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_2), the other at 305 msw while the diver was breathing 0.4 ATA O_2 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 $l \cdot s^{-1}$) and minute ventilation (RMV) was only 30 $l \cdot min^{-1}$, but end-tidal CO_2 did not exceed 59 mm Hg. Before the event, peak-to-peak mouth pressure (DP) averaged 21 cm H_2O.

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 $l \cdot min^{-1}$, and DP was 30 cm H_2O. 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 min^{-1}, in the second it was 163 min^{-1}. Headaches were absent. Peak-to-peak mouth pressures never exceeded 30 cm H_2O, 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, 0.54 and 0.17.

DISCUSSION

There is no obvious explanation for these incidents. A common cause of unconsciousness in divers, CO_2 narcosis secondary to hypoventilation, was unlikely because end-tidal CO_2 was normal for exercising divers, and the characteristic 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 due to the cause of these unusual events. We have postulated that the hyperbaric environment accentuates physiological differences *among* divers. Theoretically, minor functional defiats 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.

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