DRUG INFLUENCES ON SATURATION IN OXYGEN – THE ROLE OF SKELETAL MUSCLES

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Abstract
The way peripheral blood oxygen desaturation occurs, as evidenced by the oxygen saturation index, is not fully elucidated during both physical exercise and functional tests. The paper presents some physiological influences of physical effort on arterial vasodilation, highlighting the fact that nitric oxide and vasodilation products produced by striated muscles, in the case of individuals with high muscular mass, increased values of the oxygen saturation index can be recorded immediately after the physical exercises. Increased doses of nitric oxide or vasodilator prostaglandins may also occur under the influence of some drugs, and some substances designed to treat migraine are concurrently inhibitors of nitric oxide synthase. In these ways, values of the oxygen saturation index that do not correspond to cardio-respiratory capacity can be recorded during exercise tests.

Keywords: *oxygen saturation index; muscle mass;*

INTRODUCTION

The index saturation in oxygen of arterial blood/heart rate per minute has a particular clinical importance in interpreting the results of functional explorations. Thus, the utility of this index for the prediction of severe airway obstruction in children with bronchial asthma was studied [10]. A number of conditions can alter the oxygenation of peripheral blood. It is known that during dental treatments, children suffering from congestive heart failure may experience oxygen desaturation of peripheral blood [16]. Oxygen desaturation of peripheral blood can occur during physical effort (Seixas et al., 2013), especially in athletes who train intensively (Powers et al., 1988), and how is change the relationship between ventilation and perfusion during heavy exercise is unknown (Dempsey & Wagner, 1999). Knowing the pharmacological interferences between physical therapies and stress tests, this paper proposes the analysis of some drug influences on which peripheral saturation in oxygen (SpO_2) depends.

Physiological factors on which arterial vasodilatation depends

Considering that the pulse oximeter measures oxygen saturation of the arterial compartment [5] it is necessary to present the physiological factors on which arterial vasodilatation depends. During exercise, an increase in muscle blood flow is considered by the body to be "safer" than an increase in oxygen extraction [14]. According to a modern theory, vasodilation is electrically driven from the periphery to the arteries that feed the muscle with blood, being moderated by sympathetic neuroefector signals that govern the muscular blood flow at rest and during exercise [2]. The vasodilator role of nitric oxide (NO) [8] produced by endothelial cells [13] is known. Skeletal muscles have the ability to synthesize vasodilator prostaglandins and NO in increased amounts in response to contraction [11], so it is not excluded that individuals with high muscle mass produce increased amounts of NO during exercise.

Prostaglandins E1 (PGE1) and E2 (PGE2) have vasodilatory role [4].It results that physical exercise, especially if performed by subjects with high muscle mass, can result in increased SpO_2 values.

Pharmacological factors that can influence peripheral saturation in oxygen

Some adrenergic blockers (doxazosin), conversion enzyme inhibitors (imidapril) or calcium channel blockers (amlodipine) increase NO production [18]. The vasodilation produced could mask the poor adaptation of the respiratory or cardiovascular apparatus, if it be estimated using the index saturation in oxygen/heart rate per minute. Hypoxia increases the generation of NO from nitroglycerin, a drug used to treat ischemic cardiopathy [1]. In patients with obstructive chronic bronchopneumopathy, oxygen desaturation of arterial blood appears after the 6 minute walk test [7]. If these patients would be treated with

nitroglycerin, high NO release could induce errors in assessing the degree of ventilator dysfunction. The same interference between medication and functional samples would be possible in patients with migraine.

For the acute or prophylactic treatment of migraine, nitric oxide synthase (NOS) inhibitory drugs have been designed: LN (G)-methylarginine hydrochloride [L-NMMA], NOS inducible selective inhibitors named GW273629 and GW274150, NXN-188 neuronal NOS inhibitor and 5-HT1B /1D receptor agonist) [3]. In this case, low SpO_2 values would be recorded during exercise, which would erroneously suggest a cardiac and/or respiratory failure. In the child, PGE2 can be administered for the management of dependent ductal congenital malformations [17]. PGE1 can also be given during dental procedures [19] or after hepatic transplantation [9].

DISCUSSIONS

The physio-pharmacological interferences described above may have an impact both in sports activity (performance and leisure time) and in the interpretation of functional exploration tests. The questioning of subjects on the medication followed could explain, for example, the circumstances of the occurrence of oxygen desaturation during the incremental walking test in healthy subjects, which is not related to either gender, age or respiratory parameters [15]. Nitric oxide and vasodilator prostaglandins produced by the active striatal muscle may play a protective role in the development of hypoxia.

CONCLUSIONS

Through nitric oxide and vasodilator prostaglandins produced by the striated muscles, high levels of SpO_2 can be recorded in individuals with large muscle mass immediately after exercise.

Increased doses of nitric oxide may also occur under the influence of some drugs, such as doxazosin, imidapril, nitroglycerin, resulting in a "false" increase in the saturation index in oxygen / heart rate per minute.

In the case of LN (G)-methyl-L-arginine hydrochloride, GW273629, GW274150, and NxN 188 (nitric oxide synthase inhibitors designed for the treatment of migraine), low SpO2 values, that do not correspond to functional cardio-respiratory capacity, may be recorded during stress tests.

SpO_2 values that do not correspond to actual cardiorespiratory capacity may also occur during PGE1 and PGE2 treatments (vasodilator prostaglandins).

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