招請講演
Health effects of diving

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The diving exposure is complex. There are several factors associated with diving that may have acute and long term health effects. Effects on bone, the lung, the central nervous system (CNS) have been and are still of major concern.

All dives can be characterized by the simple physical parameters pressure, time and gas mixture. Exposure to hyperoxia, decompression stress and gas density are all directly related to these physical factors irrespective of diving methods. Other factors that are not specific for diving such as submersion, respiratory heat and water loss and breathing equipment may have additional health effects, and environmental factors such as pollution and work related psychological stress may pose an increased risk to the diver.

Effects of diving on the lung

Acute effects of hyperoxia (oxygen toxicity), venous gas microembolism (decompression stress) and increased respiratory load (gas density, breathing equipment) have been extensively studied both experimentally and in relation to operational diving [1～5]. The acute effects on the lung are well characterized and the effects of single exposures appear to be almost fully reversible. However, epidemiological studies, both cross-sectional [6～11] and longitudinal [12, 13], indicate that there are residual effects that over time accumulate to a long term effect. In most of these studies there has been a relationship between cumulative diving exposure and the rate of decline in lung function. Physiologically, the long term changes in lung function in divers are of an obstructive pattern.

Effects of diving on the central nervous system

Acute effects of diving on the CNS are related to gas mixture and compression rate, such as nitrogen narcosis and the high pressure nervous syndrome. These effects have so far been considered fully reversible. It is the effects of free gas and gas microembolism causing decompression illness (DCI) and the sequelae of DCI that are of concern. Long term sequelae can be demonstrated in 30-50% of subjects treated for neurological DCI. It is well known that venous gas microemboli are commonly demonstrated after uneventful dives without any symptoms of DCI. It is also well known that in a considerable number of subjects there is arterialization of these microemboli, still without obvious symptoms of DCI [14,15].

Neurological symptoms and signs are on the other hand often found in subjects with joint or skin DCI where the neurological symptoms are not dominant [16]. In her study of long term effects of diving in saturation divers, Todnem et al. [17, 18] demonstrated that the prevalence of neurological symptoms, signs and EEG pathology was related to a history of DCI. When correcting for that, there was still an effect of cumulative diving exposure. Later, perfusion MRI scanning has demonstrated perfusion deficits in the water-shed areas of the brain in a group of these divers [19].

Other organ systems

Bone necrosis and its relationship to decompression stress are well described. Long term effects of diving on the vestibular system and hearing has been reported, which could be due to diving and exposure to noise [20, 21]. Acute effects on the audio-vestibular system including barotrauma should be well known.

Work related events and psychological stress

Diving operations are technically complex and may take place in a rough environment. In a survey of former North Sea divers, most had encountered one or more life-threatening events during diving. The ones most commonly encountered were gas cut, entrapment/entanglement and proximity to falling objects. Two thirds of the divers had participated in recovering dead bodies. Several events were likely to cause posttraumatic stress reactions as assessed by the Impact of Event Scale (IES-R) [22].

Morbidity and mortality

Health related quality of life as assessed with the SF-36 questionnaire has been shown to be reduced in former North Sea divers who were professionally active up to the 1990ies [23]. The prevalence of respiratory, cardiovascular and neurological disease was, however, not increased compared to the general population, but there was an increased prevalence of musculo-skeletal and psychological symptoms. In a survey of the Norwegian divers included in the registry of the Labour Inspectorate – including most of the Norwegian professional divers working inshore and offshore (n＝5526) - overall mortality was not increased. However, mortality due to cardiovascular, cerebral and respiratory disease, and neoplasms were lower, but mortality due to

accidents and suicide was higher when compared to the general male population of the same age [24]. This finding is of concern with respect to the high prevalence of adverse events and traumatic experiences during diving which may cause posttraumatic stress reactions.

References

1) Thorsen E, Segadal K, Reed JW, Elliott C, Gulsvik A, Hjelle JO. Contribution of hyperoxia to reduced pulmonary function after deep saturation dives. J Appl Physiol 1993; 75: 657 - 662.
2) Suzuki S. Probable lung injury by long-term exposure to oxygen close to 50 kilopascals. Undersea Hyperb Med 1994; 21: 235-243.
3) Thorsen E, Hjelle J, Segadal K, Gulsvik A. Exercise tolerance and pulmonary gas exchange after deep saturation dives. J Appl Physiol 1990; 68: 1809 - 1814.
4) Dujic Z, Obad A, Palada I, Valic Z, Brubakk AO. A single open sea air dive increases pulmonary arterial pressure and reduces right ventricular function in professional divers. Eur J Appl Physiol 2006; 97: 478-485.
5) Thorsen E, Skogstad M, Reed JW. Subacute effects of inspiratory resistive loading and head-out water immersion on pulmonary function. Undersea Hyperb Med 1999; 26: 137 - 141.
6) Thorsen E, Segadal K, Kambestad B, Gulsvik A. Divers' lung function: small airways disease? Br J Ind Med 1990; 47: 519 - 523.
7) Tetzlaff K, Friege L, Reuter M, Haber J, Mutzbauer T, Neubauer B. Expiratory flow limitation in compressed air divers and oxygen divers. Eur Respir J 1998; 12: 895-9.
8) Adir Y, Shupak A, Laor A, Weiler-Ravell D. Large lungs in divers: natural selection or a training effect? Chest 2005; 128: 224-228.
9) Watt S. Effect of commercial diving on ventilatory function. Br J Ind Med 1985; 42: 59-62.
10) Dmitrouk AI, Gulyar SA, Ilyin VN, Moiseenko EV. Physiological mechanisms of adaptation of divers to the condition of deepwater in the antarctic. In: Proceedings of 16th annual meeting of the European Undersea Biomedical Society (Sterk W, Geeraedts L, eds), Amsterdam 1990: 311-9.
11) Suzuki S, Thorsen E. Long term effects of saturation diving on pulmonary function – reduction in FEF50% and FEF75% in early period of diving career. XXVIIIth annual meeting of European Undersea Biomedical Society, Brugge, 2002 (abstract).
12) Skogstad M, Thorsen E, Haldorsen T, Kjuus H. Lung function over six years in professional divers. Occup Environ Med 2002; 59: 629-633.
13) Thorsen E, Segadal K, Kambestad B, Gulsvik A. Pulmonary function one and four years after a deep saturation dive. Scand J Work Environ Health 1993; 19: 115 - 120.
14) Brubakk AO, Peterson R, Grip A, Holand B, Onarheim J, Segadal K, Kunkle TD, Tønjum S. Gas bubbles in the circulation of divers after ascending excursions from 300 to 250 msw. J Appl Physiol 1986; 60: 45-51.
15) Ljubkovic M, Zanchi J, Breskovic T, Marinovic J, Lojpur M, Dujic Z. Determinants of arterial gas embolism after scuba diving. J Appl Physiol 2012; 112: 91-95.
16) Sundal E, Grønning M, Troland K, Irgens Å, Aanderud L, Thorsen E. Risk of misclassification of decompression sickness. Int Marit Med 2011; 62: 17 - 19.
17) Todnem K, Nyland H, Kambestad BK, Aarli JA. Influence of occupational diving upon the nervous system: an epidemiological study. Br J Ind Med 1990; 47: 708-714.
18) Todnem K, Skeidsvoll H, Svihus R, Rinck P, Riise T, Kambestad BK, Aarli JA. Electroencephalography, evoked potentials and MRI brain scans in saturation divers. An epidemiological study. Electroencephalogr Clin Neurophysiol 1991; 79: 322-329.
19) Moen G, Specht K, Taxt T, Sundal E, Grønning M, Thorsen E, Troland K, Irgens Å, Grüner R. Cerebral diffusion and perfusion deficits in North Sea divers. Acta Radiol 2010; 51: 1050-1058.
20) Molvær OI, Albrektsen G. Hearing deterioration in professional divers: an epidemiologic study. Undersea Hyperb Med 1990; 17: 231-246.
21) Goplen FK, Grønning M, Irgens A, Sundal E, Nordahl SH. Vestibular symptoms and otoneurological findings in retired offshore divers. Aviat Space Environ Med 2007; 78: 414-419.
22) Troland K, Sundal E, Irgens A, Grønning M, Thorsen E. Potentially traumatic events and posttraumatic stress reactions in retired North Sea divers. XXXVIIth annual meeting of European Undersea Biomedical Society, Gdansk 2011 (abstract).
23) Irgens Å, Grønning M, Troland K, Sundal E, Nyland H, Thorsen E. Reduced health related quality of life in former North Sea divers is associated with decompression sickness. Occup Med 2007; 57: 349-354.
24) Irgens Å, Troland K, Thorsen E, Grønning M. Mortality among professional divers in Norway. Occup Med 2013 ; 63 (8) : 537-43.