

# ANOMALOUS REACTIONS TO RESPIRATORY LOADING DURING DRY EXERCISE AT DEPTHS TO 305 m

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## INTRODUCTION

New breathing resistance **limits** for Underwater Breathing Apparatus were tested during 7 research saturation dives involving 31 divers at depths to 450 msw. The **limits** were derived by a retrospective analysis of earlier data (1), and describe in probabilistic terms the influence of breathing resistance on exercise tolerance. As predicted, divers frequently could not complete 20 min of heavy exercise when breathing through moderate resistances. Surprisingly, however, two divers nearly lost consciousness. Those two incidents and the **unusual** symptoms accompanying them are described below.

## METHODS

One event occurred at 46 msw while ~~the~~ diver was breathing **air** (1.2 ATA O<sub>2</sub>), the other at 305 msw while the diver was breathing 0.4 ATA O<sub>2</sub> in **He**. Divers exercised dry on a cycle ergometer for 5 min at 50 watts, then for 20 min at 150 watts. Wearing an **AGA full** face mask, the divers breathed through a resistor with a two-way valve separating inspired and expired flows. Inspiratory flowrate **was** monitored by a Rudolph screen pneumotachometer, and the diver's **EKG** was recorded on a strip chart or FM recorder. A mass spectrometer continuously analyzed gas sampled at the mouth. The divers provided a modified Borg dyspnea score at 2 min intervals.

Neither diver **was** a smoker, but both had significant pulmonary histories. The first was allergic to ragweed, the second had childhood asthma and exposure **to both asbestos and granite dust** as a young adult.

## RESULTS

Both divers completed exercise without difficulty in control runs with low resistances. At 46 msw with a moderate resistance (**incident 1**) one diver suddenly experienced graying of vision and vertigo after 2 min at 150 watt. Immediately before the event the diver had described only slight breathlessness (score of 2, with 11 maximum). Inspiratory flowrate was low ( $1.3 \text{ l} \cdot \text{s}^{-1}$ ) and minute ventilation (**RMV**) was **only**  $30 \text{ l} \cdot \text{min}^{-1}$ , but end-tidal CO<sub>2</sub> did not exceed 59 mm Hg. Before the event, peak-to-peak mouth pressure (**DP**) averaged 21 **cm % O**.

A different diver at 305 msw gradually experienced increased breathlessness to a very severe level (score 9 of 11) during the 150 watt workload. The sudden appearance of visual symptoms caused ~~the~~ diver to stop work abruptly after 11 min at 150 watts. Just before termination, RMV was  $50 \text{ l} \cdot \text{min}^{-1}$ , and DP was 30 cm H<sub>2</sub>O. The diver reported **lightheadedness, dizziness** defined as an inability to maintain balance, scotomas, and tunnel vision. Tunnel vision lasted about 1 min after the diver removed the mask, but **dizziness** cleared within seconds.

Both divers maintained normal **sinus rhythm with** no unusual changes in heart rate. In the **first** diver, **maximal** exercise heart rate was  $120 \text{ min}^{-1}$ , in the second it was  $163 \text{ min}^{-1}$ . Headaches were absent. Peak-to-peak mouth pressures never exceeded  $30 \text{ cm H}_2\text{O}$ , and were equally distributed over inspiration and expiration. There was no hydrostatic loading since divers were not immersed, so mean intrathoracic pressure should have been **nil**. From the probabilistic model, *the* predicted probabilities of diver discomfort were **similar**, **0.04** and 0.17.

## DISCUSSION

There is **no obvious** explanation for these incidents. A **common cause** of unconsciousness in divers,  $\text{CO}_2$  narcosis secondary to hypoventilation, was **unlikely** because end-tidal  $\text{CO}_2$  was **normal** for **exercising** divers, and the characteristic  $\text{CO}_2$  headache was absent.

In some **individuals**, syncope *can* be caused by large positive intrathoracic pressures created by straining or Valsalva maneuvers. **Although** not a common occurrence, venous pooling may **paradoxically** result in vagal induced bradycardia and severe **hypotension**. However, in these cases intrathoracic pressures should not have been elevated (mouth pressure was low) and there was no **bradycardia**. Furthermore, inspiratory efforts **against** a resistance promote venous return **more** than expiratory efforts impede it. **Consequently**, resistances **with** equal inspiratory and expiratory components cause few cardiovascular perturbations. Breathing through resistors *can* induce **large** increases in peripheral bloodflow (2), but there are no reports of associated changes in arterial or central venous pressure.

**Exertional** syncope is usually a pathological sign, **as in** aortic stenosis. In normal **individuals**, leg exercise promotes forearm vasoconstriction, thus **reducing** peripheral blood flow. However, when ventricular baroreceptors are **stimulated** by high left ventricular pressures (due to stenosis) vasodilatation can occur, **resulting** in syncope (3). While we cannot rule this mechanism **out**, its action in healthy divers is unlikely.

Pulmonary history may provide the only **clue** to the cause of these unusual events. We have postulated that the **hyperbaric** environment accentuates physiological differences **among** divers. **Theoretically**, minor functional deficits that do not **impair** exercise at the surface can be **magnified** at depth, to become apparent when the diver is **subjected** to cardiopulmonary stress. Just how those stresses could **elicit** the observed symptoms, however, is not known.

## REFERENCES

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