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Respiratory Mucosa in Patients with Chronic Obstructive Pulmonary Dysfunction

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Abstract

The vast majority of pulmonary medications are used to promote broncho-dilation and improve alveolar ventilation and oxygenation. These effects should improve an individual's ability to exercise and more effectively develop training effects. Because of the side effects of many pulmonary medications, however, exercise tolerance and the normal adaptations to habitual exercise conditioning may be retarded. The following section is not presented to discourage the use of these medications nor of exercise training; rather, it should highlight the important role exercise has for patients with pulmonary disease who are taking pulmonary medications.

Keywords: Pulmonary medications; Ventilatory efficiency; Peripheral adaptations; Enzymatic activity; Bronchiectasis

Introduction

Habitual exercise performed at the proper intensity, duration, and frequency typically elicits both peripheral and central physiologic adaptations. The central adaptations include improved ventilatory efficiency and improved cardiac performance. Peripheral adaptations include an increase in the number and size of mitochondria, improved extraction of oxygen from circulating blood by the exercising muscles, increased muscle strength, increased mitochondrial enzymatic activity, proliferation of capillaries, an increase in the mean transit time of blood through muscle capillaries, a lowering of peripheral vascular resistance, and an increased arterio-venous oxygen different [1]. Moreover, these central and peripheral adaptations produce a reduction in resting and submaximal heart rates, blood pressure, respiratory rate, and rating of perceived exertion, improved skeletal muscle and coronary blood flow, increased exercise-induced lipolysis and translocation of lactate from muscle cells to the blood, and improved oxygen consumption and physical work capacity [2]. Unfortunately, many of the favorable effects associated with habitual exercise are markedly delayed or absent in patients with pulmonary disease. In addition, many of the medications routinely used in the treatment of pulmonary disease mask or impede the beneficial effects of exercise. In particular, glucocorticoids have several deleterious effects on exercise performance and bodily function [3]. The major deleterious manifestations of corticosteroids include cataracts, diabetes, peptic ulcers, emotional-ability, ecchymoses, oedema, osteoporosis, weight gain with cushin-goid appearance, skeletal muscle myopathy occurring in the proximal and possibly other muscle groups, and atrophy of muscle fibres [4]. It is of particular concern for patients with pulmonary disease that steroid myopathy and muscular atrophy have been identified not only in the peripheral skeletal muscles but also in the muscle fibres of the diaphragm.

Methodology

The impact of glucocorticoids and other pulmonary medications on selected variables associated with exercise performance is summarized [5]. All of the untoward effects associated with glucocorticoids are significantly dosage dependent. Moreover, the deleterious side effects of glucocorticoids may be reduced in their severity, or forestalled, with the implementation of a regular aerobic exercise regimen and proper nutritional support. When anabolic steroids are used in conjunction with glucocorticoid therapy and exercise, trained patients with severe pulmonary disease have exhibited increased fat-free mass and improved ventilatory muscle strength and endurance in comparison with patients receiving only glucocorticoids [6]. Because one of the major objectives of pharmacologic intervention and exercise therapy is the improvement of a patient's ability to breathe at rest and during activity, it is important to understand the impact of obstructive and restrictive pulmonary diseases on the pattern of breathing [7]. Making this task difficult, however, is the fact that the exercise-related breathing patterns of patients with obstructive lung diseases have been described as falling somewhere between two extremes, rapid and shallow, or slow and deep. This apparent lack of agreement stems from the fact that patients with obstructive lung disease must rely to a greater extent on breathing frequency to augment minute ventilation, because their tidal volumes tend to be significantly smaller than normal due to the disease process [8]. As a consequence, patients with obstructive lung disease reach their maximum minute ventilation at lower work-loads than do asymptomatic individuals. Patients with obstructive pulmonary diseases are also hampered by a greater degree of alveolar and terminal respiratory unit hyperinflation compared with asymptomatic persons [9]. This leads to larger residual volumes and smaller tidal volumes, further decreasing efficiency and increasing the work of breathing. It seems that the greater the severity of the obstructive lung disease, the longer it takes to achieve an increase in tidal volume [10].

Discussion

Although patients with asthma and cystic fibrosis may demonstrate different pathological changes in the lung, the pattern of breathing during exercise is similar to that used by patients with chronic bronchitis and emphysernic obstructive lung disease. The exercise-related pattern of breathing exhibited by patients with restrictive lung disease also appears to be similar to that of patients with obstructive

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lung disease. Patients with restrictive lung disease appear to increase minute ventilation only minimally during exercise, and, as a result, there is a decrease in the total breathing cycle time due to a subsequent decrease in inspiratory time and a concomitant increase in inspiratory flow [11]. Because there is less opportunity to increase tidal volume and a greater degree of pulmonary hypertension in patients with restrictive lung disease, exercise tends to be terminated prematurely due to marked oxygen desaturation and dyspnoea. Thus, like the patient with obstructive lung disease, the patient with restrictive lung disease tries to increase minute ventilation by increasing breathing frequency, but only slightly increases tidal volume [12]. The appropriate and judicious use of supplemental oxygen can improve the metabolic activity and work capacity of skeletal muscle, as well as the breathing pattern, of patients with pulmonary disease who are hypoxic. Additionally, the use of bi-level positive airway pressure at rest and during exercise has been shown to significantly increase arterial oxygen saturation levels and decrease the respiratory rates of patients with end-stage lung disease. Careful prescription and titration of medications, as well as appropriate monitoring of the cardiorespiratory responses at rest and during exercise, can permit individuals with lung disease to participate to a greater extent in activities of daily living and exercise training programs [13]. Although the side effects of Nitric oxide are not completely understood, the marked oxidizing effects of nitrogen dioxide are known to decrease alveolar permeability, decrease partial pressure of carbon dioxide in arterial blood, and diffusing capacity of the lungs for carbon monoxide, cause pulmonary oedema, cause loss of cilia and disintegration of bronchiolar epithelium, and decrease pulmonary function tests i.e. forced expiratory volume in a second. The few studies investigating the toxic effects of Nitric oxide have shown that at concentrations of Nitric oxide greater than twenty parts per million, partial pressure of carbon dioxide in arterial blood, decreased and airway resistance increased. When used judiciously, oxygen therapy has few side effects. Dependent on the oxygen concentration and its duration of administration, however, oxygen toxicity can occur. In general, the pathological changes in the lungs associated with oxygen toxicity can be described in three phases, exudative, proliferative, and recovery. In the exudative phase, there is damage to the alveolar-capillary membrane, which results in an increased permeability to water, electrolytes, and protein. Secondarily, the capillaries become plugged with platelets, and the interstitium is invaded by polymorpho-nuclear leukocytes. As lung damage progresses, the inflammatory response is intensified [14]. In the proliferative phase, fibroblasts and type eleven epithelial cells proliferate in conjunction with interstitial collagen deposition. The recovery phase may result in complete healing or areas of fibrosis. Regardless of oxygen aetiology, arterial hypoxemia is the most common indication for oxygen therapy. The therapeutic administration of oxygen can elevate the arterial oxygen tension and increase the arterial oxygen content which shifts the oxy-haemoglobin dissociation curve to the right, improving peripheral tissue oxygenation. Additionally, the constriction of the central pulmonary vascular beds that is associated with hypoxia can be reduced. Oxygen therapy, therefore, can be quite beneficial in the reduction of the abnormally high pulmonary arterial pressures seen with pulmonary hypertension, both at rest and with exercise. Systems for such low gas flow rates are generally prescribed on the assumption that the patient is breathing at a relatively constant rate and depth. When breathing patterns deviate from the norm, however, patients cannot be assured of receiving the intended fraction of inspired oxygen. Therefore, when higher gas flow rates are indicated e.g. respiratory rate greater than thirty respirations per minute, or more accurate titrations of fraction of inspired oxygen, are required, oxygen is typically delivered by any one of several types of moderate-and high-flow face masks providing oxygen concentrations, or higher. The approximate oxygen concentrations achieved with different oxygen delivery devices. Care must always be exercised to avoid the potential for depression of the hypoxic drive to breathe in patients with chronically elevated partial pressure of carbon dioxide in arterial blood, levels primarily patients with Chronic obstructive pulmonary disease. It is often very difficult to establish the causative agent in an acute pulmonary infection because trans-oral sputum is often contaminated, yielding mixtures of multiple organisms on culture. Nevertheless, the organisms Diplococcus pneumonia and Hernophilus influenza are generally thought to be the primary causative agents of infection of the respiratory mucosa in patients with chronic obstructive pulmonary dysfunction. Precise diagnosis generally requires that sputum samples be obtained by trans-tracheal aspiration, bronchoscopy, or trans-pulmonary aspiration. There are not many effective drugs for the treatment of viruses in humans.

Conclusion

Research into this area of pharmacology, however, is in a period of explosive discovery. Interferons represent just one of the areas of potential pharmacologic and physiologic benefit, advancements with vaccines is another. Fungal and protozoal infections have historically been associated with tropical and subtropical environments, or with less-developed areas of the world where sanitation and hygiene are inadequate. Recently, however, the incidence of these infections has become more prevalent because immune-insufficiency, acquired or induced, is more widespread. The use of antifungal and antiprotozoal agents, therefore, is becoming more common.

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Conflict of Interest

None

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