTION

Alcohol consumption exerts complex physiological effects across multiple body systems, particularly the cardiovascular, autonomic nervous, respiratory, and metabolic systems. While light to moderate alcohol intake has occasionally been associated with short-term vasodilatory benefits, cumulative evidence highlights its long-term deleterious impacts, including elevated heart rate, hypertension, reduced heart rate variability (HRV), and impaired myocardial function [1-3].

Alcohol stimulates central GABAergic pathways and activates the sympathetic nervous system, leading to increased vascular tone, elevated resting heart rate (tachycardia), and peripheral vascular resistance [4]. Chronic intake induces negative inotropic effects, myocardial structural remodeling, and reduced cardiac output [5]. Observed reductions in heart rate following abstinence likely reflect restored sympathovagal balance and improved cardiac autonomic regulation, which can also be indexed by enhanced HRV−a well-validated predictor of cardiovascular resilience [6].

Alcohol also compromises oxygen transport and utilization. Acute intake transiently reduces hemoglobin oxygen saturation, while chronic exposure impairs peripheral circulation, capillary perfusion, and mitochondrial oxidative capacity, all contributing to decreased VO2max [7,8]. Moreover, alcohol exacerbates sleep apnea and upper airway inflammation, leading to decreased SpO2 and pulmonary function, thereby further undermining exercise performance.

In contrast, alcohol cessation may reverse these processes by improving alveolar gas exchange, restoring mitochondrial function, and enhancing parasympathetic modulation. These adaptations can lead to observable improvements in VO2max, SpO2, and pulmonary efficiency, thereby enhancing physical performance in both athletic and general populations.

While most alcohol-related studies focus on liver function or addiction treatment, fewer have quantitatively explored cardiopulmonary and neuromuscular improvements in healthy individuals following abstinence. This study addresses that gap by evaluating short-term physiological responses to alcohol cessation in a physically active young adult male.

2. METHODS

2.1 Participant Selection Criteria

A single healthy 26-year-old male meeting the following inclusion criteria was enrolled:

- Age 20‑30 years
- Consistent alcohol intake (1‑2 sessions/week for 6+ months)
- Regular physical activity (3+ sessions/week including aerobic and resistance training for 6+ months)
- Willingness and capacity to abstain from alcohol for 4 weeks

Exclusion criteria included:

- Chronic illness (hypertension, cardiopulmonary, or musculoskeletal disease)
- Medication or substance use within 3 months
- Sleep disorders, smoking, or obesity (BMI ≥ 30)

The participant was a personal acquaintance of the investigator and provided informed verbal and written consent. Routine lifestyle and exercise patterns were maintained during the intervention period.

2.2 Measurements and Instruments

2.2.1 Resting Heart Rate Measured using the Omron HEM-7120 automated sphygmomanometer following a 5-minute rest in a quiet room. Two readings were averaged to enhance reliability.

2.2.2 Peripheral Oxygen Saturation (SpO2) Assessed using a fingertip pulse oximeter (Contec CMS50D) on the right index finger. A 30-second stable reading was recorded.

2.2.3 Maximal Oxygen Uptake (VO2max) Estimated via the YMCA Submaximal Cycle Ergometer Test based on heart rate responses to graded workloads.

2.2.4 Pulmonary Function (FVC, FEV1) Measured using a handheld spirometer (Vitalograph Micro). Forced expiratory maneuvers were performed following maximal inspiration. FVC and FEV1 were recorded to assess lung capacity and airway function.

2.2.5 Muscular Strength Grip strength assessed using a digital handgrip dynamometer (Takei T.K.K. 5401). Lower limb strength was measured via 1-repetition maximum (1RM) on a leg press machine (BodySolid GLPH1100).

All tests were conducted in a controlled environment at the same time of day. The subject fasted for at least 12 hours and avoided caffeine or exercise for 6 hours prior to testing.

3. RESULTS

Following the four-week abstinence period, the subject's resting heart rate decreased from 82 to 72 bpm, indicating restored parasympathetic dominance and improved autonomic cardiovascular regulation. Peripheral oxygen saturation (SpO2) increased from 96% to 98%, reflecting enhanced alveolar gas exchange efficiency and potentially improved respiratory stability during sleep. Estimated VO2max improved from 85% to 93% of the predicted value, suggesting gains in cardiopulmonary endurance and exercise performance capacity.

Pulmonary function tests showed increases in both FVC (3.9L to 4.3L) and FEV1 (3.1L to 3.5L), consistent with improvements in bronchial dilation and lung elasticity. Muscle strength also improved: grip strength rose from 42 kg to 45 kg, and leg press 1RM increased from 120 kg to 130 kg, indicating enhanced protein synthesis and more efficient muscle recovery, likely due to reduced alcohol-induced suppression of anabolic pathways.

Parameter	Pre-Abstinence	Post-Abstinence	Change
Resting Heart Rate (bpm)	82	72	- 10 bpm
SpO2 (%)	96	98	2%
VO2max (est.) (%)	85	93	8%
FVC (L)	3.9	4.3	0.4 L

FEV1 (L)	3.1	3.5	0.4 L
Grip Strength (kg)	42	45	3 kg

4. DISCUSSION

This case study demonstrated that even a short-term (4-week) cessation of alcohol intake led to meaningful physiological improvements in a physically active young male. Decreases in resting heart rate, and increases in SpO2, VO2max, lung function, and muscular strength were observed, reflecting multi-system recovery from chronic moderate alcohol exposure.

The notable decline in heart rate suggests enhanced vagal tone and reduced sympathetic drive, aligning with prior findings on alcohol withdrawal and HRV enhancement [9,11]. Likewise, the increase in oxygen saturation and spirometry values (FVC, FEV1) underscores the pulmonary benefit of removing alcohol-induced inflammation and improving airway compliance [12-14].

VO2max gains are particularly noteworthy, given that it reflects an integrative measure of cardiac output, pulmonary efficiency, and peripheral oxygen utilization. The estimated 8% improvement is consistent with reversals in mitochondrial inhibition and cardiovascular strain documented in the literature [15-17].

Muscle strength enhancements may be attributed to improved protein synthesis, hormonal recovery (e.g., testosterone normalization), and reduced catabolic signaling (e.g., cortisol and mTOR suppression reversal) post-abstinence [18,19].

Despite its insights, this study has limitations: the single-subject design limits generalizability; measurement tools relied on submaximal or predictive tests rather than gold-standard assessments; and confounding factors such as nutrition and sleep were not strictly controlled.

Nevertheless, the findings reinforce the idea that alcohol abstinence, even over short durations, can facilitate measurable improvements in cardiovascular, respiratory, and musculoskeletal function in healthy adults.

5. CONCLUSION

A four-week alcohol cessation period elicited significant physiological benefits in a healthy young male, including decreased resting heart rate, improved pulmonary and aerobic function, and increased muscle strength. These findings support the integration of alcohol moderation strategies in health promotion, physical conditioning, and athletic performance enhancement programs.

REFERENCES

[1] Eckardt MJ, et al. (1998). Effects of moderate alcohol consumption on the cardiovascular system. *Alcohol Clin Exp Res*, 22(5), 998-1003.

[2] Piano MR. (2017). Alcohol's effects on the cardiovascular system. *Alcohol Res*, 38(2), 219-241.

[3] Suter PM. (2005). Alcohol and mortality: a review of prospective studies. *Br J Nutr*, 93(2), 227-232.

[4] Thayer JF, Lane RD. (2007). The role of vagal function in the risk for cardiovascular disease and mortality. *Biol Psychol*, 74(2), 224-242.

[5] Chen L, et al. (2009). Alcohol exposure induces inflammation in airway epithelial cells. *J Inflamm*, 6(1), 1-7.

[6] Davies MJ. (2000). Sleep apnea and alcohol. *Sleep Med Rev*, 4(6), 421-429.

[7] Barnes MJ. (2014). Alcohol: impact on sports performance and recovery in male athletes. *Sports Med*, 44(7), 909-919.

[8] Vella LD, Cameron-Smith D. (2010). Alcohol, athletic performance and recovery. *Nutrients*, 2(8), 781-789.

[9] Piano MR. (2017). Alcohol’s effects on the cardiovascular system. *Alcohol Res*, 38(2), 219-241.

[10] Klatsky AL. (2003). Alcohol and hypertension: a review. *J Hypertens*, 21(3), 381-386.

[11] Thayer JF, Lane RD. (2007). The role of vagal function in the risk for cardiovascular disease. *Biol Psychol*, 74(2), 224-242.

[12] Chen L, et al. (2009). Alcohol exposure induces inflammation in airway epithelial cells. *J Inflamm*, 6(1), 1-7.

[13] Davies MJ. (2000). Sleep apnea and alcohol. *Sleep Med Rev*, 4(6), 421-429.

[14] Sapkota A, et al. (2015). Alcohol consumption and pulmonary function. *Am J Epidemiol*, 182(8), 701-709.

[15] Steiner JL, Lang CH. (2015). Alcohol and mitochondrial function. *Am J Physiol Endocrinol Metab*, 309(6), E454-E466.

[16] Barnes MJ. (2014). Alcohol: impact on sports performance and recovery. *Sports Med*, 44(7), 909-919.

[17] Parr EB, et al. (2014). Alcohol impairs skeletal muscle protein synthesis. *PLoS One*, 9(2), e88384.

[18] Vella LD, Cameron-Smith D. (2010). Alcohol, athletic performance and recovery. *Nutrients*, 2(8), 781-789.

[19] Steiner JL. (2015). Alcohol reduces anabolic response to resistance exercise. *Med Sci Sports Exerc*, 47(6), 1206-1213.