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THE DIFFERENCE IN VENTILATORY
DRIVE IN TRAINED AND UNTRAINED
PERSONS -SYSTEMATIC REVIEW

RAZLIKA U VENTILATORNOM NAGONU
KOD FIZIČKI AKTIVNIH (UTRENIRANIH) I
NEUTRENIRANIH OSOBA - SISTEMSKI
PREGLED

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Ključne reči

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Abstract

Introduction: The simplified concept of respiratory urge is to consider it as the integrated "exit" from the CNS to the respiratory muscles of the "pump". This urge is a result of a complex central respiratory sample generation, and may occur in abnormal situations, e.g. of pathological or pharmacological origin, be altered. The ventilatory drive is affected by some factors such as chemosensitivity, basal arterial oxygen or carbon dioxide tension, mechanical impedance and breathing muscle dysfunction. Dulled ventilatory urge or a decrease in the perception of dyspnea in certain conditions could lead to a reduce in the alarm reaction to dangerous situations such as severe airway obstruction, severe hypoxemia, or severe hypercapnia. **Aim:** The purpose of this systematic review is to obtain differences in respiratory drive among untrained and trained subject. We have searched Pubmed database and the key words were: respiratory drive. We have thoroughly studied 46 articles. **Conclusion:** Physical activity plays crucial part in respiratory adaptation, improving respiratory drive which could lead to better and faster response on stronger effort, without fatigue in untrained subjects. To reach respiratory adaptation to stonger stimuli, physical activity must be continuous and intense.

INTRODUCTION

The goal of the respiratory system is to relieve the diffusion of essential gases and chemicals among vascular system and ecological air and the circulatory system. The essential process is oxygen intake and the removal of carbon dioxide from the body, which relives metabolism on a cellular stratum during the body and takes a relevant role in keeping the overall physiological pH⁽¹⁾. The main role of respiratory system depends on respiratory rates^(1,2). The breathing rate represents the number of inhales a person takes every minute and is one of the capital vital signs, next to blood pressure, pulse, and temperature. When a person inhales, oxygen gets into lungs and then travels to the organs, and on expiration carbon dioxide leaves the body. Breathing rates determined

based on many health and activity factors, and differs in adults and children⁽²⁾. A normal rate takes a decisive role in maintaining the balance of oxygen and carbon dioxide even⁽¹⁻⁴⁾. For example, during exercise, body requirement for oxygen will rise in order to keep the aerobic metabolism of fuel to make energy. In the same time, the stage of metabolic carbon dioxide will rise. If at any period the oxygen supply is not enough to meet the necessary request, aerobic metabolism will no longer be potential and the energy production will failure. As well, if carbon dioxide levels are permitted to accumulate without disposal, the blood will become more acidic, guiding to cellular damage on a systemic scale, which may finally lead to organ failure or death. Neither result is desirable⁽⁵⁻⁹⁾.

Respiratory function. From a functional viewpoint, there are three groups of respiratory muscles: the diaphragm, the rib cage muscles and the abdominal muscles (10–12). Each group affect on the thorax and its sections, i.e. the lung-connected thorax, the diaphragm-connected thorax and the abdomen. Diaphragm shortening expands the upper parts of abdomen and the lower part of the chest (abdominal rib cage). Thoracic muscles, including the intercostal, parasternal, scalene and neck muscles, are both inspiratory and expiratory, and mostly interact on the upper part of the thorax (pulmonary rib cage). The abdominal muscles are expiratory and act on the abdominal rib cage and the abdomen. When contractions of an isolated muscle group exist, the contraction is predominant liken to the other groups, unappetizing effects occur on at least one of the compartments (like a “paradoxical” internal or external motion during inspiration and expiration, relatively). To avoid these effects, a highly coordinated engaging of two or three muscle groups is necessary. Throughout breathing at rest, this is achieved by the coordinated activity of the diaphragm and inspiratory thoracic muscles. Usually no expiratory muscles are used (1–3,10).

Control of respiratory function. It has been known since ancient times the mechanism of breathing (1–3). In general, the breathing control mechanisms respond to entrant from neural and chemical receptors. But if we considering mentioned above, the respiratory drive is a system that can be broken down into three components: the neural central control system, the sensory input systems, and the muscular effect systems.

Neural control. The neural central control system represents an intrinsic ventilation-rate pacemaker as well as a pacemaker for the air intake volume. Respiratory centers in the brain integrate all intrinsic and extrinsic inputs thus providing neuronal drive to the breathing muscles, which keep upper airway openness and expand the lungs to define the level of ventilation (11–13). The sensory systems offer input to the central nervous system to modulate the total rate and volume of breathing. Finally, a uniform signal is sent to respiratory muscles, in order to engage the respiratory drive into exchanging the air. Together these processes function to expel low oxygen, high carbon dioxide air from within the alveoli of the lung and to intake high oxygen, low carbon dioxide air from the atmosphere to facilitate gas exchange on a cellular level (14–16).

Diaphragm control. On the other side, throughout exercise the diaphragm is originally a “flow generator”. Its mechanical power is mostly expressed as velocity of contraction rather than pressure. Throughout exercise the expiratory muscles have an active role in breathing, which is not a case in steady state. Their action is highly coordinated with action of thoracic muscles within each single breath. During inspiration, while the abdominal muscles gradually relax, thoracic muscles contract, and *vice versa* during expiration. This mechanism has few effects: 1) it prevents distortion of the thorax; 2) it unloads the diaphragm allowing it to act as a flow generator 3) it decreases the volume of the abdomen down below resting levels (17–19). As a result, end-expiratory lung volume is reduced during exercise and the mechanics of breathing is set for some purposes. Tidal volume hap-

pens in the most compliant part of the breathing system: 1) the diaphragm is elongated and therefore works close to its optimum length 2) during the previous expiration part of the required inspiratory work is stored in the shape of elastic energy (20–22). Oppositely, thoracic and abdominal muscles are primarily “pressure generators” (22,23).

Thoracic control. Sensory information regarding the volume of the lung space is supplied to the breathing center of the brain from the mechanoreceptors located in the airways, lung and pulmonary. There are two primary kinds of thoracic sensors: slow adapting and rapid adapting receptors. Slow acting mandrel sensors conduct only volume information. Oppositely, the rapid-acting receptors match to both the volume information and chemical irritation triggers such as foreign harmful agents. Both types of mechanoreceptors signal over cranial nerve X (the Vagus Nerve) to the brain to rise the rate and volume of breathing, or to stimulate errant coughing model of breathing secondary to irritants in the airway (19,22–24). Throughout exercise, the abdominal muscles along with the diaphragm play a role as an “auxiliary heart”. A significant quantity of blood probably from the splanchnic vasculature, is shifted between the trunk and the extremities at each act of breathing, contributing to rise cardiac output (22–24). To a probably lesser extent, lower extremity muscle contraction contributes to venous return facilitating the propulsion of blood from the skeletal muscle vasculature, through the so called “skeletal muscle pump”. But, these mechanisms are only valid at moderate levels of exercise (21,22). Expiratory flow limitation and extended expiratory time throughout hard exercise result in higher average positive intrathoracic pressures that abate ventricular transmural pressure and act like a Valsalva manoeuvre, thus reducing the rate of ventricular filling throughout diastole, reducing stroke volume, venous return and cardiac output. These effects of respiratory muscles on the cardiovascular system disturb systemic oxygen delivery (21–24) and make the extremity muscles even more susceptible to tiredness.

Peripheral chemoreceptors. The carotid and the aortic bodies represent peripheral chemoreceptors, and function to control the partial pressure of arterial oxygen in the blood. Acidosis and hypercapnia modify functioning of these receptors by increasing their sensitivity. The aortic bodies are situated within the aortic arch and the carotid bodies are situated at the common carotid artery bifurcation. Once stimulated by hypoxia, they transmit a signal over cranial nerve IX (the glossopharyngeal nerve) to the nucleus tractus solitarius in the brain which in turn stimulates excitatory neurons to rise ventilation. It has been evaluated that the carotid bodies include 15% the total driving force of respiration (14,15,22).

Respiratory function in trained subjects. An appreciation of differences in respiratory physiology among athletically trained persons and non-athletic persons is important to enable meaningful comparison of any received physiological measures. It is usually expected that athletes will have resting lung values above normal physiological ones. However, various studies reveal that lung volumes generally reflect body-size characteristics and genetic influences of a person (7,9,22). There is restricted evidence which suggest that exer-

cising changes the structural parameters of the respiratory system. However, lung volume is connected to aerobic capacity. Accordingly, in athletes for whom aerobic capacity is an important component of success, average lung volumes will tend to be higher than in the general population (6,7,8,9,21,22).

Demands set up on the respiratory system throughout exercise in healthy individuals do not stress the capacity limits within the system. Some scientist described how the breathing system adapts throughout exercise to keep performance and minimize airway resistance, nevertheless significant increases in ventilation. This process includes synchronous sequences of activation and deactivation of breathing skeletal muscles and bronchial smoothly muscle to alleviate the rise in tidal volume and optimize elastic work performed by the respiratory muscles (6,7,8,9,22).

Throughout heavy exercise the high levels of respiratory muscle work must be maintained. This causes respiratory muscle tiredness and may start up the metaboreflex, bring to vasoconstriction of limb locomotor muscle vasculature, exacerbating peripheral tiredness of working muscles and intensifying effort perceptions *via* feedback, thus contributing to limitation of high-intensity endurance exercise performance (19). Still, there is an open question whether the respiratory muscle metaboreflex is enough to predominate on the local vasodilator effects present in locomotor muscles, thus redistributing blood flow to respiratory.

It is not possible to reduce stress of the respiratory muscles throughout. Using mechanical ventilators, low-density

gas compound (such as heliox) or supplemental oxygen is not practicable nor permitted for healthy athletes. Only by training, one can improve mechanical efficiency of respiratory muscles and the fatigue resistance. There is still no definitive evidence as to whether it is possible to augment exercise tolerance. Trusty imminent studies showed that respiratory muscle training has a small but fair and notable effect on endurance while exercising (7,8,9,23,24). What needs to be resolved is the mechanism by which training of respiratory muscle ameliorates exercise performance, concerning alleviation of respiratory muscle tiredness, limb muscle tiredness, the attenuation of the respiratory muscle metaboreflex, and alleviation of the discomfort connected with high levels of respiratory muscle work (18,19,24).

CONCLUSION

We could conclude and support our recent findings that healthy, untrained subjects could adapt their respiratory system to minimal "stress" as the high or moderate intense effort and they do not disturb their ventilator drive in any way. Physical activity could only improve their tolerance to effort and faster adaptation of respiratory system to stronger effort, but it has to be continuously.

Sažetak

Uvod: Ventilatorni nagon se pojednostavljeno može prikazati kao integrisani, izlazni odgovor centralnog nervnog sistema ka respiratornoj muskulaturi. Ventilatorni nagon predstavlja rezultat kompleksnog generisanja obrazaca respiratornog centra, pri čemu oni mogu biti izmenjeni pod dejstvom patoloških ili farmakoloških faktora. Ovaj sistem je pod direktnim uticajem hemoreceptora, parcijalnog pritiska kiseonika i ugljen dioksida, i zavisi od otpora i eventualne disfunkcije respiratorne muskulature. Njegovo zatajenje ili redukcija percepcije dispnee u okviru pojedinih stanja može voditi neadekvatnoj reakciji na ugrožavajuće situacije poput ozbiljne opstrukcije disajnog puta, teške hipoksemije odnosno hiperkapnije. **Cilj:** Svrha ovog pregleda je da se dobiju podaci o razlikama respiratornog nagona kod neutreniranih i utreniranih osoba. Pretražili smo Pubmed bazu sa „respiratorni drajv“ kao ključnom reči i detaljno smo proučili 46 članaka. **Zaključak:** Fizička aktivnost igra značajnu ulogu u respiratornoj adaptaciji. Kao intenzivna i kontinuirana, fizička aktivnost unapređuje ventilatorni nagon i čini odgovor neutreniranih osoba bržim i boljim na teži fizički napor, bez osećaja umora. st mora biti kontinuirana i intenzivna.

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