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Could Nasal Breathing During Exercise Inhibit the Development of Cardiac Fibrosis and Arrhythmia Associated with Endurance Training? A Brief Literature Review with Theoretical Analysis

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Abstract: The increased incidence of myocardial scarring and atrial fibrillation in lifelong endurance athletes has been attributed to a dose-response relationship resulting from high-volume, high-intensity training carried out over the long term. However, this outcome is contradictory to and inconsistent with the well-established benefits of cardiovascular endurance training. In this short literature review along with theoretical analysis from previously published data, we propose that an athlete's breathing technique may play a role in this process. Based on current evidence, it is plausible that adapting to nasal-only breathing during exercise may be a viable strategy for endurance athletes to mitigate the relative hyperventilation created by breathing orally during exercise, and counter the conditions conducive to myocardial ischemia. Breathing nasally could increase myocardial blood flow at a given exercise intensity compared to breathing orally, with the most significant effects likely to occur at the highest intensities of exercise, and thereby prevent pathogenic myocardial changes. In particular, the higher pulmonary end-tidal carbon dioxide values seen while using nasal breathing during exercise suggest that arterial carbon dioxide may be relatively higher in the nasal breathing condition, thereby acting as a vasodilator to increase myocardial perfusion. Consequently, a nasal breathing approach during exercise might attenuate the effects of exercise-induced myocardial vasoconstriction and ischemia produced by breathing orally which is a probable mechanism for the increased myocardial fibrosis and arrhythmia seen in endurance athletes without concurrent cardiovascular disease, making it a topic worthy of increased research focus.

Keywords: Respiration, Myocardial Ischemia, Atrial Fibrillation, Endurance Exercise Training

1. Introduction

Despite a large body of scientific research that supports a positive relationship between physical exercise and cardiovascular health (Pinckard *et al.,* 2019), long-time endurance athletes have an increased incidence of myocardial scarring and atrial fibrillation (La Gerche, 2013; van de Schoor et al., 2016) compared to controls. In the subjects studied, these athletes typically have no prior history of congenital or hereditary cardiac abnormalities, nor do they present with classic symptoms of coronary artery disease, although rates of atrial fibrillation are elevated in comparison to controls. (Eijsvogels *et al.,* 2018; La Gerche, 2013; van de Schoor *et al.,* 2016). Additionally, while the mortality risk associated with high fitness levels is lowest in the fittest groups, some evidence suggests mortality may increase to some degree in those individuals doing the most exercise compared to their more moderately trained counterparts (Eijsvogels *et al.,* 2018). The World Health Organization, currently recommends that adults aged 18-64 engage in at least 150 minutes of moderate-intensity aerobic activity or 75 minutes of vigorous-intensity aerobic activity per week. However, individuals engaging in exercise volumes exceeding 10 times these guidelines appear to exhibit increased mortality risk, indicating a potential threshold where

the benefits of exercise may diminish or even reverse (Eijsvogels *et al.,* 2018).

To date, the scientific community's only explanation for this seemingly adverse high training load-associated finding, which contrasts with the well-documented benefits of endurance exercise in general, is that there must be a dose-response relationship whereby excessively high-intensity and/or high-volume training when carried out over the long-term, creates damage to the myocardium potentially engendering the arrhythmia problems associated with this phenomenon observed in some individuals.

It is theorized that a lifetime accumulation of heavy exercise may induce significant myocardial stress in some athletes, although the exact mechanism and time frames underlying this effect are as yet undetermined. However, the majority of studies addressing this topic compare both amateur and professional athletes between 30 – 60 years of age with greater than 10 years participation in endurance sports with untrained control groups of similar age (Małek & Bucciarelli-Ducci, 2020). By contrast, some evidence suggests that seemingly non-pathogenic cardiac remodeling occurs after a single year of marathon training in previously untrained runners (Arbab-Zadeh *et al.,* 2014). However, it is still unclear what the minimal stimulus might be for pathogenic fibrosis to begin occurring.

The "Extreme Exercise Hypothesis" (La Gerche, 2013) suggests that endurance training can sometimes induce cardiac remodeling and fibrosis which then manifests clinically as cardiac arrhythmia and fibrillation (Eijsvogels *et al.,* 2018).

Although this phenomenon is acknowledged in the medical community, to date no evidence-based upper duration limit or intensity threshold for exercise training has been set that delineates safe amounts of training versus training that potentially produces increased risk for adverse pathogenic cardiac remodeling. The only proposed solution so far has been to recommend reducing exercise intensity and volume if cardiac issues develop (Eijsvogels *et al.,* 2018). However, there is a lack of research exploring the underlying causes of these abnormal developments in affected individuals.

In this theoretical literature review paired with new data analysis, we propose the hypothesis that adapting to a nasal breathing approach during exercise might protect against the phenomenon of cardiac fibrosis and arrhythmia associated with long-term endurance exercise.

2. Methods

This brief literature review with theoretical analysis was conducted using Google Scholar with search terms including "effect," "nasal," "oral" "oronasal," "hyperventilation," "hypoventilation," "myocardial blood flow," "fibrosis," and "arrhythmia" to identify results relevant to this hypothesis. Additional analysis from a previously published data set (G. Dallam *et al.,* 2018), was approved by the institutional review board of record, the Colorado State University-Pueblo Institutional Review Board, and included as well.

3. Discussion

3.1 Ischemia and the Resulting Myocardial Damage during Exercise

Oxygen demands of working muscles increase significantly during aerobic exercise and this requires a rise in cardiac output to supply the necessary increase in blood flow to the skeletal muscles. The elevated workload of the myocardium also demands an increase in oxygenation and blood flow to maintain proper function. During high-intensity exercise, the cardiac output can increase up to 6 times over the resting condition (Morantz, 2003).

Coronary vascular capacity, however, can only sustain a maximum five-fold increase (Duncker & Bache, 2008). The resulting differential in blood flow between what the heart demands and what can be readily provided as work intensity increases yields an increased potential for deficit that could result in a temporary exercise-induced ischemia in myocardial tissue.

In a classic coronary artery disease model, repeated exposures of intermittent exercise-induced ischemic conditions have an aggregate effect of causing necrosis in the myocardial tissue and subsequent scarring to the affected area. (Geft *et al.,* 1982). Consequently, it is certainly plausible that a mechanism other than coronary artery disease, which causes a relative coronary blood flow restriction during exercise, might offer a reasonable explanation for the development of myocardial fibrosis seen in otherwise healthy endurance athletes.

3.2 The effect of relative changes in ventilation on end-tidal carbon dioxide, arterial carbon

3. 2. 1. Dioxide, and myocardial and cerebral blood flow at rest and during exercise. Resting Observations

Under resting conditions, oral hyperventilation-- such as might occur when breathing orally during a panic attack or during anticipation of high-intensity exercise-- decreases pulmonary end-tidal carbon dioxide (PET CO2) and arterial carbon dioxide (PaCO2) concentrations resulting in relative systemic hypocapnia. In turn, hypocapnia triggers vasoconstriction of the cerebral and myocardial blood vessels reducing blood flow to the brain and heart (Beaudin *et al.,* 2011; Chelmowski & Keelan, 1988; Guensch *et al.,* 2014; Neill & Hattenhauer, 1975). At rest, the reduction in blood flow results in ischemia in the heart which is accompanied by chemical signs of myocardial hypoxia (Neill & Hattenhauer, 1975). Numerous other physiologically and psychologically negative effects occur as well including increased arrhythmia occurrence, skeletal muscle weakness, increased water loss and anxiety sometimes leading to panic (Gilbert, 1999; Svensson S. *et al*., 2006).

By contrast, a nasal breathing approach reduces total ventilation and increases PET CO2 which results in an increase in PaCO2, or relative hypercapnia systemically. As a result, the relative hypercapnia increases both cerebral and coronary blood flow reducing the potential for cardiac and cerebral ischemia and essentially optimizing the physiological state (Beaudin *et al.,* 2011; Guensch *et al.,* 2014).

3.2.2 Exercise Observations

During progressive cycling exercise, the vast majority of athletes switch from nasal to oronasal ventilation when ventilation exceeds approximately 35 l/min. (Niinimaa *et al.,* 1980). In the sole study examining breathing patterns in runners during racing, nearly all participants were observed to breathe orally while running (Niinimaa, 1983). Upon surpassing the second ventilatory threshold, oronasal ventilation increases disproportionately, leading to hyperpnea. This type of hyperpnea results in a greater expulsion of carbon dioxide (CO2) compared to nasal breathing (LaComb *et al.,* 2017). Since studies show hyperventilation at rest induces hypocapnia, which predictably causes vasoconstriction in coronary and cerebral vessels and thereby reduces myocardial and cerebral blood flow (Beaudin *et al.,* 2011), it is plausible that similar effects could occur during exercise, particularly when breathing orally at high workloads. Further, it has been proposed that during the disproportionate increase in ventilation is when the respiratory rate disassociates from PET CO2 and is primarily modulated by central command and muscle afferent feedback (Nicolò *et al.,* 2018). This modulation plays a critical role in adjusting respiratory patterns during physical activity, ensuring that ventilation meets the metabolic demands of exercise. However, the reliance on mouth breathing, which may bypass some of the regulatory mechanisms associated with nasal breathing, could lead to predictive errors in respiratory control (Allen *et al.,* 2023). These errors might result in hyperventilation and an unnecessary increase in respiratory rate, further increasing the likelihood of hypocapnia and excessive sympathetic activation above the second ventilatory threshold (VT2) threshold, which can exacerbate cardiovascular strain. Given that substantial evidence links an elevated resting respiratory rate with cardiac arrest and that respiratory rate has been identified as the most accurate vital sign to predict such events (Nicolò *et al.,* 2020), the potential risks of oronasal breathing during high-intensity exercise become more concerning. The rise in respiratory rate, particularly during exercise, could worsen the cardiovascular strain already imposed by high workloads, further increasing the risk of adverse outcomes like ischemia.

In elite endurance athletes, increased respiratory rate is associated with significant deoxygenation of the intercostal muscles (Contreras-Briceño *et al.,* 2021). This deoxygenation suggests a higher work of breathing, which is well established to contribute to respiratory muscle fatigue during prolonged exercise. Such fatigue may trigger a metaboreflex, leading to a redistribution of blood flow that further strains the cardiovascular system (Sheel *et al.,* 2018). Considering that myocardial blood flow capacity is potentially compromised under heavy exercise loads, the vasoconstriction induced by relative hyperventilation could exacerbate this strain, potentially creating conditions conducive to exercise-induced ischemia in the myocardium. Over time, ongoing disruptions in cardiac perfusion could result in pathological myocardial damage, potentially manifesting as myocardial scarring and atrial fibrillation. Thus, maintaining adequate PaCO2 levels in the blood while exercising by reducing this relative hyperventilation state may be the critical factor in sustaining vasodilation of systemic blood vessels

generally, as well as both the myocardial and cerebral blood vessels specifically. We have demonstrated that adapted nasal breathers can achieve the same peak work and maximal oxygen uptake (VO2max) in comparison to oral breathing while ventilating 22% less and still maintaining adequate tissue oxygenation to avoid an increase in blood lactate (G. Dallam *et al.*, 2018). The reduction in ventilatory rates observed with nasal breathing not only yields significantly higher PETCO2, fractions of expired O2 (FEO2), and pulmonary end-tidal O2 (PETO2) (G. Dallam *et al.*, 2018), but also results in a reduction in the respiratory rate (RR). This decrease in RR may play a key role in diminishing the central command and sympathetic drive during exercise, which are known contributors to increased cardiovascular strain (Nicolò *et al.*, 2020).

In submaximal work, athletes working at 70% VO2max also exhibit a notably higher PETCO2 when breathing nasally compared to oronasal breathing (Paidisetty *et al.*, 2023). This suggests an elevation in PaCO2 and relative arterial hypercapnia while breathing nasally during submaximal exercise as well (González Henríquez, *et al.*, 2016). These conditions have been demonstrated to increase myocardial and cerebral blood flow at rest (Beaudin *et al.*, 2011; Chelmowski & Keelan, 1988; Guensch *et al.*, 2014; Neill & Hattenhauer, 1975) and may produce similar effects during exercise, thus reducing the potential for ischemia. Furthermore, the lower RR associated with nasal breathing may alleviate respiratory muscle fatigue, a factor that can lead to increased cardiovascular strain through the metaboreflex (Sheel *et al.*, 2018). This reduction in respiratory muscle fatigue, coupled with decreased central command and sympathetic activity, could further reduce the risk of exercise-induced cardiac events.

While the effects of one's breathing approach during exercise on cardiac and cerebral blood flow were previously speculative, recent work has now examined this phenomenon directly. A 2023 Baylor University study found higher PETCO2 levels and increased cerebral blood flow during nasal breathing at the same relative to VO2max cycling workloads compared to oral breathing (Moris, 2023). Because subjects not adapted to nasal breathing achieve a lower VO2max and running velocity at VO2max while breathing nasally by about 8% and 10%, respectively (G. M. Dallam & Kies, 2020; Morton et al., 1995), the Moris study (Moris, 2023) strongly supports the hypothesis that nasal breathing better preserves cerebral and coronary blood flow only at a given relative level of exertion. The primary implication of this finding is that nasal breathing during exercise might also be protective of the myocardium and cerebrum at a given level of exertion, reducing the conditions of local ischemia in the brain and heart. Additionally, exercising in relative hypercapnia could offer numerous other benefits, including improved sleep, enhanced immunity, and greater reductions in body fat (Moris *et al.*, 2023). Further contributing to this growing body of evidence is recent research from Calamai et al., which established that patients with diagnosed heart failure and acute or chronic coronary syndrome significantly improved their ventilatory efficiency during submaximal exercise by nasal breathing (Calamai *et al.*, 2023). This study demonstrated that adopting a nasal breathing approach during cardiac rehabilitation exercise resulted in a marked improvement in the abnormal ventilation pattern of participants with cardiac disease through a significant increase in PETCO2 (Calamai *et al.*, 2023), which might, in turn, have also increased myocardial blood flow in these patients during their rehabilitative exercise training.

In our study examining runners previously adapted to breathing nasally at all levels of work, who were then able to produce the same peak work and VO2max in both oral and nasal breathing conditions, we found similar results, although in this case at the same absolute work levels versus at similar levels of exertion. While running at a steady state at 85% of the velocity they achieved previously at VO2max, the subjects maintained a significantly higher steady state PETCO2 in the nasal breathing condition, which predicts a higher PaCO2 level (Dallam *et al.*, 2018) and strongly suggests the conditions for improved myocardial and cerebral blood flow as well. In addition, the subjects had varying levels of experience with nasal-only breathing during exercise, ranging from less than one year to 12 years, yet all had a consistent pattern of higher PET CO2 in the nasal breathing condition, suggesting these outcomes are sustainable over time. These results are illustrated in Table 1, which contains measured PETCO2 levels from our study and predicted PaCO2 levels based on the equation developed by González et al. for exercise conditions (González Henríquez *et al.*, 2016). Consequently, this data suggests that improved cerebral and myocardial blood flow occurs while breathing nasally in comparison to when breathing orally even at the same absolute work levels, once one is adapted to breathing this way.

Table 1. Effect of Nasal versus Oral Breathing during running at 85% of velocity achieved at VO2max on predicted PaCO2*

Nasal Breathing Condition	Oral Breathing Condition
PaCO2 = 8.607 + 0.716 × PET CO2 (44.7) = 40.61 mm/hg	PaCO2 = 8.607 + 0.716 × PET CO2 (40.2) = 37.39 mm/hg

*from (G. Dallam *et al.,* 2018)

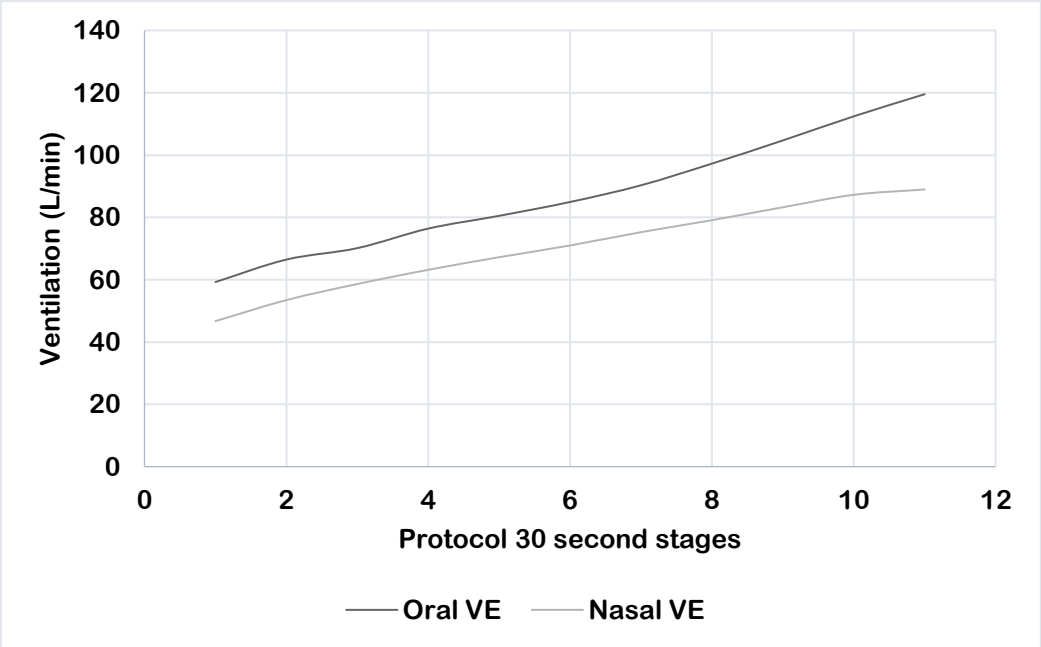

Figure 1. Increase in Oral vs Nasal Minute Ventilation during Graded Exercise Testing (G. Dallam *et al.,* 2018).

*Note that individualized protocols (N=10) of differing lengths (~6-8 minutes) were matched against the final stage at which VO2max occurred, with the earlier stages in longer protocols removed so that each stage represents all subjects.

In addition, the reduction in RR, alongside decreased central command and sympathetic activity, provides a further rationale for nasal breathing as a protective mechanism against exercise-induced cardiac events.

Additionally, in runners adapted to nasal breathing, ventilation is greater at all stages of the same graded exercise test (GXT) when breathing orally versus nasally, as illustrated in figure 1, which is based on further data analysis from our previously reported study (G. Dallam et al., 2018). Ventilation increases to a greater degree past the VT2 at minute 8 when breathing orally in comparison to breathing nasally resulting in a greater hyperpnea as intensity increases in the GXT to VO2max. In these uniquely adapted to nasal breathing subjects, the VO2max and the peak work achieved in the separate GXT protocols were not significantly different (G. Dallam et al., 2018). This suggests that PaCO2 and myocardial blood flow may be impacted negatively while breathing orally in comparison to breathing nasally at the same absolute work levels in those adapted to breathe nasally at all intensity levels. Any deleterious effects of breathing orally are likely to magnify past VT2. This effect is further illustrated in figure 2 which illustrates the fall in PET CO2 past VT2.

During the nasal breathing GXT, PET CO2 is higher, indicating elevated PaCO2 from the early stages of the test, with only a slight decline beyond VT2. This suggests the maintenance of coronary blood flow throughout the test. In contrast, during the oral GXT PETCO2 is lower beginning early in the test and shows a more pronounced fall past VT2, implying vasoconstriction in the coronary arteries and an increased possibility of ischemia, particularly past VT2.

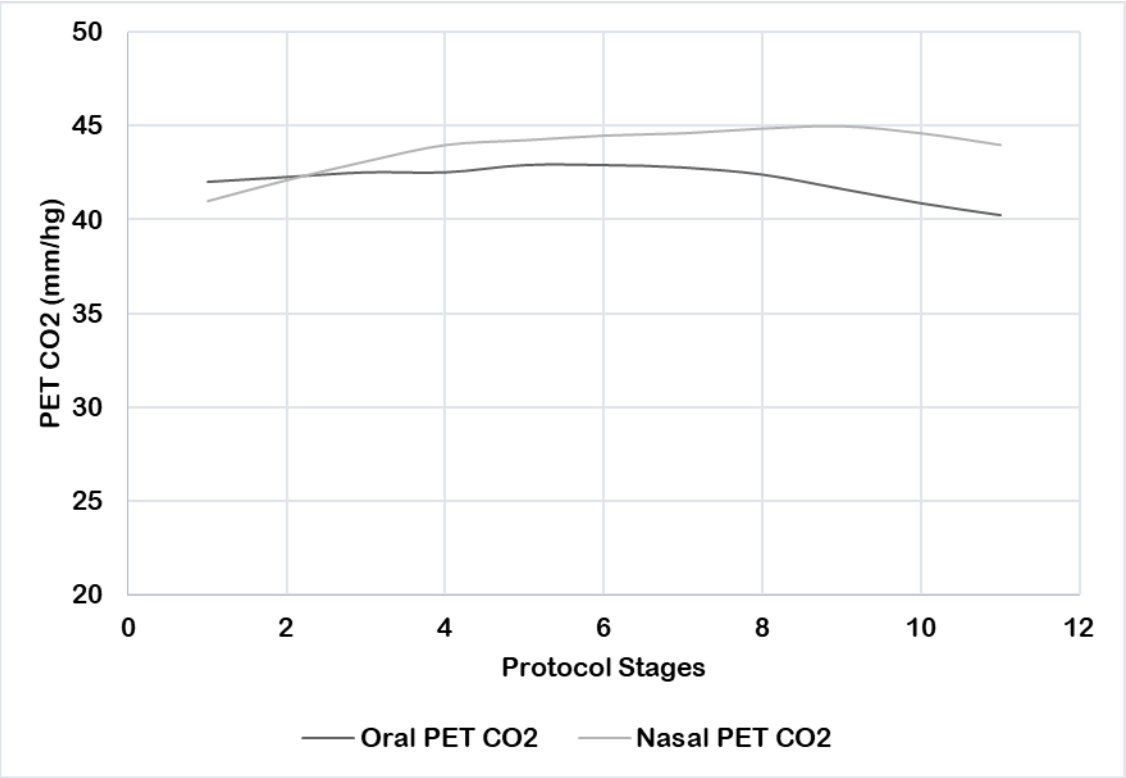

Figure 2. Effect of Oral vs Nasal Ventilation on Pulmonary End Tidal CO2 during Graded Exercise Testing * *N=8. *From (G. Dallam *et al.,* 2018)

Table 2. Overview of the effect of nasal versus oral/oronasal breathing on cerebral and myocardial blood flow at rest and during exercise.

At Rest	**Oral/Oronasal Breathing**	**Nasal Only Breathing**
	Dysfunctional. Operantly learned by decreasing air hunger due to stress, nasal obstruction, etc.	Functional. Baseline ventilation. Natural.
	Creates a relative hyperventilation. Ventilatory efficiency decreased. PET CO2 decreased – lower air hunger. PaCO2 decreased – relative hypercapnia. Myocardial/Cerebral Blood Flow decreased.	Baseline ventilation. Ventilatory efficiency increased. PET CO2 normal PaCO2 normal. PET CO2 normal.
During Exercise	Increasingly dysfunctional as intensity increases. Creates a relative hyperventilation controlling the progression of air hunger. Occurs naturally in response to increasing PaCO2.	Functional as able continue. Baseline ventilation becomes an increasing hypoventilation as work intensity increases creating air hunger. Can be learned/adapted to, at which point air hunger is reduced and ventilation becomes the baseline.
	Ventilation increased. Ventilatory efficiency decreased. PET CO2 decreased. PaCO2 decreased. Myocardial/Cerebral Blood Flow decreased.	Ventilation decreased. Ventilatory efficiency increased. PET CO2 increased. PaCO2 increased. Myocardial/Cerebral Blood Flow increased.

In short, breathing nasally during exercise, compared to oral breathing at the same intensity, results in an increased PET CO2, which in turn suggests a corresponding increase in PA CO2 and enhanced myocardial blood flow. These relationships are further illustrated in Table 2. Increased myocardial blood flow may provide protective benefits against the development of myocardial fibrosis and arrhythmias

that appear in endurance athletes who habitually breathe orally during exercise.

Consequently, the primary limitation to conferring such protective effects lies in the ability to adapt to nasal breathing across all levels of exercise intensity.

3.3 Is adaptation to nasal breathing during exercise possible?

Adopting a nasal breathing approach during exercise appears to initially inhibit work capacity and VO2max (Morton *et al.,* 1995) while also producing increased air hunger (Retty, 2022) in those not accustomed to breathing this way during exercise. This increased air hunger, which is a component of dyspnea and a primal high-arousal emotional effect (Banzett *et al.,* 2021), can drive an elevated RR which might only be achievable by switching to an oronasal breathing pattern (Homma & Masaoka, 2008) as the brain perceives a homeostatic threat. Given that air hunger functions as a feedforward system, primarily influencing the behavioral component of ventilation (RR) rather than tidal volume (VT) (Nicolò *et al.,* 2018), the adaptation process to nasal breathing may indeed be challenging initially. However, previous experimental work illustrates that sustained exposure to increased PaCO2 results in reduced air hunger over time. This effect has been observed in mechanically ventilated humans (Bloch-Salisbury et al., 1996), in humans breathing orally during endurance training at reduced RR (Kapus J, 2013), as well as through regular yoga practice that emphasizes reduced breathing frequencies (Spicuzza *et al.,* 2000).

The predictive processing framework, as described by Allen *et al.* (2023), posits that the brain continuously refines its interoceptive predictions through exposure to sensory stimuli and their associated prediction errors. Within this framework, repeated and controlled exposure to air hunger may facilitate habituation by reducing interoceptive prediction errors and enhancing the precision of respiratory predictions. This habituation process could reduce sympathetic activity, thereby lowering RR during exercise and potentially decreasing central and peripheral vasoconstriction. Such changes might explain why complete adaptation to nasal breathing takes considerable time, potentially accounting for the inconsistencies observed in various studies regarding the efficacy and impact of nasal breathing during exercise. The concept of affective habituation, where the athlete undergoes a process of becoming accustomed to the affective sensation of air hunger, aligns with this idea. Moreover, studies such as those by Sato *et al.* (1986) have shown that the chemoreflex to CO2 can be altered through interventions like hypnosis, suggesting that managing one's arousal state during exposure to mild air hunger, as seen in hypoventilation training, can play a crucial role in reducing anxiety and panic attacks (Meuret *et al.,* 2018). This modulation of arousal may be a central adaptation process, as suggested by predictive processing theories, rather than a peripheral chemoreceptor change. Supporting this, studies of hypnosis-induced dyspnoea relief (e.g., during inspiratory threshold loading or CO2-stimulated air hunger) have demonstrated reduced sensory and affective distress, likely mediated by cortical processing changes rather than reductions in neural drive alone (Morélot-Panzini *et al.,* 2024). These findings align with evidence of altered signal-to-noise ratios in brain areas processing homeostatic threat (Wolf *et al.,* 2022), reinforcing the view that interoceptive prediction error minimization plays a pivotal role in habituating to distressing respiratory sensations.

Our recent study examining the possibility that someone can adapt to breathing nasally during exercise without a loss in VO2max or peak work capacity and with improved economy (G. M. Dallam & Kies, 2020), strongly suggests this could be a viable strategy to prevent cardiac fibrosis and increased arrhythmia associated with long-term participation in endurance sports training. It can be speculated, drawing from comparisons of existing studies (G. M. Dallam & Kies, 2020), that the nasal breathing adaptive process involves both a reduced chemoreflex to PaCO2 and increased peak ventilation while breathing nasally. For individuals already accustomed to exercising with oral breathing, this process may take several months of consistent nasal breathing during exercise, gradually increasing the intensity at which this is possible with the most significant limitation being air hunger if one attempts to progress too quickly (G.M. Dallam & Kies, 2020).

The nasal conditioning developed through consistent nasal breathing has also been demonstrated to be protective against the development of exercise-induced bronchoconstriction (EIB) in asthmatic individuals (Kirkpatrick *et al.,* 1982; Mangla & Menon, 1981; Shturman-Ellstein *et al.,* 1978) most likely as a function of improved air conditioning in the nasal cavity resulting in fully humidified and temperature

regulated air passing to the lung which is then less likely to simulate the conditions associated with EIB. Moreover, an increasing number of published trials illustrate the potential benefits of adapting to a nasal breathing approach for performance in elite cyclists (Gonzalez-Montesinos *et al.,* 2021) as well as a rehabilitation strategy for COPD sufferers (Arnedillo et al., 2020; Gouzi *et al.,* 2023) and coronary heart disease patients (Calamai *et al.,* 2023). In each case, these studies reference an improvement in ventilatory efficiency as the mechanism for improved outcomes, an adjustment that is broadly associated with nasal breathing during exercise and further associated with an increased PET CO2 in this breathing condition (G. M. Dallam & Kies, 2020).

Very limited evidence also suggests that nasal breathing during exercise might reduce systolic blood pressure (Petruson & Bjurö, 1990), although this outcome was reached while using an invasive nasal dilator, that likely reduced the resistance to nasal breathing to such an extent that the subject's achieved peak ventilation similar to oral breathing. In more recent work, nasal breathing failed to reduce blood pressure during moderate-intensity cycling exercise, although it did reduce blood pressure at rest in the same subjects (Watso *et al.,* 2023). These findings suggest that while hypertension and cardiovascular disease are directly related to each other (Hollander, 1976), nasal breathing during exercise appears unlikely to significantly address cardiovascular concerns via reduced blood pressure.

Spicuzza *et. al.* have demonstrated that a reduced chemoreflex to PaCO2 at rest can be achieved using breathing exercises as in yogic practice (Spicuzza *et al.,* 2000), which might in turn provide an alternative avenue to increased PaCO2 tolerance during exercise. However, the available research does not support this transfer (Beutler *et al.,* 2016), logically because the stimulus for downregulation during short term increases in PaCO2 during breathing exercises is inadequate to create improvements during the far more extended exposure that occurs while breathing nasally during exercise.

In addition, Kowalski et. al. demonstrated chemosensitivity at rest, as measured by the Body Oxygen Level Test, also known as the Control Pause Test, did not have significant associations with performance during graded exercise testing and Wingate testing for maximum aerobic and anaerobic performance respectively when breathing orally (Kowalski *et al.,* 2024). However, as we have demonstrated in this paper, elevated PaCO2 is unlikely to be a primary limiter to graded exercise performance when breathing orally as PET CO2 levels drop approaching VO2max and breathing route has no effect on Wingate test performance as previously shown by Recinto et. al. (Recinto *et al.,* 2017). Consequently, the Kowalski et. al. finding is not contradictory to the hypothesis presented here that nasal breathing during exercise results in relative hypercapnia in comparison to oral/oronasal breathing stimulating increased cerebral and myocardial blood flow and that individuals can, in turn, adapt to nasal breathing during exercise by reducing chemosensitivity to realize this physiological benefit.

4. Limitations

The primary limitation of the hypothesis presented in this paper, that nasal breathing during exercise may confer protection against myocardial ischemia and fibrosis resulting in arrhythmias, is the lack of direct studies examining the effect of breathing nasally on myocardial blood flow during exercise. Additionally, the hypothesis we propose is based on a relatively small number of experimental studies directly addressing nasal versus oral breathing during rest and exercise. Consequently, our primary purpose in writing the paper is to highlight these gaps and encourage further research into this promising area of inquiry.

5. Conclusions

In conclusion, existing research strongly supports the concept that adopting a nasal breathing approach during exercise as a means of protecting against myocardial damage in endurance athletes, in addition to providing other health and performance-related benefits, warrants further research by exercise scientists. Future studies should directly examine the adaptive processes required for effective nasal breathing during exercise, as well as the impact of nasal versus oral/oronasal breathing patterns on myocardial blood flow in individuals adapted to nasal breathing.

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Author Contribution Statement

All the authors equally contributed to this work and approved the final version of the manuscript.

Conflict of Interest

The authors declare that there was no conflict of interest.

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