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ANALYSIS AND COMPARISON OF ARTERIAL BLOOD GASES (ABGS) COMPONENTS (pH, pO_2, pCO_2) WITH AND WITHOUT EXTENSIVE EXERCISE IN SELECTED GROUPS OF ATHLETES AND NON-ATHLETES

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ABSTRACT

Current research studied the changes and comparison in pH, pO_2, pCO_2 and related parameters in set of 3 groups.
Group I = Non -athletes with no extensive exercise.
Group II = Non- athletes with extensive exercise.
Group III = Athletes with extensive exercise.
Blood was collected from radial arteries 30 minutes prior to exercise or walk and 15 minutes after exercise in resting condition from subjects in all three groups. Blood was analyzed for pH, pCO_2, pO_2, HCO_3 using Nova Phox Pro Arterial blood gas analyzer (Nova Biomedical, Massachusetts, and USA). Data is presented as pre and post exercise Mean ± Standard error of mean (SE) and compared statistically Pre and Post exercise using Students't-test for paired samples having P value < 0.05 as significant.
(Standard error is the standard deviation of sampling distribution)
Group I show mild, moderate and no significant difference when pre and post arterial blood gases (ABGs) parameters were compared with each other thus depicting non ius respiratory/Ventilatory distress. However, both group II and III exhibited one moderate and remaining very markedly significant difference amongst arterial blood gases (ABGs) parameters, Pre, and Post extensive exercises, suggesting activation of corrective, retaliatory and compensatory mechanisms.

Key words: ventilatory, arterial blood gases (ABGs), pCO_2 and pO2.

INTRODUCTION

It is documented that values of arterial pH, pressure of Oxygen (pO_2) and of Carbon dioxide (pCO_2) changed with minimal increments during exercise (Burton *et al*., 2004). However, increased dependence on glycolysis during vigorous exercise leads to production of pyruvate, and then lactate, that eventually cause elevated pCO_2 and declined pH. Besides, compensatory hyperventilation results in a decrease of pCO_2, decline in further lactic acid production and precluding decline in blood pH as well (Azizi, 2011; Martin, 2008; Litch 2006; Brown *et al*, 2008). In many cases during routine exercise, arterial oxygen and carbon dioxide pressures and ventilatory actions are not abnormal enough to stimulate hyper-respiration (Burton *et al*, 2004; Powers and Howley, 2004). However, it was reported that changes in ventilation is also a linear function of production of carbon dioxide (CO_2) volume during light to heavy exercise (Arena *et al*., 2003; Guazzi *et al*., 2005; Phillips *et al*., 2020). When exercise was performed above respiratory compensation threshold, ventilation volume becomes disproportional to carbon dioxide (CO_2) volume due to excessive acidosis and lactic acid production (Phillips *et al*., 2020).

Such changes in arterial blood components and elevation are mostly reported in conditions such as chronic heart failure (CHF), pulmonary hypertension (PH), interstitial lung disease (ILD), and chronic obstructive pulmonary disease (COPD). Significantly, such altered response contributes to dyspnea and exercise intolerance, which might lead to diminished endurance capacity of athletes (Phillips *et al*., 2020). However, in some cases of compromised pulmonary functions, increasing ventilatory efficiency through anti-coagulant therapy does elevate exercise capacity of that individual (Huang *et al*., 2020).

Therefore, current research studied the changes and comparison in pH, pO2, pCO2 and related parameters in set of 3 groups, Group I who were not athletes and did not perform any exercise, group II non athletes but went through exercise regiments and Group III, who were athletes and exercised.

MATERIALS AND METHODS

Selection of subjects:
Normal healthy adults, n = 60, aged 23-30 years, either gender was selected as per research criteria (Azizi, 2001) and classified in three groups as follows,
Group I = Non athletes, with no extensive exercise (n = 20) [control group]
Group II = Non Athletes, with extensive exercise (n = 20)
Group III = Athletes, with extensive exercise (n = 20)
Complete medical history was evaluated before inclusion of these subjects for research.

Research Protocols:
Group I was subjected to routine walk for 20 minutes, where as Group II and III were subjected to extensive aerobic exercise (Azeemi *et al*, 2020) for 20 minutes. Blood was collected from radial arteries 30 minutes prior to exercise or walk and 15 minutes after exercise in resting condition. Blood was analyzed for pH, pCO_2, pO_2, HCO_3 using Nova Phox Pro Arterial blood gas analyzer (Nova Biomedical, Massachusetts, USA).

Statistical analysis:
Data is presented as pre and post exercise Mean ±Standard error of mean (SE) and compared statistically Pre and Post exercise using Students't-test for paired samples having P value < 0.05 as significant

RESULTS

Group I show mild, moderate and no significant difference when pre and post ABGs parameters were compared with each other viz pO2 (mmHg) < 0.0019 [moderate], pCO2 (mmHg) P < 0.0418 [mild], HCO_3 (mM/L) P < 0.0009 and pH P < 0.2648 [NS], thus depicting non respiratory/ventilatory distress (Table 1). However, both group II (Table 2) and III (Table 3) exhibited one moderate and remaining very markedly significant difference amongst arterial blood gases (ABGs) parameters, Pre and Post extensive exercises (Table 2 and 3), therefore illustrating respiratory/ Ventilatory distress, acidosis and dawdling compensatory mechanism. Comparison of Blood Gasses changes after extensive exercise training in group II (Table 2), who were non athletes, significantly marked changes in pO_2 (mmHg) P< 0.00001, pCO_2 (mmHg) P < 0.00001, pH P < 0.00001 and moderate in HCO_3 (mM/L) P < 0.0017 were noted pre and post exercise. Similar findings were observed in Group III.

Table 1. Comparison of Blood Gases changes in group I with no extensive exercise Non athlete.
Data presented as Pre and post Mean ± SE

Parameters	Pre exercise	Post-exercise	P < 0.05	t-value
pO_2 (mmHg)	88.90 ± 0.73	89.75 ± 0.43	< 0.0019	-3.32484
pCO_2 (mmHg)	44.25 ± 1.99	45.10 ± 1.25	< 0.0418	-2.11201
HCO_3 (mM/L)	25.50 ± 1.21	27.00 ± 1.16	< 0.0009	-4.3589
pH	7.36 ± 0.001	7.35 ± 0.002	< 0.2648	1.13177

Table 2. Comparison of Blood Gases changes after extensive exercise training in group II non athletes.
Data presented as Pre and post Mean ± SE

Parameters	Pre exercise	Post-exercise	P < 0.05	t- value
pO2 (mmHg)	88.35 ± 0.77	81.85 ± 1.93	< 0.00001	17.7253
pCO2 (mmHg)	44.15 ±3.5	46.90 ± 2.62	< 0.00001	-4.9698
HCO3 (mM/L)	25.35 ± 1.50	26.6 ± 0.99	< 0.0017	-3.5411
pH	7.38 ± 0.02	7.26 ± 0.03	< 0.00001	5.97121

Table 3. Comparison of Blood Gases changes after extensive exercise training in group III athletes
Data presented as Pre and Post Mean ± SE

Parameters	Pre exercise	Post-exercise	P < 0.05	t- value
pO2 (mmHg)	88.75 ± 1.04	98.15 ± 0.56	< 0.00001	-33.2888
pCO2 (mmHg)	44.85 ± 1.50	58.45 ± 1.35	< 0.00001	-36.2454
HCO3 (mM/L)	25.75 ± 2.09	27.05 ± 0.58	< 0.0019	-3.5590
pH	7.32 ± 0.02	7.22 ± 0.05	< 0.00001	7.3697

N.B. Standard error of mean (SE) is used in dynamic studies where outcome is dependent on Pre-status compared with Post-status in biological systems.

DISCUSSION

Our study showed elevated levels of pO_2, pCO_2 and HCO_3 in post exercise state as compared to resting state before start of exercise, more markedly in athletes than non athletes and those who did not go for extensive exercise. Previously reported study also exhibited similar pattern where extensive aerobic exercise did influenced changes in arterial blood gases and pH (Azizi, 2011). It is well documented that apparent dyspnea that occurs during extensive exercise elevate demands for ventilatory accelerations, causing metabolic changes resulting in stress on dissolved gases oxygen (O_2) and carbon dioxide (CO_2) (Faisal *et al*., 2015; Ofir *et al*. 2008, a,b; Phillips *et al*., 2020). This strain due to reactionary ventilatory inefficiency on available metabolic stores cause onset of an early critical dynamic mechanical constraints and increase in respiratory neural drive (Phillips *et al*., 2020). Both of these physiological alterations lead to sharp perceived dyspnea and resulting exercise intolerance, thus stirring modification to compensate respiratory chain reactions, resulting in high or low pCO_2, pO_2, pH and HCO_3. Comparable interpretation can also be suggested for our findings as well where extensive exercise regiments caused marked changes in ABGs parameters, even in seasoned athletes.

It was argued that at peak of extensive exercise ventilation volume and carbon dioxide volume (VCO_2) is often at much elevated level as compared to resting state because individuals were characteristically hyperventilating which was preceding excessive metabolic acidosis (Neder *et al*., 2017; Phillips et a., 2019). However, it was shown that individual who didn't possess the capability or potential to exercise above anaerobic threshold exhibited mild to moderate changes in dissolved gases, arterial blood gases (ABGs) or no change at all (Nader *et al*., 2015; Phillips *et al*., 2020), as we have witnessed in our case of non athletes with no exercise. Recent assessments contended that ability or potential to perform vigorous/extensive exercise depends on cardio-pulmonary system and ability of body physiology to deliver oxygen to exercising tissues is essentially required to eliminate metabolic by-products (Umapathi and Nyuagn, 2020). This phenomenon is coupled with movement of O_2 and CO_2 in and out of the human body to compensate any change sequentially. Interestingly, anticoagulant therapy in cardio-blood-pulmonary diseases does improve ventilatory efficiency to increase exercise capacity (Huang *et al*., 2020). Furthermore, plasma volume expansion was also considered as another therapy or option to increase and improve O_2 pressure in athletes (Zavorsky *et al*., 2003) to tackle exercise induced arterial hypoxemia. Conclusively, inducing acute hypervolaemia does improved blood gases status, it was shown, in athletes experiencing exercise induced arterial hypoxemia, and consequently arterial blood gases (ABGs) changes occurred for short period of time and not impaired further (Zavosreky *et al*., 2003).

CONCLUSION

Research study presented here provide data about changes in pH, pO2, pCO2 and related parameters in set of 3 selected groups and compared pre and post exercise. Group I who were not athletes and did not perform any exercise exhibited no considerable difference in parameters whereas, group II who were non athletes but went through exercise regiments and Group III, who were athletes and exercised showed moderate to marked significant difference in pre and post exercise arterial blood gases parameters thus suggesting activation of compensatory, retaliatory and corrective cardio-pulmonary and metabolic mechanisms.

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