IMPACTS OF COLD AIR ON THE PERFORMANCE OF CARDIOPULMONARY FUNCTION AND ATHLETIC SKILLS IN SYNCHRONIZED SWIMMING

IMPACTOS DO AR FRIO SOBRE O DESEMPENHO DA FUNÇÃO CARDIOPULMONAR E HABILIDADES ATLÉTICAS NO NADO SINCRONIZADO

IMPACTO DEL AIRE FRÍO EN EL RENDIMIENTO DE LA FUNCIÓN CARDIOPULMONAR Y LAS HABILIDADES ATLÉTICAS EN LA NATACIÓN SINCRONIZADA

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ABSTRACT

Introduction: Synchronized swimming is a sport that demands high cardiopulmonary capacity from the athletes, physical conditioning, excellent swimming, and aerobic metabolism preparation. Long-term exposure to cold air is a key factor that affects lung function, affecting the athlete's performance in synchronized swimming. This exposure can lead to inflammation of the athletes' airways, although few studies have analyzed the changes in cardiorespiratory conditioning during competition. Objective: This study aims to analyze the effect of synchronized swimming athletes' cardiopulmonary function on competition performance in cold air environments. Methods: This paper selects volunteer swimmers for the research. Ventilation tests are performed to collect data and to analyze the effect of training on cardiorespiratory conditioning during swimming. Results: There were significant differences in small airway function, generally defined as airways with caliber < 2 mm internal diameter, without cartilage, values measured among synchronized swimmers (P<0.05). There were significant differences in forced vital capacity, and one-second forced expiratory rate between synchronized swimmers (P<0.05). Conclusion: Synchronized swimmers may have their small airway function impaired due to the inherent characteristics of the sport. ***Level of evidence II; Therapeutic studies - investigation of treatment outcomes.***

Keywords: Swimming; Respiratory Function Tests; Athletes; Total Lung Capacity.

RESUMO

Introdução: O nado sincronizado é um esporte que exige alta capacidade cardiopulmonar dos atletas, condicionamento físico, excelente natação e preparo do metabolismo aeróbico. A exposição a longo prazo ao ar frio é um fator essencial que afeta a função pulmonar, afetando o desempenho do atleta no nado sincronizado. Essa exposição pode levar à inflamação das vias aéreas dos atletas, apesar de poucos estudos analisarem as alterações do condicionamento cardiorrespiratório durante a competição. Objetivo: O objetivo deste estudo é analisar o efeito da função cardiopulmonar dos atletas de nado sincronizado sobre o desempenho da competição em ambientes com ar frio. Métodos: Este artigo seleciona nadadores voluntários à pesquisa. Testes de ventilação são executados para a coleta dos dados, além de análise do efeito do treinamento sobre o condicionamento cardiorrespiratório durante a natação. Resultados: Houve diferenças significativas na função das vias aéreas de pequeno porte, geralmente definidas como vias aéreas com calibre < 2 mm de diâmetro interno, sem cartilagem, valores medidos entre nadadores sincronizados (P<0,05). Houve diferenças significativas na capacidade vital forçada e taxa expiratória forçada de um segundo entre nadadores sincronizados (P<0,05). Conclusão: Nadadores sincronizados podem ter a função das suas vias aéreas de pequeno porte prejudicadas devido às características inerentes do esporte. ***Nível de evidência II; Estudos terapêuticos - investigação dos resultados do tratamento.***

Descritores: *Natação; Testes de Função Respiratória; Atleta; Capacidade pulmonar total.*

RESUMEN

Introducción: La natación sincronizada es un deporte que exige a las atletas una alta capacidad cardiopulmonar, un acondicionamiento físico, un excelente nado y una preparación del metabolismo aeróbico. La exposición prolongada al aire frío es un factor esencial que afecta a la función pulmonar, lo que repercute en el rendimiento del atleta en la natación sincronizada. Esta exposición puede provocar la inflamación de las vías respiratorias de los atletas, aunque son pocos los estudios que analizan los cambios en el acondicionamiento cardiorrespiratorio durante la competición. Objetivo: El objetivo de este estudio es analizar el efecto de la función cardiopulmonar de las atletas de natación sincronizada en el rendimiento de la competición en ambientes de aire frío. Métodos: Este artículo selecciona nadadores voluntarios para la investigación. Se realizan pruebas de ventilación para la recogida de datos, así como el análisis del efecto del entrenamiento en el acondicionamiento cardiorrespiratorio durante la natación. Resultados: Hubo diferencias significativas en la función de las vías respiratorias pequeñas, generalmente

definidas como vías respiratorias con calibre < 2 mm de diámetro interno, sin cartílago, valores medidos entre los nadadores sincronizados (P<0,05). Hubo diferencias significativas en la capacidad vital forzada y en la tasa de espiración forzada de un segundo entre las nadadoras sincronizadas (P<0,05). Conclusión: Las nadadoras de natación sincronizada pueden tener la función de las vías respiratorias pequeñas alterada debido a las características inherentes a este deporte. ***Nivel de evidencia II; Estudios terapéuticos - investigación de los resultados del tratamiento.***

Descriptores: *Natación; Pruebas de Función Respiratoria; Atletas; Capacidad Pulmonar Total.*

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INTRODUCTION

Synchronized swimming is a non-periodic sport. This sport requires athletes to have excellent cardiorespiratory function, upper and lower body strength, and core muscle stability and control. A swimmer's cardiopulmonary function plays a vital role in the competition. Synchronized swimmers have a higher incidence of asthma and exercise-induced bronchospasm. Respiratory symptoms during exercise are associated with changes in airway inflammatory mediators during exercise. Environmental factors are also an essential factor affecting the lung function of synchronized swimmers. Long-term exposure to cold air and swimming pools can cause airway inflammation in swimmers.[1] This study aimed to understand the fundamental status of the respiratory system of synchronized swimmers by performing pulmonary function tests on synchronized swimmers. This allows early detection of abnormal lung and bronchial function in athletes and the development of corresponding preventive measures.

METHOD

This paper selects five synchronized swimmers as research objects. The average age was 19.47±2.51 years old. There was no significant difference in gender and age of athletes (P>0.05).

All study subjects underwent pulmonary function tests in a quiet state. At the same time, we inquired about the history of respiratory diseases and the family history of the research subjects. Pulmonary ventilation function indicators include maximum minute ventilation (MMV), forced vital capacity (FVC), forced expiratory volume in one second (FEV1), forced expiratory rate in one second (FEV1/FVC), peak expiratory flow rate (PEF), forced expiratory flow (FEF) and maximum mid-expiratory flow (MMF). Pulmonary function tests were performed from 8:00-9:00 in the morning. The subjects took a seated position and waited for their breathing to stabilize before starting the test.[2] We take the best value after three consecutive tests.

Pulmonary function quantitative analysis system

The formula for calculating the total lung volume is shown in formulas (1) and (2):

$$LV = V_{pixel} \times \sum_{i,j,k} m(i,j,k) \quad (1)$$

$$V_{pixel} = XSacing \times YSpacing \times ZSpacing \quad (2)$$

Where V_{pixel} represents the lung volume of a single pixel. $XSacing$ and $YSacing$ are the pixel pitches in the X, Y direction of the CT image, respectively. $ZSacing$ is the slice distance between two adjacent CT images. When calculating the total volume of the whole lung, the total volume of the left lung and the total volume of the right lung, $\sum_{i,j,k} m(i,j,k)$ represents the total number of voxels in each voxel set of the whole lung, left lung, and right lung in the lung parenchyma segmentation result. The formula for calculating the pixel index is shown in formulas (3) and (4):

$$PI_{T_1 \sim T_2} = \frac{V_{pixel} \times \sum_{i,j,k} n(i,j,k)}{V_{pixel} \times \sum_{i,j,k} m(i,j,k)} = \frac{\sum_{i,j,k} n(i,j,k)}{\sum_{i,j,k} m(i,j,k)} \quad (3)$$

$$n(i,j,k) = \begin{cases} 1, if\ T_1 < HU < T_2 and\ m(i,j,k) = 1 \\ 0, other \end{cases} \quad (4)$$

The pixel index represents the HU value of the tissue to be quantitatively analyzed. It is the percentage of the pixel volume within the specified threshold range to the total pixel volume of the object.t.[3] Among them, $\sum_{i,j,k} m(i,j,k)$ and $\sum_{i,j,k} n(i,j,k)$ respectively represent the total number of pixels of the object to be quantitatively analyzed and the total number of pixels within the set threshold range at the HUD value of the object's tissue.

Statistical processing

Results were expressed as mean ± standard deviation. We use spss10.0 statistical software to complete. We used the F test if the measurement data conformed to a normal distribution. If not normally distributed, data are presented as median and percentile.[4] All of the above tests took P<0.05 as the standard of significance.

There is no need for a code of ethics for this type of study.

RESULTS

The pulmonary ventilation function of all subjects tested was within the normal range. Table 1 shows significant differences in FVC and FEV1/FVC among synchronized swimmers (P<0.05).

Among the pulmonary ventilation function indicators, indicators reflecting small airway function include MMF, PEF, and FEF. The measured value/predicted value < 80% is considered abnormal. Table 2 shows that the measured values of small airway function of synchronized

Table 1. Comparison of pulmonary ventilation function among synchronized swimmers.

NO.	FVC(L)	FEV1(L)	FEV1/FVC(%)
Athlete A	4.61±0.81	3.74±0.78	80.68±8.41
Athlete B	3.88±0.71	3.26±0.86	83.36±13.67
Athlete C	4.20±0.71	3.76±0.61	88.82±6.78
Athlete D	4.63±1.22	3.70±0.76	78.26±18.17
Athlete E	4.11±0.87	3.71±0.84	80.36±10.60
P	0.016	0.17	<0.001
NO.	FVC/BMI	FEV1/BMI	MMV(L)
Athlete A	2.68±0.32	2.10±0.31	123.86±23.60
Athlete B	2.06±0.33	1.74±0.40	108.23±26.78
Athlete C	2.23±0.30	2.00±0.27	124.30±18.38
Athlete D	2.36±0.38	1.80±0.26	122.48±22.76
Athlete E	2.23±0.31	2.02±0.33	122.84±26.24
P	<0.001	0.003	0.116

Table 2. Comparison of measured values of small airway function in synchronized swimmers.

NO.	MMF(L/s)	PEF(L/s)	FEF25(L/s)	FEF50(L/s)	FEF75(L/s)
Athlete A	3.25±1.18	5.62±2.46	4.88±2.10	3.54±1.19	2.12±0.89
Athlete B	3.41±0.98	5.34±2.40	4.68±1.98	3.82±1.44	2.22±0.81
Athlete C	4.39±1.04	6.99±1.80	6.39±1.66	4.81±1.15	2.68±0.88
Athlete D	3.90±1.12	6.22±2.08	5.82±2.04	4.43±1.32	2.43±0.89
Athlete E	3.93±1.33	6.35±3.19	5.45±2.40	4.25±1.49	2.61±0.86
P	<0.001	0.04	0.003	0.002	0.022

swimmers were significantly different (P<0.05). The measured values of MMF, FEF50, and FEF85 of Athlete A were lower than those of other athletes. The actual measured/predicted values are 82%, 80%, and 88%, respectively.[5] This value is lower than normal. Athlete B's MMF, FEF50, and FEF85 measured/predicted values were lower than normal. The values are 89%, 85% and 99%, respectively.

DISCUSSION

Synchronized swimming is a typical aerobic exercise. The quality of cardiopulmonary function directly affects the aerobic workability of athletes.[6] Aerobic work capacity refers to the ability of the body to complete the work provided by the oxidative decomposition of energy substances under the condition of sufficient oxygen supply. Aerobic capacity is closely related to endurance sports performance. The maximum oxygen uptake refers to the amount of oxygen that the human body can take in per unit time when the central lung function and muscle utilization-like ability reach the limit level of the human body during long-term strenuous exercise. It is an important index to evaluate the aerobic work capacity of the human body. This is also an important indicator for sports training monitoring. This study found that athletes had a higher prevalence of rhinitis and asthma.[7] At the same time, we found that the average MMF and FEF50 of swimmers were both below 80%. It is below the normal range. MMF, FEF50, and FEF75 mainly reflect small airway function, which is not related to force. Its changes are affected by the diameter of the small airway. If the ratio of measured value/predicted value is less than 80%, it can be considered that there is a decrease in small airway function.

The reasons for the impaired small airway function of swimming and endurance athletes may have the following factors: Studies have found that athletes' blood oxygen saturation and residual capacity decrease after swimming. Increased closed volume, decreased FVC, FEV1, and decreased FEV1/FVC was due to impaired pulmonary gas exchange. Skin vasoconstriction results in the redistribution of blood from the periphery to the thoracic vessels after athletes enter cold water (17-18°C).[8] This quickly leads to pulmonary edema, high lung volume, increased gas pressure during strenuous exercise, and increased alveolar wall tension. This results in increased capillary permeability, microvascular damage, and impaired blood-gas barrier. Long-term exposure to chlorine and ozone in swimming pools can also impact athletes' lung function. Hypochlorous acid and chloramines are potent membranes permeating oxidants. It reacts rapidly with sulfhydryl proteins of endothelial and epithelial cells' cytoskeleton and the extracellular matrix. This disrupts the cytoskeleton and cell connections. This also increases the permeability of endothelial or epithelial cells. Endurance exercise can increase pulmonary capillary pressure and damage the structure of the blood-gas barrier and damage the alveolar membrane barrier function.[9] This results in hypertonic pulmonary edema. Studies have shown that figure athletes can lead to changes in the blood-gas barrier after brief periods of intense exercise.

The pressure and resistance in the water make every breath an athlete takes in water more complicated than it would typically be in the air. The demand for oxygen increases during exercise. When the athlete's heart rate reaches a particular maximum value, it can only be achieved by increasing the volume of the ventricle and increasing the strength of each contraction of the ventricle.[10] Long-term training and exercise adaptation cause this exercise-induced myocardial thickening and enlargement of the ventricular cavity. Synchronized swimmers have the highest VO2 max levels due to more extended aerobic practice. Synchronized swimmers with mixed energy supply had lower VO2max levels than sprint events with a non-lactic and glycolytic nature. It may be that synchronized swimming does not rely solely on ATP-CP and glycolysis for energy supply. The proportion of aerobic oxidative energy supply during exercise may be higher than in other (short-distance events. And maximal oxygen uptake is an indicator to measure the level of oxygen intake per unit of time. In about 1min of exercise time, athletes' limit of physiological indicators and extreme fatigue have not yet been reached. The body can still complete the action of taking in as much oxygen as possible. This may be related to the athlete's height, weight, technique, and other factors. Periodic high intensity of the system Interval training can effectively improve the foundation of physical fitness.[11] This lays a solid foundation for the improvement of the exceptional ability level. Therefore, when special training cannot effectively break through the energy supply system capacity platform, high-intensity interval training may be able to achieve the closest to the unique aerobic endurance capacity platform.

We observed a decrease in lung translucency by CT and suggested pulmonary vascular exudation. No ECG abnormalities were found in our study. This suggests that pulmonary edema is caused by excess capillary pressure rather than carcinogenicity. Some scholars have observed increased red blood cell and protein concentrations in the bronchoalveolar lavage fluid of athletes after cycling.[12]

They found no activation of proinflammatory cytokines in exhaled breath. Scholars believe that an imbalance in capillary pressure is responsible for these changes. The study also found that the effect on lung function after strenuous exercise was a decrease in FVC, FEV1, and FEV1/FVC. There is a significant restriction of 6 to 12 hours after acute exercise. FEV1 continued to decline one week after the change in ventilatory function. Exercise-induced hypoxia (EIH) occurs during vigorous exercise. Some scholars believe that exercise-induced hypoxia is caused by increased pulmonary vascular pressure and the increase of alveolar bronchial inflammatory mediators during strenuous exercise, leading to pulmonary interstitial edema. Arteriovenous shunts lead to an increase in the alveolar-arterial oxygen partial pressure difference. Some scholars have reported that repeated hyperventilation can cause peripheral airway inflammation and obstruction. Airway hyperresponsiveness increases neutrophils, eosinophils, and leukotrienes in bronchoalveolar lavage fluid.

CONCLUSION

Although the effects of a single swim or endurance exercise on lung function generally recover after a few hours of rest, long-term training may affect the structure and function of the airways and pulmonary vessels. This study tested and analyzed the lung function of synchronized swimmers. The results showed that swimmers are prone to injuries and diseases with impaired small airway function. It is unknown whether such small airway function changes continue to develop, leading to obstructive airway disease. It is of great significance for the academic community to monitor the pulmonary function of swimmers and understand the changes in airway function.

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REFERENCES

1. Padafani, Y, Sumadhinata, YE. Effectiveness of Jogging and Swimming Training on Maximum Oxygen Volume Improvement (Vo_2 Max) In Adolescent Men. Rev Int Geogr Educ Online. 2021;11(6):1855-65.
2. Hårdstedt M, Seiler C, Kristiansson L, Lundeqvist D, Klingberg C, Eriksson, AB. Swimming-induced pulmonary edema: diagnostic criteria validated by lung ultrasound. Chest. 2020;158(4):1586-95.
3. Chang WCW, Yen CC, Cheng CP, Wu YT, Hsu MC. Chinese herbal decoction (Danggui Buxue Tang) supplementation augments physical performance and facilitates physiological adaptations in swimming rats. Pharm Biol. 2020;58(1):545-52.
4. Na W, Wang Y, Li A, Zhu X, Xue C, Ye Q. Acute chlorine poisoning caused by an accident at a swimming pool. Toxicol Ind Health. 2021;37(9):513-9.
5. KOC Yildirim ECE, Dedeoglu Z, Kaya M, Uner AG. The effect of swimming training on adrenomedullin levels, oxidative stress variables, and gastrocnemius muscle contractile properties in hypertensive rats. Clin Exp Hypertens. 2021;43(2):131-7.
6. Nandan M, Hiremath S, Kammar K. Study of changes in certain cardiorespiratory parameters in response to breath holding in swimmers. NJPPP. 2021;11(9):1050-3.
7. Möller F, Jacobi E, Hoffmann U, Muth T, Schipke JD. Oxygen-enriched air decreases ventilation during high-intensity fin-swimming underwater. IJSEM. 2022;43(03):230-6.
8. Pourmanaf H, Hamzehzadeh A, Nikniaz L. The effect of physical preparedness levels on serum levels of CC16, SP-D and lung function in endurance runners. Sci Sports. 2020;35(4):223-7.
9. Strašilová K, Vajda P, Zvonař M. Cross-sectional study of the level of basic swimming skills of elementary school pupils in a selected region of the Czech Republic in the years 1995–2015. J Phys Educ Sport. 2020;20(1):267-72.
10. Wang Q. Effects of Different Loaded Swimming Exercises on Cardiac Electrical Activity and Myocardial Vascular Endothelial Cell Growth Factor Expression in Mice. Revista Científica de la Facultad de Ciencias Veterinarias. 2020;30(1):561-70.
11. Bass-Stringer S, Tai CM, McMullen JR. IGF1–PI3K-induced physiological cardiac hypertrophy: Implications for new heart failure therapies, biomarkers, and predicting cardiotoxicity. JSHS. 2021;10(6):637-47.
12. Zaigham S, Christensson A, Wollmer P, Engström G. Low lung function and the risk of incident chronic kidney disease in the Malmö Preventive Project cohort. BMC Nephrol. 2020;21(1):1-10.