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DIVING - WHAT TO TELL THE PATIENTS WITH ASTHMA AND WHY?

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INTRODUCTION

Traditionally, asthma has been considered to be an absolute contraindication to dive. Breath-hold diving was the only means of performing underwater work until the 17th century when a primitive diving bell was used to salvage cannons from a sunken ship in the Stockholm Harbor (1). The era of modern diving was spawned by Emile Gagnon and Jacques Cousteau's development of the self-contained underwater breathing apparatus (scuba) demand regulator (2). This regulator automatically delivers the diver's required tidal volume at the ambient pressure to which the diver is exposed and allows exhalation into the water. Scuba revolutionized commercial and recreational diving; presently, more than 5 million individuals participate in this activity in the United States alone (3,4). As the scuba diving community expanded, the population of divers with a history of asthma increased. Surveys of the sport-diving community have indicated that 5 to 9% of respondents had a history of asthma (3). British clinicians noted that divers in the United Kingdom have admitted to diving within 2 hours of a wheezing episode, without any pneumothorax or gas embolism developing (5). These findings, along with the fact that there are a growing number of potential recreational divers who have a history of asthma, have renewed debate as to the safety of allowing people with asthma to scuba dive.

PHYSIOLOGIC EFFECTS OF DIVING

A more recent study that compared 28 Navy divers, 31 diving candidates, and 59 healthy male nondiving volunteers showed a significant increase in airway reactivity to histamine-bronchoprovocation challenge among divers; the amount of increase appeared to correlate to the length of diving experience (7). Changes in flow rates and airway reactivity noted in long-term compressed air divers are similar to the changes that occur in the airways of patients with asthma.

PATHOPHYSIOLOGY OF ASTHMA AND RELATIONSHIP TO DIVING

Unfortunately, asthma is one of the most common chronic diseases worldwide and the prevalence is increasing, especially among children.

Fortunately, asthma can be treated and controlled so that almost all patients can prevent troublesome symptoms night and day, serious attacks, require little or no reliever medication, have productive, physically active lives and at the end have (near) normal lung function (8).

Asthma causes recurring episodes of wheezing, breathlessness, chest tightness, and coughing particularly at night or in the early morning. Regarding the definition asthma is chronic inflammatory disorder of the airways. Chronically inflamed airways are hiperresponsive; they become obstructed and airflow is limited (by bronchoconstriction, mucus plugs, and increased inflammation) when airways are exposed to various risk factors among others exercise.

What we know about exercise-triggered asthma indicates that some sports are more or less likely to provoke asthma. Swimming, water polo, and diving seem to be the least asthmogenic, since the athletes breathe in warm, humid air while exercising (8).

The small airways of the respiratory system (< 2-4 mm in diameter) have been termed the "silent zone" of the lungs. Only approximately 10% of airways resistance can be attributed to the small airways, and abnormalities in these airways are not detected by measuring FVC or FEV1 in humans (10). A recent symposium was dedicated to the role of small airways dysfunction in asthma (10). Evidence was reviewed that showed that small airways are inflamed in patients with asthma; if unchecked, this inflammation may result in airway remodeling, which has the potential to develop into chronic airflow obstruction. Superficially, this is similar to the changes that occur in the maximal mid-expiratory flow rates of long-term scuba divers (8-10).

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SHORT AND LONG TERM EFFECTS OF DIVING ON THE LUNG

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ABSTRACT

Barotrauma is the consequence of compression or expansion of a gas filled body cavity during descent or ascent in diving. As pressure increases, the volume of gas in exposed spaces decreases (Boyle's law). If the diver ascends while breath-holding or there is a failure to exhale (e.g. active asthma, chronic bronchitis or other obstructive diseases), the lung gas will attempt to expand even to the levels that damage lung tissue (70-80 mmHg). Pulmonary barotraumas causes embolisation of other organs, especially brain, resulting in various neurological consequences and skeletal muscles leading to elevated levels of CK enzyme. Pulmonary decompression sickness (the chokes) is the result of sudden, massive blocking the pulmonary arterial circulation by bubbles. This is a rare condition in diving, usually occurring with very rapid ascents from deep dives, often from failure of buoyancy control and presents with cough, dyspnoea, chest tightness and fall in systemic blood pressure. In addition to the air embolism, pulmonary overpressure may cause pneumothorax, mediastinal, or subcutaneous emphysema. It has been reported that divers after a certain period develop some degree of air flow obstruction due to airway narrowing (decrease in the ratio between forced expiratory volume in the first second and forced vital capacity- FEV1/FVC). Another long term effect is lower diffusion capacity, which may be explained by exposures to increased pressures of oxygen. The most important aspect of investigating long term health effects is prevention. The diving environment requires a thorough knowledge of diving physiology and physics and a commitment to safety. Key words: Scuba diving, pulmonary barotrauma, decompression sickness

INTRODUCTION

Diving is an underwater activity in which environmental conditions affect body structure and functions (1). All diving disciplines are exposed to a high level of accidents, which may be considered inherent to this activity (2). Different etiopathogenic factors are responsible for a wide variety of disorders. Many of them are linked to physiological mechanisms of adaptation to the underwater environment.

On land at sea level, the human body is exposed to an ambient pressure of 1 bar (100kPa). Under water pressure rises rapidly and linearly with depth, so that for every 10m of sea water descent, pressure increases by 100kPa (1). As pressure increases during the descent of diving, the volume of gas in exposed spaces decreases according to Boyle's law, which results in reduction of air spaces in the lungs, middle ear, paranasal sinuses, and the digestive system. Displacement of tissues into the diminishing volume of these spaces causes localized barotrauma ("squeeze"), which may cause injury to the involved tissues (3).

During the ascent of diving, the gas can form bubbles which has two consequences: they may block blood vessels or initiate an inflammatory response. Blockage of vessels results in ischemia and infarction of tissues beyond the obstruction, and inflammatory changes can cause extravasation into the tissues, further compromising the circulation and resulting in edema, scarring, and long-term damage to various organs, especially brain and spinal cord. The most severe manifestations of these physiologic processes are decompression sickness (DCS) and alveolar rupture or cerebral air embolism, secondary to arterial gas embolism (AGE) (4, 5).

There are two main types of diving. The simplest form is breath hold diving in which no equipment is used and the duration and depth of the dive depend on the individual's ability to remain functional during a single breath hold. Most recreational diving is performed using a breathing apparatus that contains air such as SCUBA (self contained breathing apparatus) (1).

The lungs are very vulnerable to the changes of the ambient pressure during diving. In this article we discuss the possible short and long term effects of diving on the lungs, current therapy and prevention attempts.

SHORT TERM EFFECTS

The most common short term effects of diving on the lungs are pulmonary barotrauma, air embolism and decompression sickness (DCS). Barotrauma is the consequence of lung expansion during the ascent. It can also occur during rapid descent (lung squeeze), but this is very rare (1). If the diver breathes normally and properly ventilates the increasing gas volume, no lung expansion will occur; however, if the diver ascends while breath-holding, the lung gas will attempt to expand and further ascent will cause intrapulmonary pressure to increase to levels that damage lung tissue. Overpressure of 70- 80 mmHg initiates damage to the lung. The greatest danger from gas expansion occurs near the surface, where the rate of volume change, for a given range of ascent in the water, is the greatest (3). If something impedes exhalation, such as airway narrowing in asthma or chronic bronchitis, the lungs may rupture. The most common situations when barotrauma of ascent

occurs are where compressed gas supply runs out at depth or during loss of buoyancy control (1). Clinical features result from emboli in the brain and present as unconsciousness, dizziness, chest pain, convulsions, paralysis, visual disturbances, headache, nausea, confusion, personality changes (3). In addition to air embolism, pulmonary overpressure may cause pneumothorax, mediastinal emphysema, or subcutaneous emphysema. Blast injury is a unique form of pulmonary barotrauma that produces extensive damage and air embolism. When a blast occurs in air or water, there is an initial high-pressure wave. In that case lung injury includes severe barotrauma with pulmonary hemorrhage and pulmonary edema.

Air embolism requires recompression in a hyperbaric chamber with 100% oxygen, and this should be provided as soon as possible. The purpose of recompression is making bubbles smaller and forcing them back into solution by putting the affected diver in a treatment chamber and increasing the ambient pressure (4, 5). Failure to treat a patient with cerebral air embolism can result in permanent neurological injury. The success rate of treatment protocols is 80-90% (3).

DCS occurs following a rapid decrease in ambient pressure, which results in supersaturation, a condition when partial pressure of gases dissolved in the tissue exceeds ambient pressure. This enables gas coming out of solution as bubbles, which causes tissue damage, blockage of lung blood vessels or different biochemical reactions at the bubble surface (3-5). Bubbles in blood and tissue injury result in activation of acute inflammation which involves complement and platelet activation leading to the formation of microemboli and altered endothelial function. When the gas volume is large, pulmonary obstruction occurs. A classic syndrome (chokes) is manifested by chest pain, dyspnea, and cough (2, 3). Decompression sickness usually develops some time after diving. Symptoms may develop within minutes of ascent or may appear 12- 24 hours later. On the other hand, pulmonary barotrauma with arterial gas embolism occurs immediately on ascent and may produce initial unconsciousness.

The symptoms of DCS can range from localized light pain, paresthesia, through focal neurological deficits to collapse, convulsions and death. The combination of DCS and air embolism occurs in divers who develop pulmonary barotrauma during ascent from a completed dive. The combination of excess of gas in tissues and free gas in the arterial system forms a particularly severe form of gas bubble injury (6). The treatment of DCS consists of recompression in hyperbaric chamber with periods of breathing 100% oxygen. Supportive therapy before initiation of hyperbaric oxygen includes intravenous infusion of saline to maintain hydration and plasma volume and use of antiplatelet agents (3).

The risk of developing DCS could be minimized by following certain calculated decompression schedules that describe particular ascent profiles for various combinations of dive depth and time.

One more interesting, short term consequence of diving on the lungs is acute pulmonary edema. The diver underwater develops increasing cough and breathlessness. This becomes more pronounced during the ascent of diving. Hemoptysis is often present and symptoms may persist for 24-48 hours after dive. The cause is still unclear, but some divers have episodes of pulmonary edema which may be linked to individual susceptibility. It seems more common among old divers and there is association with hypertension (7, 8).

LONG TERM EFFECTS

Divers frequently have unusually large lung volumes associated with a low ratio of forced expiratory volume in the first second and forced vital capacity- FEV1/FVC (FEV1%), suggestive of obstructive airways disease. This may be attributed to breathing hyperbaric air, venous gas microembolism during decompression, and hyperoxic exposure (9). Radiological evidence of the development of small airways disease in divers was not found despite of positive spirometry results, implying that there is no correlation with morphological abnormalities (10).

Another long term effect is lower diffusion capacity (3, 11, 12). This has been related to low-level oxygen toxicity as a result of breathing increased partial pressures of oxygen at depth and to the effects of venous gas emboli on the pulmonary microcirculation (13, 14). Animal studies demonstrated that venous gas embolism during decompression after hyperbaric exposure may cause an inflammatory response and capillary endothelial damage in the pulmonary microcirculation. It seems that repetitive inflammatory responses and injury may cause irreversible damage to the alveolar capillary membrane (15). Decreased diffusion capacity can lead to diminished exercise tolerance, but this has functional, rather than clinical significance (16).

CONCLUSION

In this article we reflected on short and long term effects of diving on the lungs. The most frequent short term effects are DCS and air embolism, however, very rarely, acute pulmonary edema may occur. These conditions are medical emergencies which require prompt therapy. Although the treatment is successful in 80-90% of cases, some residual symptoms (such as muscular weakness, paresthesia, problems with emptying bladder) may remain (3, 8). The long term effects of diving are decreased FEV1/FVC ratio and decreased pulmonary diffusion capacity. Lower FEV1/FVC ratio can be associated with obstruction of small airways due to narrowing. Deterioration of pulmonary

diffusion capacity normally occurs with age, but the process is accelerated in divers. Further research is needed to determine the exact cause of the mentioned long term effects. The diving environment requires a thorough knowledge of diving physiology and physics and a commitment to safety.

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