Case reports

Haemoptysis in breath-hold divers; where does it come from?

Igor Barković[1,2,3], Vitomir Maričić[4], Boris Reinić[3], Frano Marinelli[1,2,3], Tamara Turk Wensveen[5]

[1] *Center for Research and Education in Underwater, Hyperbaric and Maritime Medicine, University of Rijeka, Faculty of Medicine, Rijeka, Croatia*
[2] *Department of Pulmonology, Clinic for Internal Medicine, Clinical Hospital Center Rijeka, Rijeka, Croatia*
[3] *Department for Underwater and Hyperbaric medicine, Clinical Medical Center Rijeka, Rijeka, Croatia*
[4] *AIDA - International Association for the Development of Apnea, Croatia*
[5] *Specialized Hospital for Medical Rehabilitation of Cardiac, Pulmonary and Rheumatic Diseases, Thalassotherapia Opatija, Opatija, Croatia*

Corresponding author: *Associate Professor Dr. Sc. Igor Barković, dr. med. Tome Strižića 3, 51000 Rijeka, Croatia*
igor.barkovic@uniri.hr

Key words
Bronchoscopy; Case reports; Freediving; Hemoptysis; Pulmonary barotrauma; Pulmonary oedema

Abstract

(Barković I, Maričić V, Reinić B, Marinelli F, Wensveen TT. Haemoptysis in breath-hold divers; where does it come from? Diving and Hyperbaric Medicine. 2021 September 30;51(3):299–302. doi: 10.28920/dhm51.3.299-302. PMID: 34547782.)
Introduction: The aim of reporting these two cases is to present visual evidence by bronchoscopy of the origin of haemoptysis in two elite breath-hold divers.
Case reports: Two male elite breath-hold divers of similar physical characteristics presented to our clinic after performing dives of up to 75 and 59 meters of seawater depth for 2:30 and 2:35 (minutes:seconds) respectively. Both patients presented with haemoptysis. Lung ultrasound was performed. The first patient had crackles on chest auscultation, overt pulmonary oedema clinically and 90 ultrasound lung comets. The second patient had no oedema or crackles, but presented with 20 ultrasound lung comets. Video bronchoscopy was performed which showed traces of blood coming from all three segments of the right upper lobe in both patients. The rest of the airways and lungs were intact.
Conclusions: These finding suggest that the apical parts of the lungs are the most prone to deep-dive induced damage. The precise mechanism of lung barotrauma and haemoptysis in breath-hold divers remains to be elucidated. These findings may be of importance for a better understanding of the underlying pathology of haemoptysis.

Introduction

Breath-hold diving is an extreme sport in which people may dive to substantial depths without any breathing equipment. Breath-hold divers (BHD) may suffer from pulmonary oedema, haemoptysis and chest pain after performing moderate to deep dives. There are case reports that describe these events; however, to date none report examination by bronchoscopy immediately after diving to definitively localise the source of active bleeding.[1]

The mechanism of pulmonary complications in deep BHDs remains unclear so the aim of this case report was to present two cases of haemoptysis caused by breath-hold diving and to demonstrate the place of origin of this bleeding in the respiratory system.

BHDs are exposed to a unique physiological condition, which combines extreme ambient pressure with physical exercise under prolonged hypoxic conditions. As such, our study identifies the weakest structural point in the healthy adult lung, which may be of importance for the understanding of lung pathology associated with haemoptysis.

Case reports

Both divers consented to the publication of their case details.

Two male elite BHDs presented to our clinic on separate occasions for assessment and treatment for haemoptysis after deep breath-hold diving. Both patients had similar general characteristics. Patient 1 was 34-years-old, 185 cm tall, with a weight of 84 kg. The depth of his dive that day was 75 metres of seawater (msw) and the duration of the dive was 2:30 minutes. Patient 2 was 28-years-old, 181 cm tall and weighed 85 kg. His dive was up to 59 msw and lasted 2:35 minutes.

Both divers had spirometry and plethysmography measurements before the incident dives during regular

medical checkups. Total lung capacity (TLC) and residual volume (RV) were taken as means of three measurements. For Patient 1, TLC was 9.56 L and RV 2.12 L. For Patient 2, TLC was 10.55 L and RV 2.58 L. According to Boyle-Mariotte's law (Volume = 1 / Pressure), TLC at maximal depth for both BHDs was less than RV at the surface.

Both patients were admitted to the Emergency Department of the Clinical Medical Centre in Rijeka around three hours after their last dives and were immediately examined by a pulmonologist. The first patient remained dyspnoeic during low level of exertion, and had haemoptysis and pulmonary crackles on chest auscultation. The second patient had symptoms immediately after the dive but they had resolved before arrival to hospital and chest auscultation was normal. Hospital admission was recommended to both patients but both refused.

Lung ultrasound was performed with a GE LogicV convex probe (3.5 to 5 Mhz) (GE Healthcare, Chicago, IL, USA) on both divers to detect extravascular lung water by counting the number of B-lines or ultrasound lung comets (ULCs).[2] The ULC is defined as an echogenic, coherent, wedge-shaped signal with narrow origin arising from the pleural line and extending to the far edge of the viewing area.[3] Sixty-one predetermined chest sites were used to calculate ULC and the sum of all scanning areas was recorded.[4] The first of the two patients who had crackles on chest auscultation and clinically overt pulmonary oedema had 90 ULCs. In the right apical area atelectasis was observed. The second patient had 20 ULCs and no signs of pulmonary oedema. It was hypothesized that more ULCs had been present, but were mostly resolved since the dive, as has been described previously.[4,5]

Video bronchoscopy was performed with an Olympus Bf-1T180 fibreoptic bronchoscope (Olympus Medical Systems, Tokyo, Japan) around 3.5 h after the last dive for both divers. In both divers traces of blood coming from all three segments of the right upper lobe (RUL) bronchus were visualised (Figures 1 and 2). The upper airway and trachea as well as the left bronchial tree and the right middle and lower lobe of the lungs were normal in appearance in both divers.

Discussion

Lung barotrauma arising from compression and its symptoms such as haemoptysis and lung oedema have been described in breath-hold divers before;[6] however, the anatomic origin of the blood and the cause of these symptoms remains unclear.

The current explanation of these phenomena is a combination of cardiovascular changes during immersion, increased hydrostatic pressure and apnoea. During breath-hold diving, the ambient pressure increases proportionally with depth and the volume of air in the lungs decreases (Boyle-Mariotte's law). When total lung capacity is reduced to residual volume, the volume of air in the lungs cannot decrease anymore and a further increase in ambient pressure will result in negative pressure in the thorax. This will increase blood shift to the thorax.[7] Immersion in water and apnoea induce an autonomic response that drives peripheral vasoconstriction

Figure 1
Blood found in patient 1 by video bronchoscopy in the right upper lobe of the lung, three and a half hours after performing a breath-hold dive lasting 2:30 minutes to 75

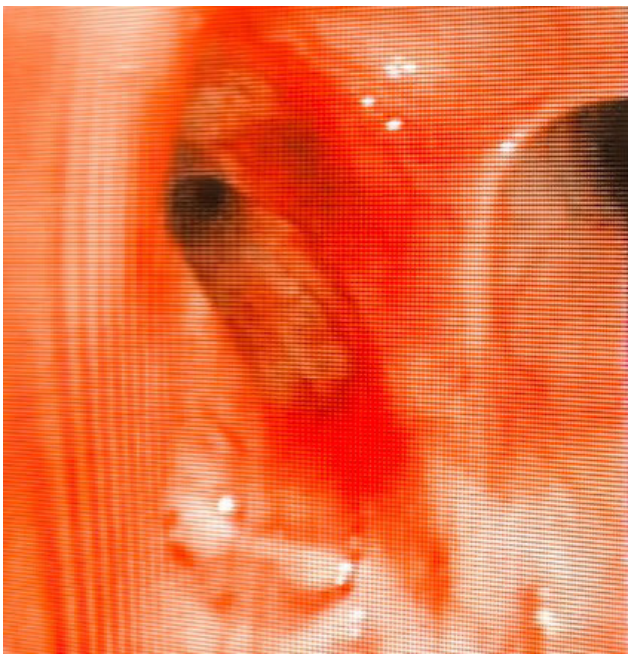
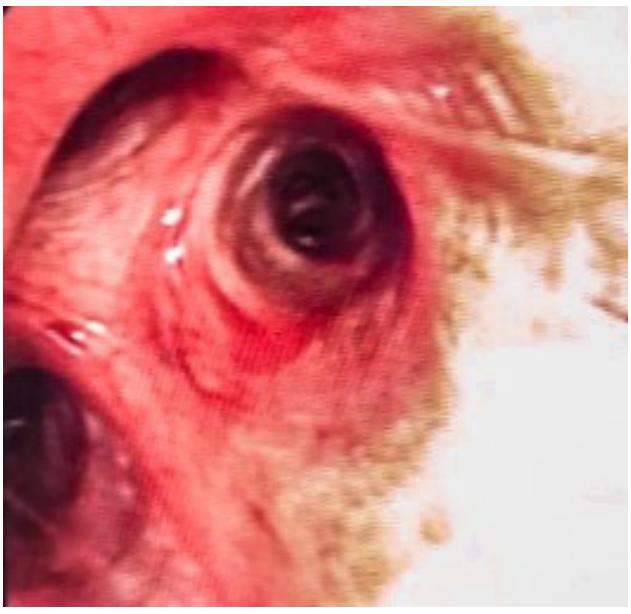


Figure 2
Blood found in patient 2 by video bronchoscopy in the right upper lobe of the lung, three and a half hours after performing a breath-hold dive lasting 2:35 minutes to 59 msw



and bradycardia.[8] Immersion also induces a 'buoyancy effect' on blood shift to the thorax as the effect of gravity is lost. Thus, immersion, increased hydrostatic pressure and apnoea cause a large blood shift to the thorax and significant pulmonary vascular engorgement. If pulmonary capillary pressure exceeds oncotic pressure, transudation from capillaries occurs and can lead to pulmonary oedema. Massive blood shift can significantly increase transmural pulmonary capillary pressure. In addition, negative pressure in the alveoli can increase transcapillary pressure and possibly result in endothelial damage and stress-induced capillary failure.[9]

Lung oedema in BH divers has previously been confirmed with X-ray,[10] computed tomography,[11] ultrasound[4] and pulmonary function tests.[12] There have been case reports of haemoptysis in which bronchoscopy with bronchoalveolar lavage was done but only several days after admission to hospital. One study confirmed haemoptysis in breath-hold divers with laryngoscopy and showed that the bleeding had its origin below the vocal cords.[12] Direct visualisation of bleeding during haemoptysis had not previously been described. We now show that traces of blood were visualised with a bronchoscope coming from all three segments of the right upper lobe bronchus following deep-dives.

These findings pose the question why the right upper lobe is the origin of the bleeding in both divers. It is true that the lung has a vertical difference of pleural pressure and there has been no research into how human lungs compress, collapse and re-expand under high pressure. Such research would be very difficult to obtain underwater and could be potentially dangerous to BHDs. One study reported a computational model of the mechanics of airway and alveolar collapse in humans during deep dives.[13] Lung mechanics under pressure cannot be determined by applying Boyle-Mariotte's law alone, because lungs and airways differ in structure, compliance, perfusion and surfactant over their various anatomical regions and will not collapse and reopen equally. One study found a lack of ventilation in several apical regions of divers' lungs when a small volume was inhaled after below residual volume exhalation in healthy subjects at sea level, which could also point towards the idea that apical parts of the lungs are the first to collapse.[14]

The paranasal sinuses have very little compliance, and their volume will remain almost constant throughout a dive. The mouth and supraglottic compartments are very compliant and will collapse and reopen without consequences, tracheal volume will decrease by inward invagination of its flexible posterior wall, and the anterior part will compress to a smaller extent. The small airways are more compliant than alveoli and will collapse prior to alveoli. Autonomic regulation of smooth muscle cells in the lower airways has an important role in controlling compliance and at the same time collapse and reopening of the alveoli. During breath-hold diving the airway pressure will remain the same throughout the lungs and airway but pleural pressure will depend on body position. If a diver dives head-first, the highest pleural pressure will be in apical parts of the lungs and apical alveoli will shrink faster.[13] As a result, their closing volume will be reached at shallower depth than for basal alveoli. When the diver reaches maximal depth, turns and ascends head towards surface apical alveoli will be under the lowest pleural pressure and will tend to re-open. Reopening of closed alveoli does not happen simultaneously in a homogenous pattern.[13]

Conclusion

To our knowledge this is the first report in which the exact part of the airway where bleeding originates in pulmonary barotrauma of compression ('lung squeeze') has been identified. Both of these divers had blood in the RUL bronchus; other parts of upper airways and lungs were normal in appearance. We conclude that in healthy adult lungs, the apical parts of the right lobe may be the most vulnerable to deep-dive induced stress. The precise mechanism of lung squeeze and haemoptysis in BHDs remains to be elucidated. Our findings may be of importance for a better understanding of pathologies associated with haemoptysis.

References

1 Boussuges A, Pinet C, Thomas P, Bergmann E, Sainty JM, Vervloet D. Haemoptysis after breath-hold diving. Eur Respir J. 1999;13:697–9. doi: 10.1183/09031936.99.13369799. PMID: 10232449.

2 Lichtenstein D, Meziere G, Biderman P, Gepner A, Barre O. The comet-tail artifact. An ultrasound sign of alveolar-interstitial syndrome. Am J Respir Crit Care Med. 1997;156:1640–6. doi: 10.1164/ajrccm.156.5.96-07096. PMID: 9372688.

3 Picano E, Frassi F, Agricola E, Gligorova S, Gargani L, Mottola G. Ultrasound lung comets: a clinically useful sign of extravascular lung water. J Am Soc Echocardiogr. 2006;19:356–63. doi: 10.1016/j.echo.2005.05.019. PMID: 16500505.

4 Frassi F, Pingitore A, Cialoni D, Picano E. Chest sonography detects lung water accumulation in healthy elite apnea divers. J Am Soc Echocardiogr. 2008;10:1150–5. doi: 10.1016/j.echo.2008.08.001. PMID: 18926391.

5 Ljubkovic M, Gaustad SE, Marinovic J, Obad A, Ivancev V, Bilopavlovic N, et al. Ultrasonic evidence of acute interstitial lung edema after SCUBA diving is resolved within 2-3 h. Resp Physiol Neurobi. 2010;171:165–70. doi: 10.1016/j.resp.2010.02.008. PMID: 20188217.

6 Cialoni D, Maggiorelli F, Sponsiello N, Tonerini M, Frammartino B. Epidemiological investigation on hemoptysis in breath hold divers. In: Bedini R, Belardinelli A, Reale L, editors. Blue 2005 Human behaviour and limits in underwater environment. Special conference on breath-hold diving Pisa, Italy: University of Chiety; 2005. p. 103–4. [cited 2021 Jan 21]. Available from: http://apnea.cz/media/products/BLUE_2005_ABSTRACTS.pdf.

7 Schaefer KE, Allison RD, Dougherty JH Jr, Carey CR, Walker R, Yost F, et al. Pulmonary and circulatory adjustments determining the limits of depths in breathhold diving. Science.

1968;162(3857):1020-3. doi: 10.1126/science.162.3857.1020. PMID: 5725383.
8 Panneton WM. The mammalian diving response: an enigmatic reflex to preserve life? Physiology (Bethesda). 2013;28:284–97. doi: 10.1152/physiol.00020.2013. PMID: 23997188. PMCID: PMC3768097.
9 Fitz-Clarke JR. Computer simulation of human breath-hold-diving: cardiovascular adjustments. Eur J Appl Physiol. 2007;100:207–24. doi: 10.1007/s00421-007-0421-z. PMID: 17323072.
10 Prediletto R, Catapano G, Fornai E, Carli C, Reale L, Passera M, et al. Stress of pulmonary gas exchange in breath hold dives. In: Bedini R, Belardinelli A, Reale L, editors. Blue 2005 Human behaviour and limits in underwater environment. Special conference on breath-hold diving Pisa, Italy: University of Chiety; 2005. p. 105–6. [cited 2021 Jan 21]. Available from: http://apnea.cz/media/products/BLUE_2005_ABSTRACTS.pdf.
11 Kiyan E, Aktas S, Toklu AS. Hemoptysis provoked by voluntary diaphragmatic contractions in breath-hold divers. Chest. 2001;120:2098–100. doi: 10.1378/chest.120.6.2098. PMID: 11742946.
12 Lindholm P, Ekborn A, Oberg D, Gennser M. Pulmonary edema and haemoptysis after breath-hold diving at residual volume. J Appl Physiol. 2008;104:912–7. doi: 10.1152/japplphysiol.01127.2007. PMID: 18202166.
13 Fitz-Clarke JR. Mechanics of airway and alveolar collapse in human breath-hold diving. Respir Physiol Neurobiol. 2007;159:202–10. doi: 10.1016/j.resp.2007.07.006. PMID: 17827075.
14 Muradyan I, Loring SH, Ferrigno M, Lindholm P, Topulos GP, Patz S, et al. Inhalation heterogeneity from subresidual volumes in elite divers. J Appl Physiol (1985). 2010;109:1969–73. doi: 10.1152/japplphysiol.00953.2009. PMID: 20864566. PMCID: PMC3006407.

Conflicts of interest and funding: nil

Submitted: 21 January 2021
Accepted after revision: 18 April 2021