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Wheezing in a commercial diver due to disinfectant

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Watt SJ, Wheezing in a commercial diver due to disinfectant. Undersea Biomed Res 1991; 18(4):347–349.—A case is described of a saturation diver with no previous history of asthma who repeatedly developed work-related symptoms of asthma at pressure, which appear to be causally related to the use of dichlorophen as a disinfectant agent. Although challenge tests were negative, suggesting that dichlorophen may have been acting as an irritant rather than as a sensitizer, the symptoms were abolished by the use of an alternative disinfectant agent. The potential importance of this effect in a diver is discussed, and the case highlights the importance of the use of nontoxic agents in the diving environment.

commercial divers disinfectant alternative dichlorophen wheezing

air flow obstruction

Occupational asthma has been reported resulting from exposure to several sterilizing or disinfectant agents including glutaraldehyde (1) and chlorhexidine (2). Other agents such as Stericol have also been implicated as a possible cause, although their action may be primarily as an irritant rather than as a sensitizer. A case is reported here in which wheeze seems to be related to exposure to dichlorophen, a chemical used both as a disinfectant and as a herbicide.

In saturation diving, divers remain at constant pressure in a living chamber in which the breathing gas is normally a helium-oxygen mixture. Bacterial contamination of chambers is a serious health hazard as Gram-negative bacteria, particularly *Pseudomonas*, thrive in the environment and contamination may be associated with severe otitis externa. Regular cleaning and decontamination procedures are carried out and dichlorophen has been widely and successfully used for this purpose, although alternative, less toxic agents are also available. The case has important implications as symptoms occurred at pressure and are an important contraindication to diving because of the increased risk of pulmonary barotrauma.

CASE HISTORY

A 29-yr-old man had been a commercial diver for 6 yr with 4 yr experience of saturation diving. Three days after the onset of a saturation dive at storage depth of

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30 m in helium-oxygen environment (oxygen partial pressure 40 kPa) he developed symptoms of chest tightness and dry cough while sleeping. He subsequently developed exertional breathlessness and productive cough over a few days. He was initially treated with antibiotics and decompressed slowly, uneventfully. Symptoms improved during the decompression but had not resolved completely when seen for examination 1 wk later.

He reported experiencing similar symptoms during each saturation dive over the previous 2 yr and that symptoms had become progressively worse and commenced earlier with successive dives. His symptoms had always resolved within a few days of completing a dive and he had no symptoms at other times. He had no previous history of asthma, eczema, or hay fever, but had had a brief episode of sore eyes, running nose, and cough after a sea anemone bite 2 yr previously. There was no family history of asthma.

Examination revealed some expiratory wheeze but no other abnormality. Dynamic lung volumes were within normal limits, forced expiratory volume (FEV₁) 4.05 liter, forced vital capacity (FVC) 5.30 liter, FEV₁:FVC ratio 76% with peak flow of 480 liter · min⁻¹. Histamine challenge suggested a mild increase in bronchial hyperactivity (PC20 3.6 μg · ml⁻¹) (3) and reproduced his symptoms. Static lung volumes were normal (total lung capacity 6.54 liter, functional residual capacity 2.96 liter, residual volume 1.41 liter) as with single-breath transfer factor for carbon monoxide (35.7 ml · min⁻¹ · torr⁻¹). Serum IgE was 99.9 U · liter⁻¹, RAST screen to common allergens was negative, and titers to atypical microorganisms showed no evidence of recent infection.

He was advised not to dive and his residual symptoms resolved without treatment over the next few days. He remained well, and at examination 10 wk later was entirely normal. Dynamic lung volumes had increased (FEV₁ 4.40 liter, FVC 5.70 liter, FEV₁:FVC ratio 77% with peak flow of 520 liter · min⁻¹). A repeat histamine challenge test remained in the same range (PC20 5.0 μg · ml⁻¹).

It seemed likely that dichlorophen, a recognized irritant, was responsible for the symptoms. Challenge tests were performed in which the patient reproduced the chamber disinfection protocol in a diving chamber at surface pressure in an air environment heated to a temperature (26°-28°C) equivalent to that required in a heliox environment at pressure. Two challenge tests lasting 2 h each using dilutions of dichlorophen of 1:1000 and 1:100 failed to induce symptoms or any alteration in FEV₁ or peak flow during the subsequent 24 h.

The challenge test could not reproduce accurately either the environmental conditions with heliox at pressure or the duration of exposure that had been associated with symptoms, and it was felt that dichlorophen remained a likely causative agent. Inasmuch as less toxic disinfectant agents than dichlorophen are available and have been successfully used, it was proposed that he carry out a further dive in the same diving system after a change in the disinfectant agent. The chamber system was cleaned with an amphoteric biocide (TEGO) and the diver made a further saturation dive without any recurrence of symptoms. A peak flow record maintained by both the diver and his bell partner showed a small fall in peak flow during the initial phase of the dive but thereafter it remained stable. The change at the start of the dive was seen in both divers and relates to environmental conditions, including the increase in gas density on pressurization.

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In addition to the absence of respiratory symptoms, all 6 divers in the chamber system reported a substantial improvement in superficial skin peeling, which had been a regular feature of previous dives.

COMMENT

This diver's symptoms are historically clearly related to the saturation diving environment. Possible causes for his symptoms include dichlorphen, microbial contamination of the chamber environmental control unit, exposure to chemical constituents of drilling muds (which may contaminate divers suits and hence the chamber environment), and an unrecognized feature of pulmonary oxygen toxicity. Microbial contamination seems unlikely because of the repetitive nature of symptoms and because the environmental control units are cleaned between dives. No contact with drilling muds was reported during the last few dives.

Removal from the environment of the most likely causative agent abolished the symptoms. The diver's previous medical history and histamine challenge tests suggest that his marginal increase in bronchial hyperreactivity was not normally associated with symptoms or airflow obstruction, although the acute episode may have been associated with an increase in hyperreactivity. Failure to reproduce his symptoms by dichlorophen challenge may be related either to the exposure dose because it was not feasible to reproduce the environmental conditions without considerable risk, or to the duration of exposure because symptoms always took several days to develop. It is unlikely that dichlorophen acted as a sensitizer, but this case may represent either irritant-induced occupational asthma (4) or acute episodes of airflow obstruction in a man with marginal hyperreactivity. It is interesting that symptoms did not occur in other divers similarly exposed, and despite the prevalence of hyperreactivity (3), symptoms have not previously been reported although dichlorophen has been extensively used in the diving industry for many years. Whatever the underlying mechanism, it is important to recognize that airflow obstruction, an important risk factor for pulmonary barotrauma, may be induced during diving as a result of exposure to dichlorophen in individuals with no previous history or symptoms of asthma. This case adds further support to the view that alternative and less-toxic disinfectant agents should be used in preference to dichlorophen in the diving environment.

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