PHYSIOLOGIC BASIS FOR CO\(_2\) LIMITS WITHIN SEMICLOSED- AND CLOSED-CIRCUIT UNDERWATER BREATHING APPARATUS
Semiclosed- and closed-circuit underwater breathing apparatus (UBA) incorporate a canister filled with carbon dioxide (CO₂) absorbent material. As the absorbent becomes expended, the CO₂ level within the breathing loop will rise. Currently, CO₂ absorbent canisters are considered expended when the effluent CO₂ is 0.5% Surface Equivalent Value (SEV). In the past, this value often represented the threshold between the linear and the exponential rise in effluent CO₂. The 0.5% SEV limit was used to reduce the diver’s risk for experiencing excessive levels of CO₂. However, with advances in absorbent canister design, this value may be overly conservative. Because the overall respiratory load of a UBA can compound the effect of the inspired CO₂, the improved breathing performance of some current UBAs also warrants reevaluating the current limit. This literature review specifically looked at low levels of CO₂ affecting work and mental performance, nitrogen narcosis, decompression sickness and central nervous system (CNS) oxygen toxicity to determine if the breakthrough limit could be changed.
19. Abstract continued:

RESULTS. A reduction in the maximal work capacity was observed when the inspired PCO₂ was 2% and greater. No deterioration in psychomotor and mental performance was detected for inhalation of up to 5% CO₂ during exercise. However, alveolar CO₂ tensions in excess of 40 mmHg (test conditions used an inspired CO₂ of 2%) may potentiate the effect of nitrogen narcosis. This factor is an important consideration for the full operational usage of the MK 16 with the 0.7 ATA of oxygen in nitrogen. Breathing CO₂ greater than 1% for over 1 hour has been associated with an increased the risk for decompression sickness. Two-percent CO₂ may increase the onset of an oxygen convulsion when combined with elevated percentages of oxygen because of the increase in brain capillary blood flow.

CONCLUSION: The literature suggests that a short duration exposure, 15 minutes, to a CO₂ level of 2% SEV will not result cause a catastrophic effect on a diver. The literature suggests that a CO₂ level of 2% produces a minimal effect on a diver's physical and mental work performance. At most, there may be a marginal increase in the risk of decompression sickness. The risk of CNS oxygen toxicity does not appear to be increased for U.S. Navy oxygen diving operations. When designing CO₂ scrubber canister limits based upon the above described physiological response, the UBA's respiratory load and canister carbon dioxide absorption characteristics, oxygen or breathing gas supply duration also must be considered.
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PHYSIOLOGIC BASIS FOR CO₂ LIMITS WITHIN SEMICLOSED- AND CLOSED-CIRCUIT UNDERWATER BREATHING APPARATUS

INTRODUCTION

Semiclosed- and closed-circuit underwater breathing apparatuses (UBAs) incorporate a canister filled with carbon dioxide (CO₂) absorbent material. This design efficiently uses the gas supply because a portion of the diver's exhaled breath is recirculated through the system. Additional gas is added to the UBA to replace the oxygen consumed by the diver. As the absorbent becomes expended, the CO₂ level within the breathing loop will rise. Because CO₂ does have a physiologic effect that may affect a diver's performance, the question becomes 'how much CO₂ is too much.'

Historically, canister limits were defined as the time it takes in minutes or the liters of CO₂ absorbed until the canister effluent gas CO₂ reaches 0.5% Surface Equivalent Value (SEV). During the current evaluations of the MK 25, a closed-circuit UBA, the SPECWARCOM Biomedical Research and Development Medical Officer raised the issue that this value for the partial pressure of CO₂ (PCO₂) limit may be unduly conservative. The purpose of this paper is to review the literature and recommend a canister limit based upon the known data of the physiologic effects of low levels (<4%) of CO₂.

METHODS

The primary concerns of low levels of CO₂ are their possible effects on a diver's mental and physical performance. However, this discussion must also address a UBA's breathing resistance, since hypoventilation will also increase the diver's blood CO₂ level and potentiate any CO₂ problem. Furthermore, high levels of CO₂ in the blood will change the diver's blood chemistry affecting work performance. Carbon dioxide also affects cerebral blood flow that may influence the diver's threshold to Central Nervous System (CNS) oxygen toxicity. In addition, the role of CO₂ on decompression sickness must also be addressed. This literature review primarily concentrated on the articles frequently quoted when discussing the effects of CO₂. It must be realized that the studies involving CO₂ levels below 3% are sparse. Typically, studies used at least 5% CO₂ to ensure a physiologic effect could be observed.

DISCUSSION

INTERRELATIONSHIP OF RESISTIVE LOADS AND CO₂

To understand the physiologic effects of inspired CO₂, its relationship to breathing with an underwater breathing apparatus must be understood. The following mathematical formula shows the relationship between the factors affecting the alveolar
CO₂ levels. In normal individuals, the alveolar PCO₂ (PₐCO₂) is equal to the arterial PCO₂ (PₐCO₂). It's the blood CO₂ that causes the physiologic effects.

\[ P_{ACO₂} = P_{ICO₂} + 863 \frac{V_{CO₂}}{V_E} (1-V_D/V_T) \]  
Equation (1)

Where:
- \( P_{ACO₂} \): alveolar PCO₂ (mmHg)
- \( P_{ICO₂} \): inspired PCO₂ (mmHg)
- 863: factor for correcting \( V \) from STPD to BTPS
- \( V_{CO₂} \): minute volume of CO₂ (liters per minute; STPD)
- \( V_E \): minute ventilation (liters per minute)
- \( V_D \): dead space volume (liters)
- \( V_T \): tidal volume (liters)

Any carbon dioxide inspired will directly affect the blood CO₂ levels. In addition, any impediment to ventilation will cause the PₐCO₂ to rise. For example:

\[ \downarrow V_E \text{ when:} \uparrow \text{gas density} \]
\[ \uparrow \text{rig resistance} \]
\[ \uparrow \text{inspired oxygen during exercise} \]

Ventilation increases with increasing exercise. When the inspired fraction of CO₂ rises, there is a concomitant rise in ventilation. Figure 1 illustrates the rise in minute ventilation with increasing work rates. When CO₂ is added to the inspired gas, the ventilation rate further elevates above the level expected from exercise alone.

Figure 1. Relationship of ventilation to \( O₂ \) uptake during exercise at different levels of inspired \( CO₂ \) tension. The average values of oxygen uptake (\( \dot{V}_{O₂} \)) at rest and at 4 different work loads were not significantly altered by changes in PₐCO₂.

Clark et al., further demonstrated that the level of inspired CO$_2$ affects the arterial CO$_2$. For exercise performed on the surface breathing air, a person actually hyperventilates during exercise performed at 50% of maximal oxygen uptake ($\dot{V}_{O_2\text{max}}$) and greater. Figure 2 illustrates that when the $P_{i\text{CO}_2}$ is increased above 20 mmHg, the $P_a\text{CO}_2$ dramatically rises for the level of exercise.

Figure 2. Arterial $P_{\text{CO}_2}$ during exposure to combined exercise and hypercapnia.


Experiments performed by Poon illustrate the ventilatory responses to hypercapnea and exercise when breathing through an inspiratory resistive load (~12 cmH$_2$O/l/s). The investigator kept the end-tidal $P_{\text{CO}_2}$ ($P_{ET\text{CO}_2}$) constant, while the subject inspired a constant fraction of ~5% CO$_2$.

Figure 3 shows that an inspiratory resistive load further reduces minute ventilation when the $P_{ET\text{CO}_2}$ exceeds 40 mmHg. Therefore, breathing patterns are a result of a balance of chemical drive and the propensity to reduce respiratory effort.

Figure 3. Responses in minute ventilation to $P_{ET\text{CO}_2}$ under no load (filled circles, solid line) and inspiratory resistive load ~12 cmH$_2$O/l/sec (open circles and dashed lines).

Application of an external resistance will also affect a person's \( P_{ET}CO_2 \). Demedts and Anthonisen demonstrated that as breathing resistance approaches 15 cmH\(_2\)O/l/sec, there's a distinct increase in \( P_{ET}CO_2 \), which increases with exercise load; whereas, at resistances less than 5 cmH\(_2\)O/l/sec there's no change in the \( P_{ET}CO_2 \) for the increasing work load\(^5\). Figure 4 illustrates the effect of resistance on \( P_{ET}CO_2 \).

In general, when a person doesn't breathe adequately, the \( P_aCO_2 \) will rise. This is the case in diving. Divers tend to have a markedly decreased breathing frequency and increased tidal volumes, which is not related to fitness, when compared to non-divers\(^6\). Also perhaps because of a diver's experience with the desire to conserve breathing gas, the diver's breathing pattern results in a relative hypoventilation, regardless of UBA type. In addition, during exercise, there is a pronounced hypoventilation and hypercapnia in divers\(^7\). Hence, with the addition of an UBA's breathing resistance, the diver typically will hypoventilate for the work being performed resulting in an increased \( P_{ET}CO_2 \).

**CO\(_2\) EFFECTS ON WORK PERFORMANCE**

Overall work performance may decrease with increased inspired \( CO_2 \). Work performed in the 1970's for the National Aeronautics and Space Administration established that maximal work capacity is reduced when the \( P_iCO_2 \) is 15mmHg (2%)\(^8,9\). The authors attribute the deterioration in work performance to the interference to eliminating the excess \( CO_2 \) from exercising muscles and the resulting acidemia. No decrements were reported for \( P_iCO_2 \) less than 2%.
Craig et al., conducted studies where exhausting exercise was performed while inhaling against a resistance with a gas mix containing 3 to 4% CO₂. Figure 5 illustrates that when CO₂ is added to the inspiratory gas with a resistance of 1.5 cm H₂O/l/sec (R1), ventilation increases and the person was able to walk for 10 minutes before becoming exhausted. However, when breathing against a resistance of 15.5 cm H₂O/l/sec (R4) combined with 4.3% PₐCO₂, the person did not produce the appropriate ventilatory response and the time to exhaustion was much shorter. Hence, if the breathing resistance is kept low, there's an adequate response to the inspired CO₂ and work performance is not impacted.

Figure 5. Respiratory minute volume during walks under varied conditions of inspiratory resistance and inhalation of carbon dioxide. Arrow marks beginning of walking. Concentrations of carbon dioxide in inspired air were 3.9% at R1 and 4.3% at R4.


**CO₂ EFFECTS ON COGNITIVE PERFORMANCE**

The increase in a person's PₐCO₂ is referred to as CO₂ retention. Therefore, the effect of an increased PₐCO₂ must be evaluated in light of an increased PₐCO₂. Henning et al., evaluated the behavioral effects of an elevated PₐCO₂ to clarify the risks due to CO₂ retention. For this study, the subjects breathed 6% CO₂ in 21% and 94% oxygen and performed various psychometric tests. The authors concluded that divers may be at risk for disequilibria, impaired decision making and disturbances in motor control immediately following a period of CO₂ retention. Furthermore, if a diver hypoventilates and if a high breathing resistance is suddenly added, the result can be sudden unconsciousness. Hence, to minimize the potential of hypoventilation and its potentially catastrophic consequences, the breathing resistance within semiclosed- and closed-circuit UBAs should be as low as technically possible.

The breathing resistance of a closed-circuit UBA can increase as the CO₂ absorbent material is depleted. Divers reported that during underwater exercise at 60 feet they had to use the diluent by-pass of the MK 15 to increase gas flow to reduce breathing
effort when the canister effluent CO\textsubscript{2} exceeded 3.8 mmHg (0.5% SEV). It should be noted that the divers in that study did not report any other symptoms even though the canister effluent CO\textsubscript{2} reached 1.5% SEV. However, theoretically, divers who are breathing at a low ventilation rate because of their breathing apparatus, combined with an increase in inspired CO\textsubscript{2} may run the risk of impaired performance if the resistive load progressively increases. Unfortunately, no controlled studies were performed at NEDU that determined the actual increase in breathing resistance with an increasing canister effluent CO\textsubscript{2} and its affect on ventilation. However, all the studies reviewed only found symptoms when the diver was exposed to very high resistances or a P\textsubscript{1}CO\textsubscript{2} of 5% or greater.

A diver's cognitive performance is critical in an underwater environment. Errors in judgement can be catastrophic. Sheehy et al., evaluated the effect of 4% and 5% CO\textsubscript{2} in 21% and 50% oxygen on cognitive performance during exercise and recovery on the surface. They reported no deterioration in psychomotor and mental performance for the inhalation of up to 5% CO\textsubscript{2}. The investigators noted that the short-term memory test they used detected effects due to strenuous exercise, whereas low levels of CO\textsubscript{2} did not cause a decrement in memory performance. Recalling Equation (1), if the P\textsubscript{a}CO\textsubscript{2} is kept below 5% by appropriate ventilation for the level of P\textsubscript{1}CO\textsubscript{2}, the diver’s cognitive performance would not be limited.

However, the MK 16 with a PO\textsubscript{2} set point of 0.7 ATA in nitrogen does have a significant level of inert gas in its breathing loop at its deep operating depth. In fact, the MK 16 limits its operational depth to 150 fsw because of the significant nitrogen narcosis that a diver experiences, especially under working conditions. Hesser et al., described a relationship between CO\textsubscript{2} and nitrogen narcosis. This study used CO\textsubscript{2} concentrations of 0, 2, 4 and 6% and was performed at the surface and at 6 ATA. The results suggested that when the inspired gas tension of nitrogen (P\textsubscript{1}N\textsubscript{2}) and the inspired CO\textsubscript{2} (P\textsubscript{1}CO\textsubscript{2}) rose simultaneously, their effect on performance was greater than the arithmetic sum of the changes induced by either gas alone. Allowing the P\textsubscript{1}CO\textsubscript{2} to increase to 2% may potentiate the risk of nitrogen narcosis.

CO\textsubscript{2} EFFECTS ON CENTRAL NERVOUS SYSTEM O\textsubscript{2} TOXICITY

An increase in the P\textsubscript{1}CO\textsubscript{2} may reduce the time for the development of Central Nervous System (CNS) oxygen toxicity symptoms. The mechanism for this belief is the fact that CO\textsubscript{2} will cause a cerebral vasodilatation resulting in an increase in brain oxygenation. Figure 6 illustrates the exponential rise in cerebral blood flow when the arterial P\textsubscript{a}CO\textsubscript{2} rises.

As discussed earlier, P\textsubscript{a}CO\textsubscript{2} will increase with exercise when breathing against a resistive load. This pattern also is seen when exercising while breathing 100% oxygen at 2 ATA, though the absolute rise for each individual may be different. Figure 7 shows 6 subjects’ P\textsubscript{a}CO\textsubscript{2} at rest at 2 ATA while breathing 10.5% oxygen in nitrogen, and then during an incremental exercise while breathing 100% oxygen. Prior to starting the exercise, the P\textsubscript{a}CO\textsubscript{2} decreased during the transition from normoxia to oxygen breathing.
with an associated increase in ventilation. Though a rise in $P_aCO_2$ increased for all the subjects, the change in the $P_aCO_2$ at the highest workload ranged from 4.4 to 14.2 mmHg in the individual subjects.

**Figure 6.** The relationship between cerebral blood flow and arterial CO$_2$ tension. The arterial PCO$_2$ varied from the normal (dots) by hyperventilation (triangles) or by inhalation of 5-7% CO$_2$ (open circles). The dashed lines bound 98% of the observations while the central polygon encloses 94% of the normal values.


**Figure 7.** Individual arterial PCO$_2$ responses to incremental exercise while breathing O$_2$ at 2 ATA. Data are shown for 6 subjects who completed all 4 workloads.

The concern is that with an increased $P_aCO_2$ there is a concomitant increase in cerebral blood flow. With the increase in blood flow, more oxygen can be delivered to the brain, which may result in higher tissue oxygen and a higher direct toxic effect on the neuron, thereby accelerating the onset of an oxygen convulsion. A series of experiments was performed at the Institute for Environmental Medicine at the University of Pennsylvania to evaluate the effect of increased oxygen pressures and carbon dioxide on cerebral blood flow. These studies simulated a variety of conditions: 1) $PO_2$ of 1, 3 and 3.5 ATA while at rest; 2) $PO_2$ of 2 ATA while exercising; 3) $PO_2$ of 3.5 ATA with 2% $PCO_2$ while at rest. Lambertson reported that only under the last condition, did the brain oxygenation drastically increase from 100 mmHg to 1000 mmHg. Figure 8 illustrates the effect of increased oxygen levels at rest and at exercise, as well as the added effect that high inspired CO$_2$ partial pressures have on cerebral blood flow.

Figure 8. Effect of increased inspired $PO_2$ on the oxygen tensions of arterial, mean brain capillary, and internal jugular venous blood (average values in normal men). The graph illustrates for each of several levels of inspired $PO_2$ the manner in which oxygen tension across the mean brain capillary is increased by progressive increases in inspired $PO_2$. The patterns of change in brain capillary $PO_2$ are calculated from experimentally determined levels of oxygen pressure in arterial and brain venous blood on the assumption of uniform $O_2$ loss. When arterial hypercapnia was introduced by administering carbon dioxide with oxygen at 3.5 ATA, brain oxygenation was drastically increased.

Because the parameters of the University of Pennsylvania's studies are outside the allowed U.S. Navy limits for oxygen diving, interpolations were made. Assuming that the relationships between the vascular responses are similar for the various conditions as exemplified in Lambertson's report, a diver breathing a \( \text{PO}_2 \) of 2.5 ATA (100% oxygen at 50 fsw) and inspiring 2% \( \text{CO}_2 \), the oxygen tension in the brain's venous blood is approximately 400 mmHg. This is a substantial increase in cerebral blood flow and should increase the rate of development of CNS oxygen poisoning.

However, laboratory experiments do not necessarily reflect the conditions experienced operationally. The University of Pennsylvania's experiments bracketed the U.S. Navy Single-Depth Oxygen Exposure Limits, which restricts the exposure of 2.5 ATA \( \text{PO}_2 \) to 10 minutes. Typically, MK 25 operations are conducted in 20 feet of seawater and shallower. Under these conditions, the low brain venous blood oxygen tension, which reflects the low capillary blood oxygen tension, has a reduced risk of CNS oxygen toxicity. To summarize, if the \( P_r \text{CO}_2 \) is allowed to momentarily rise to 2%, surface equivalent value, a 15 minute exposure would not increase the risk of CNS oxygen toxicity when diving the U.S. Navy Single-Depth Oxygen Exposure Limits.

**\( \text{CO}_2 \) Effects on Decompression Sickness**

In 1908 Boycott, Damant and Haldane proposed that an elevated \( \text{CO}_2 \) would increase circulation to the muscles and increase the elimination of inert gas. This concept favors exercise during decompression. On the other hand, other reports suggest that the opposite is true\(^{19}\). Whether to allow exercise during decompression is still a rather controversial area without a definitive conclusion. Therefore, for the purpose of this discussion only the effect of an elevated inspired \( \text{CO}_2 \) will be considered. This condition closely reflects the operational experience.

One article reported an elevated rate of decompression sickness (DCS) in deep (3.2 ATA (72.6 FSW)) caisson operations in Japan\(^{20}\). Due to ventilation problems, \( \text{CO}_2 \) within the man-lock rose between 1.8 and 2.3%. In this case the incidence of DCS was 3.05%. When the lock was ventilated to reduce the \( \text{CO}_2 \) between 0.3 and 0.8%, the incidence of DCS was 0.96%. Unfortunately, the authors did not report which particular profiles resulted in DCS. Specifically they did not report the actual \( \text{CO}_2 \) exposure time for the decompression profile. Therefore, based on this article it's unknown if there's a dose-response relationship of \( \text{CO}_2 \) exposure to an increased incidence of DCS. The only conclusion is that an elevated \( \text{CO}_2 \) may result in an increased incidence of DCS.

**Conclusion**

The literature suggests that a short duration exposure, 15 minutes, to a \( \text{CO}_2 \) level of 2% SEV may not result immediately in a catastrophic effect on a diver. Though there is a distinct relationship of \( \text{CO}_2 \) to CNS oxygen toxicity with a sustained elevated inspired \( \text{CO}_2 \), a momentary exposure would not greatly increase the risk of an oxygen convolution.
Though the MK 16 diver using 0.7 PO$_2$ in nitrogen may experience narcosis normally during a deep excursion, the operational use of this UBA suggests that when a canister effluent reaches 2%, it would be during a shallow portion of the dive where the PN$_2$ is relatively low. Hence, it is unlikely that a MK 16 diver would experience a decrement in cognitive performance. There also is little evidence to substantiate the concern that a CO$_2$ level of 2% SEV greatly increases the risk of decompression sickness.

Our literature review indicates that a diver can tolerate a CO$_2$ level as high as 2.0% SEV for 15 minutes with minimal risk. However, the current 0.5% SEV limit for CO$_2$ inhalation was established to allow a margin of error in minimizing the potential that a diver would breathe potentially catastrophic levels (greater than 4% SEV) of CO$_2$. This was essential since unmanned testing does not simulate all the variability that exists during diving operations. Specifically, unmanned simulations define a specific CO$_2$ injection rate for a presumed oxygen consumption rate at a particular respiratory minute volume. Furthermore, unmanned testing did not define the canister performance characteristics beyond 1% SEV. Because of this limited testing, the rate of rise in the CO$_2$ levels can not be ascertained so a safety margin had to be postulated.

It is important to note that canister duration limits should not be based only on the average CO$_2$ breakthrough curve, which does vary with different UBA, but also on the oxygen or breathing gas supply duration as well as the physiological variations between divers and diving missions. It is possible that in the future once the UBA characteristics and diver variability can be adequately defined, or when a reliable underwater CO$_2$ monitor becomes available, we will recommend increasing the CO$_2$ level beyond the current 0.5% SEV.
REFERENCES


