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REVIEW ARTICLE

A Review of Asthma and Scuba Diving

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ABSTRACT

An increasing number of asthmatics participate in recreational scuba diving. This activity presents unique physical and physiological challenges to the respiratory system. This review addresses the susceptibility of divers with asthma to diving accidents, acute asthmatic attacks, and long-term exacerbation of their disease. Recommendations on fitness to dive with asthma and airway hyperresponsiveness are provided.

Key Words: *Asthma; Diving; Lung; Pulmonary barotraumas; Review*

INTRODUCTION

Self-compressed underwater breathing apparatus (scuba) diving has become a popular leisure activity. There are almost 9,000,000 sport divers in the United States alone.[1] Epidemiological studies consistently indicate that 8% of the diving population reports themselves to have asthma.[2,3] Considerable demands on the lung, such as rapid changes in ambient pressure and physical exertion, may not be met adequately by asthmatic scuba divers, resulting in deterioration of the disease or a predisposition to even life-threatening injury. In particular, the risks for diving asthmatics comprise pulmonary barotrauma of ascent and its possibly

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fatal consequences, and the exposure to factors that irritate sensitized airways and eventually lead to asthmatic attacks from scuba diving.

It has long been considered that asthma is an absolute contraindication to recreational scuba diving.[4] Subsequently, more detailed views have evolved from the discussion on asthma and diving, and a tendency for less restrictive recommendations on medical fitness to dive with asthma was obvious,[5–9] mostly due to the increasing number of reports on asthmatics who dive without problems.[10–12] The wide range of available recommendations, however, may worry physicians and cause legal issues when issuing medical clearance.

Recent reports of a possible exposure to allergens even at depth, and of long-term lung function changes in scuba divers, provide new aspects to the discussion on recreational scuba diving and asthma. This article will review the mutual influence of asthma and recreational scuba diving. Since this is largely related to the physical peculiarities of gases in the underwater environment, a brief review of diving physics and physiology will be given.

DIVING PHYSICS AND PHYSIOLOGY

The physical and physiological effects of the underwater environment on the respiratory system are mainly characterized by the effects of altered ambient pressure. During descent, ambient pressure increases (compression), and during ascent from depth, the pressure decreases to sea level again (decompression). The barometric pressure at sea level is 101 kPa. Water pressure adds to the surface pressure at a rate of 101 kPa for every 10 m of sea water (msw), so at a depth of 40 msw, the ambient pressure is 505 kPa. The ambient pressure in turn affects the partial pressure of inspired gases. The partial pressures of oxygen (pO_2) and nitrogen (pN_2) at sea level are 21 kPa and 79 kPa. At 40 msw, pO_2 is over 101 kPa, comparable to breathing 100% oxygen at the surface.

During underwater scuba diving, the breathing gas is delivered from the scuba tank by a demand valve, the regulator. Modern regulators enable comfortable breathing at the common depth range associated with recreational diving. The scuba tank is filled with compressed gas, and the available gas volume for a dive can be calculated by the tank volume and the pressure under which the gas is kept. The breathing gas most commonly used in recreational scuba diving is compressed air. Air density is roughly proportional to ambient air pressure. The more the density increases, the more the ventilatory flow becomes turbulent in all airways, eventually leading to a decrease in dynamic lung volumes and flows and an increase in airway resistance. This will be pronounced with higher ventilatory demands during exercise. In consequence, a diminished breathing capacity and dyspnea would affect air-breathing subjects at considerable diving depths.[13] Due to the narcotic effects of increased pN_2, however, recreational compressed-air scuba diving is not recommended beyond depths of 30–40 msw.[4]

Henry's law states that the amount of a given gas dissolved in a liquid is directly proportional to the partial pressure of that gas. Thus, nitrogen as an inert gas dissolves in body tissues in a time- and depth-dependent manner during compression. Body tissues will get increasingly saturated with time the deeper the dive (i.e., the higher the pressure). During decompression, the fall in total ambient pressure forces pN_2 to decrease in body tissues, which is associated with bubble formation by gaseous nitrogen. Small nitrogen bubbles are formed within supersaturated tissues and the blood during and after ascent. Elimination of nitrogen bubbles usually follows by the venous return to the microcirculation of the lung. In order to allow all nitrogen bubbles to be eliminated by breathing and avoid excessive bubble formation, decompression time should correspond to the maximum depth of the dive and the time spent at depth. Moreover, the speed of ascent during recreational scuba diving should not exceed 10 msw/min.[14] In the case of a decompression failure, or in susceptible subjects, emerging nitrogen bubbles may cause symptoms by mechanical irritation within tissues or embolization of venous blood vessels. The syndrome associated with these effects is called decompression sickness.

According to Boyle's law, the volume of gas varies inversely with pressure. In ascending from 40 msw, the volume of air in the lungs will expand fivefold as pressure decreases. Overdistension of the pulmonary cavities can cause pulmonary barotrauma. The increase in volume, however, is greatest at shallow depths, because the largest decrease in total ambient pressure during ascent is between 10 msw and sea level. Transpulmonary pressure gradients (that is the difference between intratracheal and intrapleural pressure) of 8–9.3 kPa have been

shown experimentally to rupture the lungs in dogs.[15] This translates to breath-holds in open water while surfacing from less than 2 msw. Certain diving techniques, such as free ascents (without scuba) and emergency ascents with increased speed of ascent, may precipitate pulmonary barotrauma, due to the increased risk of voluntary breath holding.[16] Depending on the site of rupture, air may track along the perivascular sheaths of the pulmonary vasculature to cause mediastinal emphysema and pneumothorax. Air may also dissect into the pericardium or the retroperitoneum, and the subcutaneous tissues of the neck. The most feared sequela to pulmonary barotrauma of ascent is arterial gas embolism (AGE). This occurs when air enters the pulmonary vasculature and is carried to distant parts of the body as arterial air emboli. These emboli may occlude end arteries throughout the body and cause serious morbidity or even death.[17] The most frequent clinical feature of AGE in scuba diving is cerebral AGE, which is clinically characterized by a stroke-like syndrome with an onset time of less than 10 min after surfacing.[18]

DIVING ACCIDENT STATISTICS

There is no formal worldwide accident data reference system for scuba diving. Incidences of diving fatalities range within 1.3/100,000 dives to 2.68/100,000 diving participants, depending on regional prerequisites and database characteristics.[19] Arterial gas embolism has consistently been reported to rank second—besides drowning—among causes of diving fatalities, accounting for about one-third of all diving fatalities.[5,20,21] Annual incidences of nonfatal diving injuries such as AGE and decompression sickness among the recreational diving population are not well known, because little information is available on the number of dives made per year by sport divers. Among selected recreational scuba-diving populations, incidences ranging between 10/100,000 and 20/100,000 dives have been reported.[19,21] For AGE alone, an incidence of about 5/100,000 dives has been found in military divers.[22] Among selected high-risk populations such as submarine escape trainees who perform buoyant ascents with an increased speed of ascent, incidence of AGE may be significantly higher.[23]

One retrospective study[24] reviewed the accident database of Divers Alert Network, an international organization with headquarters at Duke University, covering the years 1987–1990. Of 1213 cases that had been reported to the database at that time, 196 were diagnosed as AGE and there were 54 divers who had a history of asthma, of whom 25 were currently asthmatic. A control group consisted of 696 divers with uneventful diving history, of whom 37 and 13 admitted to having a history of asthma or current asthma, respectively. Odds ratios were calculated in order to establish the ratio of the probability of AGE in a diver with asthma compared to the same probability without asthma. Odds ratios (with 95% confidence intervals) for asthmatics and current asthmatics were 1.58 (0.8–2.99) and 1.98 (0.65–5.33), respectively. The data suggested an approximately twofold increase in risk for AGE in current asthmatics, but did not reach statistical significance. The database over 8 years shows no statistical increase in risk for accidents.[25]

ASTHMA AND THE RISK OF PULMONARY BAROTRAUMA

Asthma is a chronic inflammatory disease of the airways with a variable degree of airflow obstruction, which is at least partly reversible, and airway hyperresponsiveness.[26] Acute bronchoconstriction, mucus plug formation, and airway edema contribute to airflow obstruction, resulting in an altered bronchopulmonary mechanical balance that is characterized by an increase in functional residual capacity. Widened airway caliber and air distribution through less stretched lung basal segments leads to intrapulmonary air trapping.[27] From a theoretical point of view, intrapulmonary air trapping increases the risk of pulmonary barotrauma, because expanding air during ascent may be unable to escape in time and eventually burst the lung following overdistension. In fact, air-trapping pulmonary lesions such as lung bullae and pleural adhesions have predominantly been found to be the underlying pathological sites of lung rupture by autopsy on diving fatalities.[28]

Apart from acute bronchoconstriction, clinical consequences of chronic inflammation and airway remodeling in asthma may intensify the risk of air trapping during ascent or may even independently predispose to pulmonary barotrauma. Residual airway obstruction and loss of elastic recoil have been described to be present in even asymptomatic

asthmatic patients.[26] Moreover, peripheral lung resistance may be increased in spite of normal FEV_1 in asthmatics. Thus, many asthmatics have physiological abnormalities even in the asymptomatic state of their disease. In a study on pulmonary function in 14 younger male divers with an uneventful medical history who had suffered from pulmonary barotrauma, static recoil pressure at maximum inspiration and an index of lung distensibility were significantly increased and decreased, respectively, when compared to 10 divers and 34 healthy volunteers who served as control groups.[29] It was concluded that relatively stiff airways may magnify the elastic stresses in peribronchial alveolar tissue, leading to septal rupture and interstitial gas dissection.

In two controlled retrospective studies, abnormalities in the flow–volume loops were frequent among patients who had suffered from pulmonary barotrauma when compared to divers whose injuries were otherwise caused,[30,31] indicating that a reduction in maximum expiratory flow rates may be a risk factor for pulmonary barotrauma. However, an analysis of all cases of pulmonary barotrauma that occurred during submarine escape training in the Royal Navy from 1975 to 1997[23] showed a greater correlation between forced vital capacity and pulmonary barotrauma ($p<0.01$) than between FEV_1 and pulmonary barotrauma ($p<0.05$).

Figure 1 demonstrates the theoretical principle that asthma may increase the risk of pulmonary barotrauma. For obvious ethical reasons, there are no data from controlled experimental trials studying pulmonary function and morphological changes in asthmatic humans after dives. In case reports of cerebral AGE in scuba divers, however, a relation to asthma has frequently been observed.[31–33]

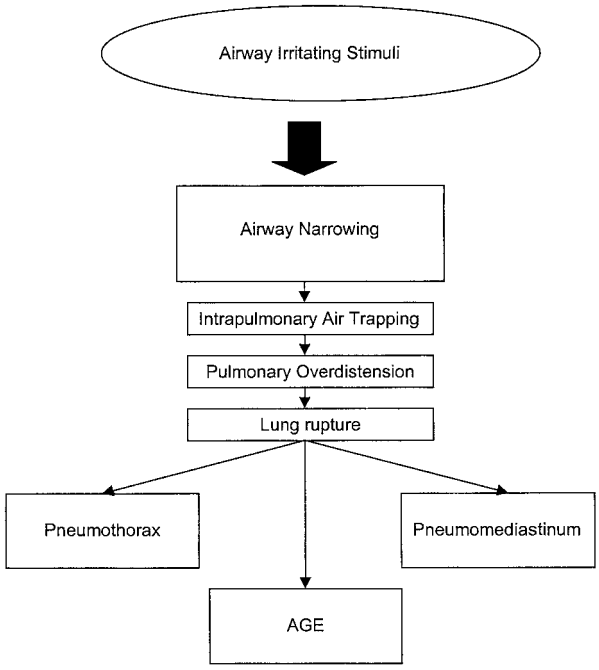

Figure 1. Pathophysiology of diving-related pulmonary barotraumas. A variety of diving-related airway-irritating stimuli may cause airway narrowing in susceptible subjects. Arrows indicate possible consequences.

ASTHMA AND THE RISK OF DIVING-RELATED ASTHMATIC ATTACKS

The chronic inflammation of the airways in asthma is associated with non-specific airway hyperresponsiveness, which may be defined as an increase in the ease in degree of airway narrowing in response to a wide range of bronchoconstrictor stimuli. These stimuli comprise factors such as exposure to allergens, viruses, indoor and outdoor pollutants, or exercise, and may even be modulated by emotion.[26,34] During scuba diving, there are several factors present that may irritate divers' airways. Table 1 summarizes a selection of important factors. Among them, inhalation of inadequately conditioned air and bypassing the nasal airways both precipitate evaporative water loss from airway mucosa, leading to local airway hyperosmolarity which may trigger bronchospasm. This mechanism is thought to be largely responsible for exercise-induced bronchoconstriction in asthmatics.[35] In subjects with clinically recognized asthma, the prevalence of exercise-induced bronchoconstriction has been reported to range from 70% to 80% in the laboratory, and from 45% to 73% in the field.[36]

Until recently, it was widely believed that the filtered, clean air used in recreational scuba diving should not cause bronchospasm.[37] But two recent case reports indicate that pollen may be trapped in the scuba tank and thus endanger the asthmatic diver. In one case, an asthmatic diver had refilled his scuba tank in a new area and the compressor was not fitted with an air filter.[38] Although being on inhaled steroid therapy (400 μg budesonide twice daily), the diver suffered from a life-threatening asthma attack during a dive caused by *Parietaria* pollen allergens.

Table 1

Factors that Affect Lungs and Airways During a Scuba Dive: (a) in a Permant Manner and (b) in Case of Equipment Malfunction or Under Particular Environmental Conditions

Factor	Occurrence	Effect
(a) Permanent		
Cold and dry breathing gas	Whole dive	Airway cooling and drying EIB
Increased gas density	Whole dive	Decreased flow velocity Increased work of breathing
Increased resistance from scuba	Whole dive	Increased airway resistance Increased work of breathing
Increased inspiratory pO_2 [a]	During compression and isopression	Acute toxicity at $pO_2 > 170$ kPa Chronic toxicity at $pO_2 > 50$ kPa
Inert gas loading [a]	During and after decompression	Reduced diffusion capacity Pulmonary hypertension
(b) Optional		
Breathing gas contamination with allergens	Whole dive	Immunological response in allergic subjects
Salt water aspiration	Temporary during dive	Airway irritation in susceptible subjects
Severe exertion (e.g., strong current)	Temporary during dive	Exhaustion Dyspnea

[a]These effects are present in extreme dives only, i.e., long and deep scuba dives > 40 msw with rapid decompression from depth.
EIB = exercise-induced bronchoconstriction.

In another case, a diver with a history of grass-induced hay fever repeatedly experienced attacks of dyspnea under water, each time after he had filled his tank from a certified portable breathing air compressor in an open seaside area containing abundant grasses.[39] These cases demonstrate an apparent risk of exposure to allergens when scuba tanks are filled in unprotected areas during the pollen season. Allergen-bearing microparticulates may pass through the filter of the compressor and will not be filtered by the nose during the dive.

Among further factors that potentially contribute to asthma attacks from scuba diving, aspiration of hypertonic sea water may lead to changes in osmolarity of airways mucosa and cause bronchospasm. This will, however, largely depend on the degree of airway hyperresponsiveness as well as the amount of aspirate, and has not been reported as a severe problem among asthmatic divers.

Apart from factors that may irritate the airways in susceptible subjects, mechanical limitation of breathing capacity caused by technical (e.g., diving equipment) and environmental factors must be considered in assessing pulmonary fitness to dive. The water surrounding the body (immersion) exerts a pressure on it that is different from intrapulmonary pressure, resulting in considerable cardiopulmonary changes such as intrathoracic blood pooling, a reduction in functional residual capacity, and an increased work of breathing. Moreover, breathing dense gas exerts a variety of effects on respiration that add to the effects of immersion. The impact of immersion and dense gas breathing on the healthy lung has been extensively reviewed elsewhere.[40] Briefly, breathing capacity at depth is affected by reduction of expiratory and inspiratory flows, and increased airway resistance. The flow–volume loop of a diver in a dense air atmosphere of 50 msw is akin to a patient with asthma in a state of expiratory flow limitation at sea level. Maximum voluntary ventilation of healthy volunteers while exercising at 30 msw has been found reduced by nearly one-half of that at sea level.[41] In healthy subjects, this ventilatory limitation will not necessarily result in reduced oxygen uptake. In chronic asthma, however, alveolar ventilation may fall when there is no compensatory increase in volume of air per minute. This reduction of alveolar ventilation will alter alveolar tension and result in hypercapnia and hypoxia.[27] One should be aware that exercise

ventilation during a dive may approach maximum voluntary ventilation, and a reduction in expired ventilatory volumes at rest will result in diminished breathing capacity under water.

AIRWAY HYPERRESPONSIVENESS AND DIVING

Airway hyperresponsiveness is an increase above normal in the degree to which airways will constrict in response to irritating stimuli. It is closely related to the underlying mechanisms of asthma as we currently understand them.[42] Airway hyperresponsiveness follows a normal distribution among the general population. Many individuals with no evidence of asthma or other respiratory disease show mild airway hyperresponsiveness to non-specific stimuli. A certain group of hyperresponsive subjects who are asymptomatic share a prognosis for subsequent asthma morbidity with those who are symptomatic,[43] while another group of asymptomatic subjects does not.[44]

The value of non-specific pharmacological inhalation challenges has been hotly debated in the diving literature.[5,8] A recent study which investigated 76 diving candidates with a history of recent or ancient asthma, or a history of allergic rhinitis, showed that nearly half of the subjects (47%) presented airway hyperresponsiveness to a carbachol challenge.[45] Only a few studies systematically investigated the association between airway hyperresponsiveness and diving. In a study of 28 consecutively recruited military scuba divers with a diving experience of at least 50 dives, 12 subjects (42.9%) significantly responded to a histamine challenge.[46] Most of these positive responses were of mild degree, in that provocation concentrations of histamine were greater than 2 mg/mL. Of 180 experienced military scuba divers with a diving experience of more than 250 dives on average, who underwent a cold air challenge, only three subjects (1.7%) showed a significant post-challenge decrease in FEV_1.[47] Occasionally, airway hyperresponsiveness has been reported to be present in single experienced divers elsewhere.[48,49]

Thus, among selected samples of scuba divers there are a larger number of asymptomatic divers with airway hyperresponsiveness who had dived uneventfully for years. There are as yet no data indicating an increased risk of diving-related injury in asymptomatic subjects with airway hyperresponsiveness. One study revealed pronounced but minor lung function changes after a deep dive to 50 msw in a small group of asymptomatic scuba divers with airway hyperresponsiveness as assessed by methacholine challenge.[49]

ASTHMA AS A CONSEQUENCE OF SCUBA DIVING?

Among a variety of possible factors that may adversely affect the airways in recreational scuba divers in the long term, inadequately conditioned air from scuba may play the most important role. Due to special processing, the air is dry in order to prevent corrosion of the tank, and becomes cold during expansion. It is inhaled through the mouth with the nose being covered by the face mask. Our knowledge of the effects of exercising in a cold environment on human airways has increased significantly in recent years. A high prevalence of asthma, asthma-like symptoms, and airway hyperresponsiveness has been reported in athletes, particularly those performing winter sports such as cross-country skiing, ice skating, and ice hockey.[50–53] Strenous training at low temperatures has been considered pathogenic in these athletes according to the concept of induction of exercise-induced bronchospasm. The mechanism thought to be primarily responsible for exercise-induced bronchospasm is evaporative water loss from the airway mucosa, resulting in increased osmolarity, or hyperemia, of the microcirculation in the airway wall.[35]

Although it has generally been accepted that even normal subjects respond to airway cooling in a dose–response relationship,[54] the possibility that healthy individuals may develop asthma because of sports activity has only recently been considered. In a canine model of exercise-induced hyperpnea, repeated exposure of canine peripheral airways results in airway inflammation, obstruction, hyperreactivity, and impaired smooth muscle responsiveness to β-adrenergic drugs.[55] It was concluded that hyperventilation of cold dry air may contribute to the development of airways disease, not just the exacerbation of asthma. Accordingly, inflammatory cellular exudate and increased subepithelial levels of tenascin, an extracellular matrix protein, were evident in endobronchial biopsies of elite, competitive cross-country skiers without prior asthma.[56] Interestingly, these inflammatory changes were

present irrespective of asthma-like symptoms, hyperresponsiveness, or atopy.

Scuba divers are exposed to inadequately conditioned air, as are subjects who exercise in cold environments. It may be argued that the amount of exposure differs in that athletes probably exercise more often and intensely than scuba divers dive, but scuba diving subpopulations such as diving instructors are likely to be exposed to a similar daily amount of cold air ventilation. In fact, there is one study that showed a significantly higher prevalence of airway hyperresponsiveness to histamine in military scuba divers when compared to non-diving control subjects, whereas divers and controls did not differ significantly with respect to atopy and smoking.[45] It is important to note that a shift from nose breathing to combined mouth and nasal breathing at ventilation levels exceeding 30 L/min may account for a greater deposition of inhaled particles to the lower airways, and inadequately conditioned air may reach the lower airways.[57] This mechanism may intensify the airways' response to dehydration injury in mouth breathing scuba divers when compared to other sports.

There is some evidence of non-asthmatic chronic airway changes from cross-sectional epidemiological studies on divers' pulmonary function, conducted in professional and military scuba divers.[47,58,59] A consistent finding from these studies was a reduction in expiratory volumes, and, more pronounced, in expiratory flows at low lung volumes, suggesting some degree of small airway dysfunction from extensive diving. Very recently, similar results were reported from one longitudinal study spanning a 6-year observation period in professional scuba divers.[60] The underlying pathophysiological mechanisms for these diving-related chronic changes in pulmonary function remain to be elucidated. The hypothesis, however, that asthma could be induced by extensive scuba diving is still preliminary and must be confirmed. But the presented studies may underscore the need for assessment of exercise-induced bronchospasm in organized scuba diving.

CONCLUSIONS AND RECOMMENDATIONS

General Considerations

Current epidemiological data are insufficient to determine the risk of asthmatics who scuba dive. This is mostly due to the low incidence of severe diving accidents, which would require a prospective study on a larger sample of asthmatic divers that has not been published to date. The presently available data on diving asthmatics[10,11] are flawed, because they rely on self-reporting of diagnoses and may not reflect all asthma-related events in scuba divers, since they don't capture asthmatics who quit diving. Moreover, the possibility that asthmatics who dive are likely to be self-selected, lower-risk subjects was not excluded. All that may be concluded from epidemiological studies so far is that asthmatics participate in scuba diving, and that selected samples of asthmatic divers may dive frequently and uneventfully.

Current recommendations on fitness to dive with asthma reveal a shift from an exclusionary view to a more differentiated approach,[6–9] implying that certain asthmatics may be allowed to dive. There is a worldwide consensus that subjects with acute bronchoconstriction or exercise-induced bronchospasm should not dive, whereas some controversy exists with respect to diving fitness in an asymptomatic episode of asthma. More conservative recommendations require that asthmatics should refrain from scuba diving for 5 years after the last disease exacerbation, despite maintenance of medication.[5] An interval of 48 hr after the most recent episode of bronchospasm, as has been recommended by some experts,[22] was considered an insufficient time to allow normalization of airways by others.[7] No data are available which support the use of prophylactic drugs before scuba diving, so that drug therapy in scubadiving asthmatics should rely on general treatment guidelines for asthma. Caution is warranted with regard to drugs that cause pulmonary vasodilation (e.g., methylxanthines), because they may increase pulmonary spillover of nitrogen bubbles to the arterial circulation.

The physician's responsibility is to screen the patient for possible medical contraindications and help the patient decide whether or not to dive. The physician should take into account that anyone may buy diving equipment and dive for recreational purposes without medical certification. An informed consent should underline that the patient received the information whether scuba diving is more risky for asthmatics than for the general population.[61,62] This approach may discourage the patient from ignoring the risks of scuba diving and using subterfuge to obtain a medical certificate when one is

required. It is the patient's level of understanding and acknowledging the risks of scuba diving that makes him dive more safely.

Recommendations

The following recommendations for fitness to dive with asthma rely on an established diagnosis and classification of disease severity, according to the current Guidelines for the Diagnosis and Management of Asthma, provided by the National Institute of Health.[42] It is important to note that asthma patients who may be allowed to dive not only should be asymptomatic but demonstrate normal lung function as indicated by spirometry and peak-flow protocol, since perception of bronchoconstriction is known to be inaccurate in a large number of asthma patients.[63]

1. Airway hyperreactivity to pharmacological stimuli alone does not preclude scuba diving. The value of testing airway hyperresponsiveness in the diving candidate lies in establishing the diagnosis and state of asthma. Since the degree of non-specific airway hyperresponsiveness in asthmatics is related to the severity of the disease and the fall in FEV_1 over time,[64] the degree of responsiveness is worth being considered. Caution should therefore be warranted in moderate to high-degree hyperresponsiveness in the diving candidate. However, an exercise or cold air challenge is preferable to a pharmacological one because of its higher specificity. Positive responses to cold air or exercise clearly indicate an absolute contraindication to diving.
2. Acute bronchoconstriction precludes scuba diving, until airway function has returned to normal and stabilized. An interval of at least two weeks with an FEV_1 above 80% of predicted and peak-flow variability below 20% is required before taking up scuba diving. Patients with childhood-only asthma who have been symptomless since may be allowed to dive.
3. Moderate to severe persistent asthma should preclude scuba diving. These disease states are functionally characterized by a reduction in FEV_1 and high variability in peak expiratory flow. Although clinical symptoms and lung function may improve following therapy, the pathophysiological changes associated with these disease states may increase the risk of acute exacerbations from scuba diving as outlined above.
4. Patients with mild intermittent or persistent asthma may be allowed to dive on an individual basis. Spirometric measures in the asthmatic diving candidate at rest should be within the normal range of predicted. It is strongly recommended that diving asthmatics monitor their peak expiratory flow once daily in the morning, and, if possible, before and after their scuba dives.

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