Fatal Respiratory Failure During a "Technical" Rebreather Dive at Extreme Pressure

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A diving fatality at the extreme depth of 264 m fresh water is described. The diver was equipped with an underwater video camera which recorded events leading to his death. These events corroborated predictions about respiratory complications at extreme pressure made by early researchers. Review of the video and relevant literature resulted in the following physiological interpretation: an increase in respired gas density during descent caused a progressive increase in resistance to flow in both the airways and the breathing circuit. Initially, this was associated with a shift to ventilation at higher lung volumes, a relative degree of hypoventilation, and mild permissive hypercapnia. The promotion of turbulent airway flow by increasing gas density resulted in effort-independent expiratory flow at lower flow rates than usual. The consequent inability to match ventilation to the demands of physical work at the bottom precipitated a spiraling crisis of dyspnea, increasing P_{aCO_2}, and wasted respiratory effort, thus producing more CO_2. Extreme hypercapnia eventually led to unconsciousness. This tragic case provides a timely and salient lesson to a growing population of deep "technical" divers that there are physiological limitations that must be understood and considered when planning extreme dives.

Keywords: technical diving, diving physiology, carbon dioxide, ventilation, breathing.

STANDARD TEXTS ON respiratory physiology frequently refer to the effects of "extreme" environments on respiratory function (17). One such environment is the underwater realm. Diving physiology research enjoyed much attention between the 1950s and 1980s, driven largely by military and commercial interests. Recently, work at extremes of depth is frequently left to unmanned remote operated vehicles that are safer and cheaper to run than deep diving teams. The decline in commercial incentive to send man to great depths resulted in a lull in research addressing these challenges.

However, since the late 1980s, an increasing number of so-called recreational "technical divers" have begun using helium-based gas mixtures, complex underwater breathing apparatus including rebreather devices, and advanced (but largely experimental) decompression techniques in order to visit depths approaching 300 m sea water (msw)/31 atmospheres absolute (ATA). These individuals are engaging in a level of diving previously known only to military or commercial divers who had the benefit of teams of attending technicians, physicians, and physiologists. Both the divers and their instructors often have little more than a superficial familiarity with the complex physiological changes that occur at these extreme depths.

In this article we present a technical diving fatality at a depth of 264 m fresh water (mfw). The accident was unique in that the diver was equipped with an underwater video camera and actually recorded the events leading to his death. These events are strongly suggestive of critical constraints on respiratory performance at extreme depths and appear to corroborate the results of previous physiological experiments at high pressure.

CASE REPORT

A 51-yr-old fit male diver undertook a 264-m dive in a vertical fresh water cave. During a previous dive 4 mo earlier he found the mortal remains of a diver who had gone missing 10 yr before. He had marked the site and planned to perform a second dive to retrieve the body. The plan was to descend over 12 min, spend 5 min collecting the diver's remains with a body bag, then make a stepwise 9-h decompression. The body would be handed over to a series of support divers who would take it to the surface.

The dive was planned around the use of a Biomarine Mark 15.5 closed circuit mixed gas rebreather developed for the U.S. Navy in the early 1980s. These devices recirculate exhaled gas around a breathing "loop" comprised of breathing hoses, one-way valves, a CO_2 scrubber containing soda lime, and a counter-lung. A pre-set partial pressure of oxygen (P_{O_2}) is maintained in the loop throughout the dive by an oxygen addition and dilution system. The P_{O_2} in the loop is constantly monitored by galvanic fuel cells that activate a solenoid valve to add oxygen when the P_{O_2} falls below the "set

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point." Conversely, the oxygen is diluted by means of a "diluent" gas made up of helium (for its low density and non-narcotic properties) and nitrogen. Nitrogen is included because it ameliorates the effects of the high-pressure neurological syndrome, but the fraction present is limited to prevent excessive nitrogen narcosis. For this particular dive the diluent was 4% oxygen, 82% helium, 14% nitrogen, and the P_{O_2} set point was 1.3 ATA (988 mmHg). Allowing for this P_{O_2} and the proportions of diluent gas constituents, the respired gas at 264 mfw was expected to be very similar to the diluent gas itself. This dive together with the previous one represented the deepest dives ever performed using a rebreather from surface to surface (as opposed to deployment out of some form of underwater "bell" or habitat).

A series of support divers followed the lead diver in a staged relay to ensure continuous transport of the corpse back to the surface. However, when the first support diver arrived at 200 m he saw that the lead diver's lights were immobile 60 m below him. He attempted to reach the lead diver but suffered an equipment failure, forcing him to return to the surface. In the process he suffered severe decompression sickness.

Three days later the lead diver was found to have floated to a shallow portion of the cave with the corpse attached to him by a tangled line. The video camera worn on his helmet was still intact and analysis of the audiovisual recording revealed the following events. The descent followed the main "down-line" from the surface to the cave floor and lasted 12 min as planned. There was little change in the audible pattern of respiration over the 12-min period of descent. The corpse was located by following a short line previously laid across the cave floor. The diver then opened a body bag and attempted to manipulate it around the corpse. The task was more difficult than anticipated as the corpse was saponified, bulky, and still attached to diving equipment. This short period of exertion caused an audible, progressive increase in respiratory rate. On reaching the allocated 5 min without adequately securing the corpse, he abandoned the task and attempted to leave the site, only to discover that his torch had become entangled in the marker line. Efforts to free himself caused progressively worsening dyspnea with increasingly frequent "grunting" on exhalation. The diver initiated the first of several attempts to flush the rebreather loop with fresh diluent gas using a manual bypass valve 2 min after leaving the corpse. Audible venting of this gas through the counter-lung over-pressure relief valve indicated that the flush was successfully displacing gas from the loop, but there was no discernable relief from the dyspnea. The diver's exhalations developed a choking quality 3 min after leaving the corpse and approximately 1 min later, at 21 min total dive time, breathing and movement stopped. It is notable that throughout this sequence of events, the level of dyspnea exhibited by the diver seems markedly excessive in relation to the work undertaken, even after the entanglement when there was no violent struggling or obvious panic.

The rebreather was carefully examined. The unit appeared functional and there was no evidence of water ingress. However, there were several potentially important findings. First, in the standard Mk 15.5 configuration, a low-density foam moisture absorbing pad lies at the inlet and outlet of the CO_2 scrubber canister. These pads occupy space in the gas flow path, but are carefully cut to provide minimal obstruction to gas flow. In this case these pads were non-standard and made from felt rather than foam. The cut of the inlet pad was such that it occupied a much greater space in the gas flow path than the standard pad, and almost certainly imposed more resistance to flow. In addition, the outlet pad lay directly over the outlet ports of the scrubber canister instead of being held off these by a plastic grill usually used for this purpose. This may also have imposed more resistance to flow.

Second, although the grain size was not objectively determined, the scrubber canister was packed with fine grade soda lime. Moreover, this material was not packed in the standard manner described in the relevant Navy Manuals. Instead of being filled to the rim of the canister and then subjected to a compressive force by a foam compression pad and the lid, the material was filled to approximately 1 cm below the canister rim, and the compression pad was on top, acting as a space occupier but exerting little or no compressive force.

DISCUSSION

The most likely cause of death was acute respiratory failure and CO_2 toxicity. This hypothesis is based on the following considerations which will be expanded on later: 1) the progressive dyspnea culminating in unconsciousness over a period of time is consistent with the diagnosis of CO_2 toxicity; 2) CO_2 accumulation is plausible in the context of the dive; 3) the aberrations in the rebreather assembly would predispose to CO_2 accumulation; 4) the diver's efforts to flush the rebreather counter-lung with fresh diluent gas indicate that he suspected hypercapnia to be the cause of his dyspnea; and 5) there is no evidence to implicate any other terminal event. In particular, there is circumstantial evidence that the P_{O_2} in the rebreather loop was appropriate at the time of death. The audio tape clearly records the oxygen solenoid valve "firing" in the typical intermittent pattern of correct operation. This indicates that the rebreather was actively maintaining the programmed P_{O_2} set point. The active flushing with diluent, if anything, would also have preserved the oxygen set point of 1.3 ATA (approximately 988 mmHg). Therefore, it is apparent from consideration of the alveolar gas equation that hypoxia would be unlikely prior to the onset of CO_2-induced coma. Finally, there were no seizure-like movements suggesting hyperoxia at the point of loss of consciousness. It is noteworthy that the same assembly had been used by the same diver on an almost identical dive 4 mo earlier without significant difficulty. The most obvious difference between the dives was the small increase in physical exertion during the second dive while attempting to

retrieve the corpse and trying to overcome the accidental entanglement.

In the absence of CO_2 rebreathing (which is discussed later), alveolar and, therefore, arterial CO_2 tensions are determined by a balance between alveolar ventilation and metabolic production of CO_2 as described by the simple formula:

$$P_ACO_2 = \dot{V}CO_2/\dot{V}_A \times K$$

Where: P_{ACO_2} = the alveolar pressure of CO_2, $\dot{V}_A$ = alveolar ventilation, and K is a constant (23). Thus a rise in P_{ACO_2} would be the result of either an increase in metabolic production of CO_2 and/or a decrease in alveolar ventilation. Accordingly, we must consider the effects of elevated pressure on alveolar ventilation and the consequent effects on CO_2.

For the purposes of this case, the direct or indirect effects of pressure on effective alveolar ventilation may have resulted from: 1) increased resistance to flow in airways and equipment; 2) ventilation/perfusion mismatching; or 3) perturbation of respiratory control. These are considered below. As a prelude to this discussion, it should be noted that most of the experimental studies cited used air as the respired gas. To facilitate comparisons, at 264 mfw (26.5 ATA), the helium-based mix used on the dive would result in a gas density of 10.2 $g \cdot L^{-1}$. This is approximately eight times the density of air at 1 ATA and, therefore, equivalent to air respired at 70 msw (8 ATA).

Increased Resistance to Flow in Airways and Equipment

Under conditions of increased environmental pressure, the corresponding increase in inspired gas pressure inevitably leads to an increase in respired gas density and greater airways resistance. Both maximum voluntary ventilation (MVV) and maximum exercise ventilation (MEV) are progressively reduced as pressure and gas density increase (7,13,15). In fact, MVV decreases approximately as a square root function of gas density (15). The mechanisms of this progressive limitation of the respiratory capacity are best appreciated by considering exhalation and inhalation separately.

With regard to exhalation, a Starling resistor mechanism relates the lung's static recoil pressure to maximum expiratory flow or so-called "effort-independent flow" (14). A diver at high pressures is more likely to experience effort-independent flow during exhalation (24). This is explained by an earlier onset of turbulent flow at high gas densities and the related observation that airway pressure falls more quickly where turbulent flow prevails. Thus, with increasing gas density, the point at which airway pressure equals intrathoracic pressure (the "equal pressure point") will be reached more quickly and effort independent flow will supervene. Furthermore, the actual flow rates at the point of effort-independence will be reduced significantly. For instance, during air breathing at pressures between 7.8 and 10 ATA (which represent gas densities very similar to those in the case presented), effort-independent flow rates at 60% of forced vital capacity drop by 50 to 75% of those measured in air at 1 ATA (15,24). It follows that the capacity for increasing expiratory flow rates is markedly reduced when breathing dense gas, consistent with the observations of reduced MVV and MEV at depth.

There are two further relevant observations concerning effort-independent expiratory flow. Firstly, any attempt to increase expiratory flow at the point where it has become effort-independent is wasted work. Not only does this fail in its objective but it contributes further to the accumulation of CO_2 (24). Secondly, at increased gas density the disadvantageous effect of decreasing lung volume (and narrowing of airways) on expiratory flow is amplified. At higher lung volumes higher flows are facilitated by radial traction on the airways. This may allow development of greater transpulmonary pressure before effort-independent flow ensues. Therefore, not surprisingly, divers encountering effort-independent flow will subconsciously increase their expiratory reserve volume and breathe at higher lung volumes (7,15). This has implications for the mechanics of inspiration (see below) and comes at a higher metabolic cost.

With regard to inspiration, there is no Starling resistor effect under normal circumstances. In fact, the physical distension of intrapulmonary airways during inspiration facilitates flow. Nevertheless, isovolumetric pressure-flow curves were constructed for inspiration of gas at densities between 1.29 and 10.1 $g \cdot L^{-1}$, and showed that at equivalent lung volumes and levels of inspiratory effort, flow was markedly reduced as gas density increased (15). It is notable that at the highest density, which almost exactly matches the dive reported here, a degree of effort-independence was observed in curves from the lower range of lung volumes (20 to 40% of FVC) once inspiratory transpulmonary pressures exceeded -20 to -40 mmHg. The author attributed this to the larynx acting as a fixed dimensional flow restrictor, possibly with some additional contribution from in-drawing of the "soft deformable" posterior wall of the extrathoracic trachea just below the level of the cricoid cartilage. A significant reduction in inspiratory flows during maximal exercise at a respired gas density of 7.74 $g \cdot L^{-1}$ was also noted by Hesser et al. (7) who attributed it to a reduction in the inspiratory driving pressure generated when breathing at higher lung volumes. Others have observed that the inspiratory muscles are disadvantaged in this setting (13). Indeed, it has been suggested that inspiratory muscle function may be the ultimate limiting factor on ventilation at depth (2).

In addition to the effects of increased gas density on airway resistance, the impact of additional external resistance must be considered. The use of an underwater breathing apparatus (UBA) almost invariably adds breathing resistance and this can make a critical difference to respiratory and work performance. Based on a database of experimental trials it was predicted that despite the use of breathing apparatus compliant with the U.S. Navy design specification for external resistance, 5 to 25% of divers would fail under conditions of

moderate work (150 W) and elevated gas density (6.3 g $\cdot L^{-1}$) (4). Dive "failure" was defined as the inability to complete 6 min of exercise, experiencing abnormal respiratory discomfort, or an end tidal of $CO_2 > 70$ mmHg. This has relevance to our case as the rebreather was built for the U.S. Navy under this specification, but was operated at a respired gas density 1.65 times greater than the dives on which the above calculations were based (4). Moreover, the use of fine grade soda lime in the scrubber canister, and the various modifications or configuration errors detailed in the case report could only have increased the risk of failure to meet the accepted standard for external resistance.

Consideration of equipment-related breathing resistance also highlights an important difference between a rebreather-type UBA and the more conventional "open-circuit" equipment frequently referred to as "scuba" (the acronym for self-contained underwater breathing apparatus). In a rebreather, exhaled gas flows through a system of one-way valves, hoses, a counter-lung, and a CO_2 scrubber. This flow is generated entirely by the inspiratory and expiratory effort of the diver. In open circuit scuba, gas is exhaled directly into the environment through a single one-way valve with minimal resistance. Gas is inhaled from a negative pressure-activated demand valve that opens a pressurized gas supply. If the demand valve is well designed and tuned, the inhalation resistance can be very low.

Unfortunately, on open circuit, all exhaled gas is lost and at extremes of depth the vast quantities required are difficult to transport. Rebreathers are attractive because only the oxygen consumed by the metabolism needs to be replaced. It follows that they offer much in breathing gas conservation, but at the cost of greater technical intricacy and additional breathing resistance. This case suggests that at least some may be less well-suited to the physiological demands of exertion at extreme depths.

Ventilation/Perfusion Mismatch

There is a possibility that high pressure may reduce effective alveolar ventilation by increasing physiological dead space. The V_D/V_T ratio was measured in a dry chamber pressurized to 47 and 66 ATA during rest and exercise while breathing various gas mixes (20). At rest, the ratio was higher under pressure (42% vs. 35% at 1 ATA). More importantly, it improved little during exercise compared with 1 ATA where it fell to 20%. The significance of this finding to the immersed diver is not known.

Perturbation of Respiratory Control

There is evidence that respiratory reactivity to CO_2 is reduced in the presence of increased resistance to breathing (1,3,5,25). Inspiratory resistive loading has been shown to depress the slope of the $\dot{V}_E/\dot{V}_{CO_2}$ curve (18). This was attributed to a propensity for the respiratory controller to balance the chemical drive to breathe against a reduction of respiratory effort in such a way that it would tolerate higher CO_2 levels in favor of avoiding an increase in respiratory effort. Although this study used graded flow restrictors to increase breathing resistance, similar phenomena have been observed using increasing gas density. Individuals exercising sub-maximally at pressures from 1 to 4 ATA showed that as inspired air density increased, the response to P_{aCO_2} (estimated from end tidal measurements) was suppressed so that work of breathing increased only minimally at the cost of a rise in CO_2 (8). In further support of these findings, an investigation of respiratory responses to P_{aCO_2} (estimated from end tidal measurements) over a wide range of inspired gas densities (0.4–22 g $\cdot L^{-1}$) concluded that there was gross diminution of ventilatory responses to CO_2 with increasing inspired gas density (6).

Other related work concluded that pressure per se appeared to promote hypercapnia, independently of any effect on gas density, over a range between 1 and 8.5 ATA (19). The authors were unable to provide an explanation for this. It has also been suggested that the narcotic or anesthetic properties of nitrogen under pressure might contribute to alteration of respiratory control (11). However, investigation by separate groups concluded that nitrogen did not exert an important effect, whereas gas density did (6,11). Finally, though the mechanism is unclear, it has been reported that divers may develop reduced ventilatory responses to CO_2 even when not immersed (8–10).

For completeness, it must be noted that not all studies have shown perturbation of respiratory control under relevant conditions, particularly in individuals at rest or under conditions of light work (20). However, the combination of variable methodology, small numbers of subjects, and the proven presence of significant inter-subject variability makes it difficult to draw either comparisons between studies or universally applicable conclusions about respiratory control in divers under relevant conditions (15,22).

A final possible contributing factor to the fatality is the possibility that CO_2 was incompletely removed from the exhaled gas; that is, it bypassed the CO_2 scrubbing system and entered the counter-lung to be rebreathed. This would seem unlikely under normal circumstances. The Mark 15.5 rebreather is popular among extreme deep divers because it has a large well-insulated scrubber canister that anecdotally performs well at extreme depths and cold temperatures. However, in this particular case the scrubber material, while fresh, was incorrectly packed. Accordingly, subtle shifts may have caused uneven gas flow or "channeling" along paths of uneven resistance. In addition, it is believed that scrubbers are less efficient at greater gas density because CO_2 molecules must compete with much higher concentrations of other gas species for contact area on the absorbent granules (12).

It will never be known whether CO_2 breakthrough and rebreathing contributed to this accident. However, there are potentially disastrous consequences when this occurs. Work producing a $\dot{V}_{CO_2}$ of 1.5 L $\cdot$ min^{-1} (STPD) requires alveolar ventilation ($\dot{V}_A$) of 32.4 L $\cdot$ min^{-1} (BTPS) to maintain a P_{aCO_2} of 40 mmHg when the

inspired gas is free of CO_2 (2). If the inspired P_{CO_2} is 10 mmHg, then the $\dot{V}_A$ required to maintain P_{aCO_2} = 40 mmHg increases by 33% to 43.2 L min^{-1}. In an environment where $\dot{V}_A$ is already maximal, catastrophic CO_2 accumulation will result. For the case presented, the authors favor respiratory failure over CO_2 rebreathing as the primary cause for the following reason: if CO_2 contamination of the counter-lung and inspiratory limb of the rebreather loop had been the primary problem, the diluent flush maneuver by the diver would have been an appropriate remedy—much like running an anesthetic circle circuit at high fresh gas flow rates. However, it did not appear to offer relief. Nevertheless, it remains possible that both respiratory failure (hypoventilation) and CO_2 rebreathing were involved.

In summary, multiple predispositions to hypercapnia during compressed gas diving have been identified. Most notably, as gas density increases there is a progressive mechanical limitation on ventilation, potentially augmented by resistance imposed by the UBA. There may also be altered control of ventilation that allows P_{aCO_2} to rise, especially if increased respiratory work would otherwise be required to maintain normocapnia. Finally, in a rebreather-type UBA it is possible that CO_2 may bypass the scrubber and be rebreathed. The recorded events of this case are potentially consistent with all of these processes and with many of the specific experimental findings cited above. Accordingly, we propose the following hypothetical physiological interpretation.

The increase in respired gas density during descent caused a progressive increase in resistance to flow in both the airways and the rebreather circuit. This was associated with a relative degree of hypoventilation (caused initially by reduced ventilatory response to CO_2), a shift to ventilation at higher lung volumes, and a modest permissive hypercapnia. The respiratory demands of work at depth then resulted in expiratory flows that met the threshold for effort-independence, with the progressive tachypnea suggesting that ventilation became inadequate and that P_{aCO_2} was increasing. The stress and modest exertion associated with entanglement at the point of leaving the site precipitated a spiraling crisis of increased respiratory demand which could not be met because of flow limitation. Futile attempts to do so only resulted in a vicious cycle of wasted work and accumulation of more CO_2 (24). Once established, this cycle would have been hard to break unless resistance to breathing could be rapidly reduced. One option available to the diver would have been to "bail-out" onto open circuit scuba equipment. However, this would have required removing the rebreather mouthpiece and replacing it with the scuba regulator; an intervention that dyspneic divers in crisis situations are reluctant to perform (21).

It is notable that during the last 2 min there was a "choking" quality to the exhalations, precisely as described by Wood and Bryan when a 200-W work load was attempted at 10 ATA breathing air (density 12.93 g $\cdot L^{-1}$) (24). In the short pre-terminal period there were intensifying but futile attempts to increase ventilation in the face of a steadily increasing P_{aCO_2}. Ultimately there was exhaustion of the respiratory muscles, rapidly rising CO_2, and unconsciousness due to CO_2 narcosis. An additive contribution from nitrogen narcosis as proposed by Morrison et al. (16) cannot be ruled out. The other significant unknowns remain the extent to which the configuration of the rebreather contributed to an increase in work of breathing and whether CO_2 "breakthrough" occurred across the scrubber canister to cause CO_2 rebreathing. While we acknowledge that this analysis is speculative, the events are consistent with previous experimental findings for which they offer a poignant corroboration. This case provides a tragic but timely and salient lesson to a growing population of deep "technical" divers that there are physiological limitations that must be understood and considered in planning extreme dives.

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