

## Diving-, Underwater-Cave Diving- and Caving-Related Respiratory Disorders and Diseases: Public Health Views

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### Abstract

Caving has increasingly become popular. In the United States, approximately 2 million people visit national park caves each year. Decompression illness, a common diving-associated disorder has been caused by bubbles in blood or tissues during or after a decrease in ambient pressure with encompassing two pathophysiological syndromes, namely “arterial gas embolism” and the more common “decompression sickness”. Three leading causes of morbidity and mortality among recreation divers are forming bubbles within bodily tissues, barotrauma, and drowning/near drowning. Scuba divers’ pulmonary edema, an uncommon disorder in healthy persons is more common in divers with advanced aging. Fortunately, the respiratory function, hypoxemia, abnormal radiological signs, and clinical symptoms in this disorder resolve rapidly in minutes or hours. The empirical treatment includes oxygen supplement, positive pressure respiration, and possibly diuretics. The explanations for swimming-induced pulmonary edema can be described as the following: 1) increased perfusion in the dependent lung with sidestroke swimming; 2) hydrostatic pressure effects; 3) increased pulmonary vascular resistance due to cold exposure; 4) pulmonary vascular blood pooling due to immersion; and 5) increased cardiac output due to physical exertion. Breath-hold diving-induced pulmonary edema is believed to be a feature of pulmonary barotrauma of descent or “lung squeeze” due to the reduction of lung volume below the residual volume developing according to Boyle’s Law. The hypothesized explanations for pulmonary edema in freediving can be described as the following: 1) pulmonary trauma due to “lung packing”; 2) increased cardiac output due to physical exertion; 3) increased pulmonary vascular pressure due to cold exposure; 4) pulmonary vascular blood pooling due to immersion; and 5) negative intra-alveolar pressure gradients due to descent. Pulmonary barotrauma of descent may present with chest pain and hemoptysis with hemorrhagic pulmonary edema. Treatment of this disorder includes 100% oxygenation, CPAP, and fluid replacement. Histoplasmosis or “cave disease”, the most common cave-associated disease can be acquired by occupationally or recreationally inhaled exposure to environmental sources. The diagnosis of histoplasmosis is made by identifying *Histoplasma capsulatum* in the clinical samples, testing serum and other body secretions for antigen or antibodies, radiological methods, and occasional transbronchial biopsy. The patients with symptomatic histoplasmosis should be treated with itraconazole, orally for 6 - 12 weeks or intravenous deoxycholate formulation of amphotericin B for 1 - 2 weeks and followed by itraconazole, for a total of 12 weeks.

**Keywords:** Caving; Underwater Caving; Diving; Respiratory Disorders; Diseases

### Abbreviations

AGE: Arterial Gas Embolism; CPAP: Continuous Positive Airway Pressure; *C. neoformans*: *Cryptococcus neoformans*; DCI: Decompression Illness; DCS: Decompression Sickness; FEF<sub>25-75</sub>: Mid-Expiratory Flows; FEV<sub>1</sub>: Forced Expiratory Volume in One Minute; FVC: Forced Vital Capacity; *H. capsulatum*: *Histoplasma capsulatum*; IPE: Immersion Pulmonary Edema; IV: Intravenous; kPa: kilopascal; O<sub>2</sub>: Oxygen; SD: Standard Deviation; SDPE: Scuba Divers’ Pulmonary Edema, S<sub>a</sub>O<sub>2</sub>: Arterial Oxygen Saturation; US: United States

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## Introduction

Caves, the first shelters and oldest sanctuaries of man. Caving has increasingly become popular. In the United States (US), nearly 2 million individuals visit national park caves each year [1]. Small numbers of sport cavers (cavers for those exploring wild caves) are at risk for some high risk conditions and expedition cavers (cavers for those exploring previously uncharted caves) are at risk for obscure infections [2]. Cave diving involves entering a flooded, overhead environment and is technique and equipment intensive [3]. Technical diving, frequently used by cave divers is defined as using techniques and equipment to execute longer or deeper dives than recreational dives [3]. Decompression illness (DCI), a common diving-related disorder has been caused by bubbles in tissue or blood during or after a reduction in ambient pressure with encompassing two pathophysiological syndromes, namely "arterial gas embolism (AGE)" and the more common "decompression sickness (DCS)" [3]. One technical cave diving project in a deep Mount Gambier, Australia revealed a DCS probability with 95% confidence interval of 10 - 340 per 10,000 dives (0.1 - 3.4%) [4], whereas DCI in recreational divers has been estimated at 0.96 per 10,000 dives (0.01%) in a cold-water recreational diving population [5]. In series studied by Harris, *et al.* in Australia revealed the estimated incidence of DCS of 2.8 : 10,000 dives that is consistent with other series studying DCI in recreational divers, but may potentially be higher (5 : 10,000 dives) [6]. The incidence of DCI varies between 1 : 10,000 to 9.5 : 10,000 depending on whether the divers are involved in technical [4], scientific [7,8], recreational [5,9-11], commercial [12] or military activities [13]. Three leading causes of morbidity and mortality among recreational divers are forming bubbles within bodily tissues, barotraumas due to expanding air while ascending and DCI, caused by the dissolution or forceful introduction of gas, and drowning/near drowning [14-19]. Making a rapid ascent, a loss of buoyancy control, and running out of air have been found to be related to these types of diving morbidity and mortality [19-24], according to three types of separated contributing factors: human, equipment, and environmental factors [18,25,26].

The most pronounced adjustments in diving response to breath-hold diving are selective peripheral vasoconstriction and bradycardia [27,28]. Both apnea and stimulation of facial cold-receptors (e.g. immersion) initiate diving response [29,30]. Most studies of human diving abilities and related reflexes have been based on laboratory studies, allowing advanced techniques and a controlled environment [30,31]. Apnea with face immersion is a model used by many laboratories to simulate diving [30,31]. Diving response has an apnea-prolonging effect and the arterial oxygen ( $O_2$ ) store is conserved during apnea both during exercise and rest [32-34]. Reduction of blood flow in tissues tolerant to hypoxia and lower myocardial  $O_2$  consumption during bradycardia result in  $O_2$  conservation and contributes to a slower pulmonary  $O_2$  depletion [35-37]. Nevertheless, in a natural diving situation, the diver's entire body is often constantly immersed in cool water, occurring during the apneas [38,39], in addition to the face immersion that its impact depends on the temperature difference between the skin and the water [31]. Cooling body will result in a cold-induced vasoconstriction accompanying possible abolishing  $O_2$  conservation [40]. A previous study demonstrated that simultaneous vertical body and face immersion in cold water at 20°C considerably reduced breath-holding time accompanying a strong bradycardia, whereas breath-holding in warm water at 35°C lengthened the breath-holding time without bradycardia, in comparison to breath-holding while sitting in the air [41]. These results were explained by chilling that increased respiratory drive and metabolic rate at 20°C [41]. Another study revealed that during continuous body immersion, apnea without facial immersion resulted in a bradycardia similar to that found while breath-holding with facial immersion with the body in the air [42]. It is not known to what extent  $O_2$  conservation applies to the immersed divers [40]. For the divers, Bruijn, *et al.* suggested that it is important to expose the facial area that mainly is triggered the diving response for achieving maximal  $O_2$  conservation [40] via the ophthalmic branch of the trigeminal nerve (i.e. forehead and eye regions) [43].

## Immersion pulmonary edema (IPE)

Most of cases were radiologically verified the diagnosis [44]. The clinical manifestations include cough in 82%, dyspnea in 80%, hemoptysis in 62%, crackles (rales) in 25% and wheezing in 10% [44]. A previous study revealed that chest pain, weakness, and confusion were less common [44]. The mean oxygen partial pressure was 66.2 (SD 17.4) mmHg (8.82 +/- 2.32 kPa) with a mean arterial oxygen saturation ( $S_aO_2$ ) of 88.8% (SD 7.3) [44]. The majority of divers resolved their symptoms within five minutes to 24 hours [44]. Among

the Special Forces combat swimmers, heavy exertion was incriminated. Considering the free divers, there was relationship between pulmonary edema and thoracic squeeze (pulmonary barotrauma of descent). Most of the affected free divers and swimmers were healthy. Among scuba divers, increased age was noted with pulmonary edema [44].

### Scuba divers' pulmonary edema (SDPE)

The actual incidence of SDPE, an uncommon disorder in healthy individuals is unknown [45-47]. This is probably due to under-diagnosis [45-47]. SDPE is more common in older divers that differs from other IPEs [47-50]. The respiratory function, hypoxemia, and radiological signs resolve rapidly (minutes or hours) in most cases as well as symptoms [46,51-53]. Treatment of SDPE includes oxygen supplement, positive pressure respiration, and possibly diuretics [54].

### Swimming-induced pulmonary edema (SIPE)

Extreme exertion in both cold and warm (> 20°C) water have frequently associated with dyspnea and pulmonary congestion during surface swimming [44,51,55-57]. A previous trial on strenuous swimming time demonstrated that 8 of 30 young male swimmers were affected of SIPE with a water temperature of 23°C within the first 45 minutes [57]. All these swimmers had consumed 5 liters of water prior to the swim for countering the anticipated dehydration, thus, over-hydration may have contributed to SIPE [57]. Two swimmers had repeated episodes without the provocation of such over-hydration and extreme exercise, whereas the swimmers wore only bathing suits and fins [57]. A previous study on  $S_aO_2$  and spirometry findings in 29 incidents from 21 of 35 young male swimmers exposed to strenuous swimming over a two-month period, demonstrated the same changes as an Israeli military swimming fitness programs (70 cases of SIPE with productive cough, chest pain, wheezing, hemoptysis, and inspiratory crackles) in water temperature of 19.6 (SD) 3.2°C [56], with a fall in  $S_aO_2$  from 99% to 91% and significant lowering of the forced expiratory volume in one minute ( $FEV_1$ )/forced vital capacity (FVC) ratio, pre-incident FVC, mid-expiratory flows ( $FEF_{25-75}$ ) and FVC [58]. These changes may be predictive of SIPE [54]. The pathophysiologically common presentation was hypothesized to be exertion and increased inspiratory load with hydrostatic effects of immersion superimposition that contribute to capillary stress, aggravated these effects in the dependent lung by the lateral decubitus swimming position (sidestroke), particularly the older swimmers with a cardiac basis [54]. Finally, the explanations for SIPE can be described as the following: 1) increased perfusion in the dependent lung with sidestroke swimming; 2) hydrostatic pressure effects; 3) increased pulmonary vascular resistance due to cold exposure; 4) pulmonary vascular blood pooling due to immersion; and 5) increased cardiac output due to physical exertion [46,48,56,58].

### Freediving, pulmonary barotrauma of descent, and pulmonary edema

Through a combination of anatomical and physiological factors and responses and of modern diving techniques, breath-hold dives to a depth of over 200 meters have been achieved in recent times. Breath-hold diving-induced pulmonary edema is believed to be a feature of pulmonary barotrauma of descent, "lung squeeze", due to the lung volume reduction below the residual volume developing according to Boyle's Law [44,59-61]. The hypothesized explanations for pulmonary edema in freediving can be described as the following: 1) pulmonary trauma due to "lung packing"; 2) increased cardiac output due to physical exertion; 3) increased pulmonary vascular pressure due to cold exposure; 4) pulmonary vascular blood pooling due to immersion; and 5) negative intra-alveolar pressure gradients due to descent [54]. The clinical manifestations of pulmonary barotrauma of descent may include chest pain and hemoptysis with hemorrhagic pulmonary edema [44]. Treatment includes 100% oxygenation, CPAP, and fluid replacement [44].

### Respiratory infectious diseases-related to caves

The most common cave-related disease, histoplasmosis ("cave disease") can be acquired by recreationally or occupationally inhaled exposure to environmental sources [62]. *Histoplasma capsulatum* (*H. capsulatum*), fungal causative agent of histoplasmosis favors its propagation in the caves [62]. Histoplasmosis may become more common as a recreational disease [62]. *H. capsulatum* can grow in soil and is enhanced by bird and bat excrement that provides a source of nitrates, which accelerate its spore formation [63]. With depending on the intensity of primary inhalation exposure and the immunity of the host, the severity of illness and clinical features vary [64]. Clinical manifestations in acute exposure to *H. capsulatum* vary from asymptomatic infection to severe pneumonitis with respiratory compromise

[64]. A previous case report of immunocompromised host demonstrated clinical features with fever, cough, and dyspnea [65]. The majority of the cases resolve their illness within one month, without treatment [64]. Clinicians should consider histoplasmosis in the differential diagnosis of febrile illness in returning travelers with history of geographic or epidemiological exposure [64].

Diagnosis of histoplasmosis is made by identifying *H. capsulatum* in the clinical specimens, testing the body fluids, such as urine, serum, etc. for *H. capsulatum* antigen, and testing serum for antibodies to this microorganism [66]. Transbronchial biopsy occasionally may be diagnostic [66]. In cases with mild-to-moderate acute pulmonary histoplasmosis, treatment is usually unnecessary, but treatment is needed in symptomatic patients with more than one month of having symptoms [67]. These symptomatic patients should be treated with itraconazole (200 mg, 3 times daily for 3 days and then 200 mg once or twice daily for 6 - 12 weeks), whereas deoxycholate formulation of amphotericin B (0.7 - 1.0 mg/kg daily, intravenously (IV)) for 1 - 2 weeks, followed by itraconazole (200 mg, 3 times daily for 3 days, and then 200 mg twice daily, for a total of 12 weeks) is recommended in patients with moderately severe to severe acute pulmonary histoplasmosis [67]. Travelers who visit caves in the areas of endemic histoplasmosis should consider using personal protective equipment to prevent *H. capsulatum* exposure during caving [62].

### Potentially respiratory transmitted diseases-related caving

Other diseases, although not epidemiologically associated to patients like cryptococcosis, psittacosis, and histoplasmosis duboisii are also potentially transmitted in caves [2]. Inhalation exposure to *Cryptococcus neoformans* (*C. neoformans*) and *Chlamydia psittaci*, cause of psittacosis may be a health risk in environments containing accumulation of bat droppings, respiratory secretions, aerosolized urine, or dried manure of infected birds [66]. *C. neoformans* uses the creatinine in bird feces as a nitrogen source [66]. *H. capsulatum* var. *duboisii* was isolated from soil admixed with bat guano in a cave in Nigeria [68]. Use of personal protective equipment is also recommended to protect against inhalation exposures to these potentially respiratory transmitted microorganisms associated caving [66].

### Discussion

It was hypothesized in a report that “labile hypertensives” with an exaggerated vasoconstrictor response to cold and/or raised oxygen pressure would be particular prone to pulmonary edema development as a result of an increase in after-load due to systemic vasoconstriction, a pre-load stress from the pulmonary vascular blood volume increase that occurs with immersion [55]. This hypothesis incriminated a vascular hyper-reactivity to a cold stimulus in SDPE, but other reports that SDPE was related to relatively warm or tropical waters [47,49,50-52,69]. Persons with SDPE were advised to forgo scuba diving [48]. Pulmonary edema can be caused by increased capillary permeability [44,55]. Characteristically, symptoms rapidly resolve once the diver is out of the water [45-47,51]. Nevertheless, many case histories demonstrated expectoration of bloody froth that indicate pulmonary capillary damage [45-47,51]. According to most authors, advanced age is a predisposing factor of SDPE [44,46,47,52,53,55]. Impaired respiratory function, hypertension, ischemic or other cardiac diseases are detrimentally enhancing effects of age in SDPE [44]. Other pulmonary diseases, such as asthma, cold urticaria, gas contaminations, and respiratory oxygen toxicity to which divers exposed may produce pulmonary edema [70]. Diving disorders, such as pulmonary barotrauma, deep diving dyspnea, and pulmonary decompression sickness may cause diagnostic confusion with uncomplicated SDPE [70]. Currently, although symptoms of SDPE and decompression effects occur on or soon after ascent, no relationship has been documented between SDPE and decompression effects [47,50,71]. Pulmonary bubble filtration during decompression in deeper scuba diving may damage capillary integrity and increase pulmonary hypertension, thus, increasing the likelihood of pulmonary edema [47,50,71]. Cause of SDPE is most likely in the combination of cold, compressed-air breathing stresses, and immersion imposed on the respiratory and cardiovascular systems [46]. A deleterious influence of tight wetsuits, a restriction to respiration may be more likely to increase the work of breathing, therefore increasing dyspnea in SDPE cases, rather than leading to the disease itself [72]. In freediving, pulmonary edema is explained by the barotraumatic effects of descent, whereas in some cases, both exercise and pulmonary volutrauma may contribute [54]. The clinical features of histoplasmosis frequently start with low-grade fever and cough that is easily missed diagnosis of community-acquired pneumonia [65]. Several previous studies have revealed that patients with histoplasmosis from endemic areas with evidence

of past histoplasmosis, largely did not develop histoplasmosis despite undergoing varying immunocompromising therapeutic regimens: chemotherapy, anti-tumor-necrosis-factor (TNF) therapy, or transplant therapy [73]. It is essential for clinicians to be aware of a patient's short-term and long-term history of residence or travel, as well as risk factors and geographic distribution for this fungal disease. Histoplasmosis and coccidioidomycosis have been included as traveled-associated illness in the "Health Information for International Travel Yellow Book (<http://www.cdc.gov/travel/diseases.htm>) [74].

## Conclusion

The mechanisms of SIPE include immersion and severe exertion, including hydrostatic effects and thoracic blood pooling on pulmonary circulation. Considering SDPE, the effects of immersion, the vascular response to hypothermia, the increase in pulmonary vascular blood volume, the negative intra-pleural pressures generated from exercise, the negative hydrostatic pressures of head-out and vertical positioning, inadequate diving equipment, and gas density, including existing cardiovascular and diving-induced diseases all may contribute to the causes. Caving preparation carefully needs to be executed and planned, including vaccination, prophylactic medications, and other protective measures. Expedition cavers may require more specific measures that are suitable for visited areas. Further clarified studies are urgently needed.

## Author's Contributions

Dr. Attapon Cheepsattayakorn conducted the study framework and wrote the manuscript. Associate Professor Dr. Ruangrong Cheepsattayakorn contributed to scientific content and assistance in manuscript writing. Both authors read and approved the final version of the manuscript.

## Competing Interests

The authors declare that they have no actual or potential competing financial interests.

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