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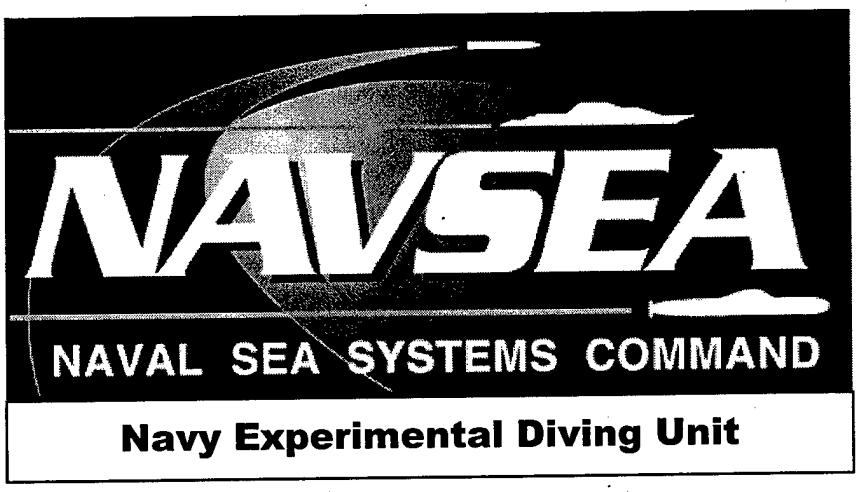


# PHYSIOLOGIC BASIS FOR $CO_2$ LIMITS WITHIN SEMICLOSED- AND CLOSED-CIRCUIT UNDERWATER BREATHING APPARATUS

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19. ABSTRACT:

Semiclosed- and closed-circuit underwater breathing apparatus (UBA) incorporate a canister filled with carbon dioxide ($CO_2$) absorbent material. As the absorbent becomes expended, the $CO_2$ level within the breathing loop will rise. Currently, $CO_2$ absorbent canisters are considered expended when the effluent $CO_2$ 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 $CO_2$. The 0.5% SEV limit was used to reduce the diver's risk for experiencing excessive levels of $CO_2$. 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 $CO_2$, the improved breathing performance of some current UBAs also warrants reevaluating the current limit. This literature review specifically looked at low levels of $CO_2$ 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.

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19. Abstract continued:

**RESULTS.** A reduction in the maximal work capacity was observed when the inspired $PCO_2$ was 2% and greater. No deterioration in psychomotor and mental performance was detected for inhalation of up to 5% $CO_2$ during exercise. However, alveolar $CO_2$ tensions in excess of 40 mmHg (test conditions used an inspired $CO_2$ 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 $CO_2$ greater than 1% for over 1 hour has been associated with an increased the risk for decompression sickness. Two-percent $CO_2$ 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 $CO_2$ level of 2% SEV will not result cause a catastrophic effect on a diver. The literature suggests that a $CO_2$ 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 $CO_2$ 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.

## ACKNOWLEDGEMENT

I thank Dr. Chris Lambertsen M.D., "Father of U.S. Combat Swimming," for his review of this paper's technical accuracy and the appropriateness of the derived conclusions based upon the presented literature and his experience.

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# PHYSIOLOGIC BASIS FOR $CO_2$ 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 ($CO_2$) absorbent material. This design efficiently uses the gas supply because a portion of the diver's exhaled breath is re-circulated through the system. Additional gas is added to the UBA to replace the oxygen consumed by the diver. As the absorbent becomes expended, the $CO_2$ level within the breathing loop will rise. Because $CO_2$ does have a physiologic effect that may affect a diver's performance, the question becomes 'how much $CO_2$ is too much.'

Historically, canister limits were defined as the time it takes in minutes or the liters of $CO_2$ absorbed until the canister effluent gas $CO_2$ reaches 0.5% Surface Equivalent Value (SEV)[1,2]. 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 $CO_2$ ($PCO_2$) 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 $CO_2$.

## METHODS

The primary concerns of low levels of $CO_2$ 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 $CO_2$ level and potentiate any $CO_2$ problem. Furthermore, high levels of $CO_2$ 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 $CO_2$ on decompression sickness must also be addressed. This literature review primarily concentrated on the articles frequently quoted when discussing the effects of $CO_2$. It must be realized that the studies involving $CO_2$ levels below 3% are sparse. Typically, studies used at least 5% $CO_2$ to ensure a physiologic effect could be observed.

## DISCUSSION

### INTERRELATIONSHIP OF RESISTIVE LOADS AND $CO_2$

To understand the physiologic effects of inspired $CO_2$, 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

$CO_2$ levels. In normal individuals, the alveolar $P_{CO_2}$ ($P_ACO_2$) is equal to the arterial $P_{CO_2}$ ($P_aCO_2$). It's the blood $CO_2$ that causes the physiologic effects.

$$P_ACO_2 = P_ICO_2 + 863\ \dot{V}_{CO_2} / \dot{V}_E\ (1-V_D/V_T) \qquad \text{Equation (1)}$$

Where:

- $P_ACO_2$ - alveolar $P_{CO_2}$ (mmHg)
- $P_ICO_2$ - inspired $P_{CO_2}$ (mmHg)
- 863 - factor for correcting $\dot{V}$ from STPD to BTPS
- $\dot{V}_{CO_2}$ - minute volume of $CO_2$ (liters per minute; STPD)
- $\dot{V}_E$ - 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 $CO_2$ levels. In addition, any impediment to ventilation will cause the $P_aCO_2$ to rise. For example:

$\downarrow \dot{V}_E$ when:
- $\uparrow$ gas density
- $\uparrow$ rig resistance
- $\uparrow$ inspired oxygen during exercise

Ventilation increases with increasing exercise. When the inspired fraction of $CO_2$ rises, there is a concomitant rise in ventilation[3]. Figure 1 illustrates the rise in minute ventilation with increasing work rates. When $CO_2$ is added to the inspired gas, the ventilation rate further elevates above the level expected from exercise alone.

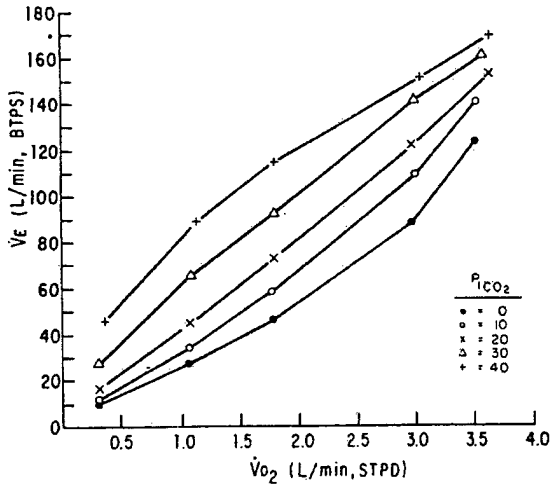


**Figure 1.** Relationship of ventilation to $O_2$ uptake during exercise at different levels of inspired $CO_2$ tension. The average values of oxygen uptake ($\dot{V}_{O_2}$) at rest and at 4 different work loads were not significantly altered by changes in $P_ICO_2$.

Reprinted from: Clark J. M., Sinclair, R. D. and J. B. Lenox. Chemical and nonchemical components of ventilation during hypercapnic exercise in man. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 48(6): 1065-1076,1980.

Clark et al., further demonstrated that the level of inspired $CO_2$ affects the arterial $CO_2$[3]. For exercise performed on the surface breathing air, a person actually hyperventilates during exercise performed at 50% of maximal oxygen uptake ($\dot{V}_{O_2}max$) and greater. Figure 2 illustrates that when the $P_ICO_2$ is increased above 20 mmHg, the $P_aCO_2$ dramatically rises for the level of exercise.

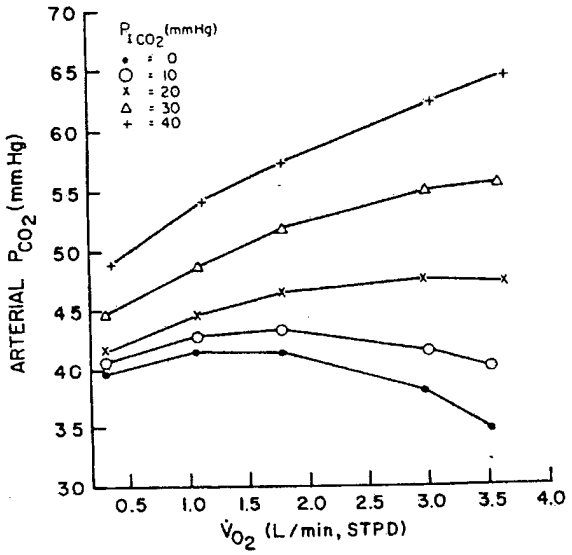


**Figure 2.** Arterial $PCO_2$ during exposure to combined exercise and hypercapnia.

Reprinted from: Clark J. M., Sinclair, R. D. and J. B. Lenox. Chemical and nonchemical components of ventilation during hypercapnic exercise in man. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 48(6): 1065-1076,1980.

Experiments performed by Poon illustrate the ventilatory responses to hypercapnea and exercise when breathing through an inspiratory resistive load (~12 $cmH_2O/l/s$)[4]. The investigator kept the end-tidal $PCO_2$ ($P_{ET}CO_2$) 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 $P_{ET}CO_2$ exceeds 40 mmHg. Therefore, breathing patterns are a result of a balance of chemical drive and the propensity to reduce respiratory effort.

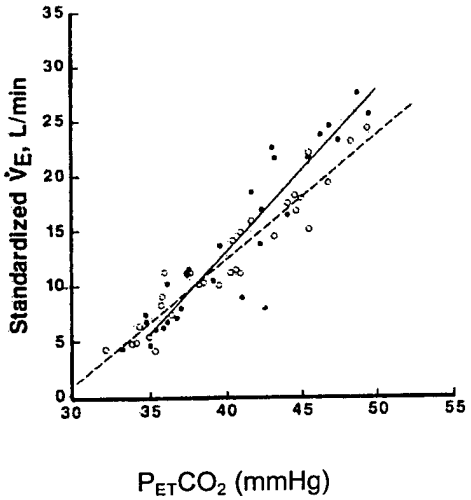


**Figure 3.** Responses in minute ventilation to $P_{ET}CO_2$ under no load (filled circles, solid line) and inspiratory resistive load ~12 $cmH_2O/l/sec$ (open circles and dashed lines).

Reprinted from: Poon, C. S. Effects of inspiratory resistive load on respiratory control in hypercapnia and exercise. J. Appl. Physiol. 66(5): 2391-2399,1989.

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 $cmH_2O/l/sec$, there's a distinct increase in $P_{ET}CO_2$, which increases with exercise load; whereas, at resistances less than 5 $cmH_2O/l/sec$ there's no change in the $P_{ET}CO_2$ for the increasing work load[5]. Figure 4 illustrates the effect of resistance on $P_{ET}CO_2$.

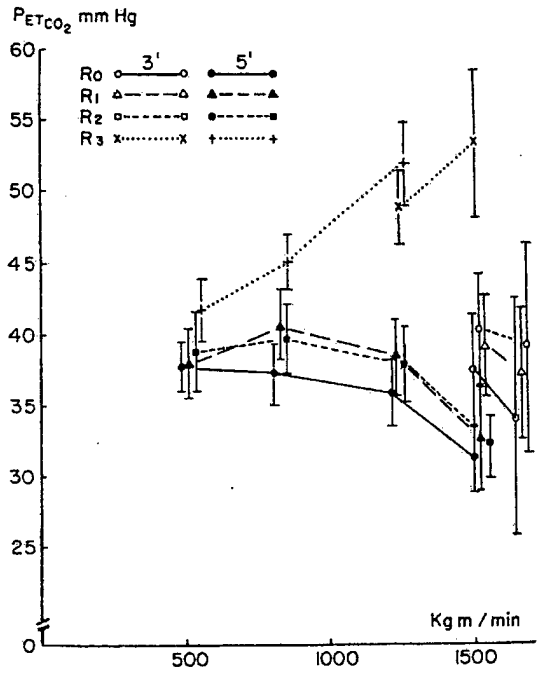


**Figure 4.** Effect of resistance on $P_{ET}CO_2$. *Ordinate:* end-tidal $CO_2$ tension (mmHg). *Abscissa*: work load in kgm/min. Mean values ±1 SE are shown for all subjects during the 5th min of exercise and when this was not available, in the 3rd min. The levels of resistance were:

$R_0$ - none
$R_1$ ~ 0.5 $cmH_2O/l/sec$
$R_2$ ~ 1.0 $cmH_2O/l/sec$
$R_3$ ~ 15 $cmH_2O/l/sec$

Reprinted from: Demedts, M. and Anthonisen, N. R. Effects of Increased External Airway Resistance During Steady-state Exercise. J. Appl. Physiol. 35(3): 361-366, 1973.

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% $CO_2$[10]. Figure 5 illustrates that when $CO_2$ is added to the inspiratory gas with a resistance of 1.5 cm $H_2O$/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 $H_2O$/l/sec (R4) combined with 4.3% $P_ICO_2$, 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 $CO_2$ and work performance is not impacted.

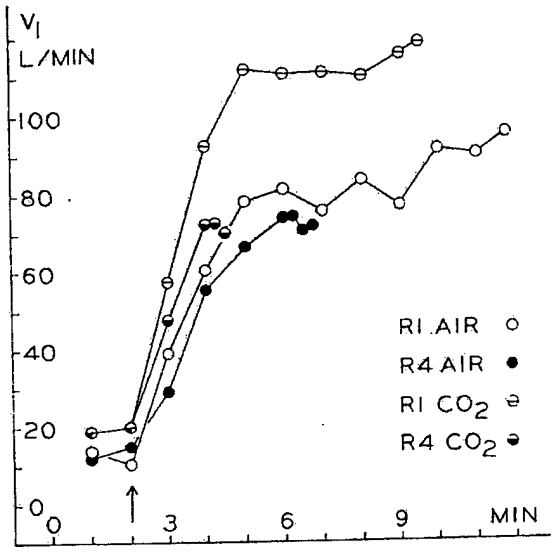


**Figure 5.** Respiratory minute volume during walks under varied conditions of inspiratory resistance and inhalation of carbon dioxide. Arrow marks beginning of walking. Concentrations of carbon dioxide in inspired air were 3.9% at R1 and 4.3% at R4.

Reprinted from: Craig, F. N. Blevins, W. V. and E. G. Cummings. Exhausting Work Limited by External Resistance and Inhalation of Carbon Dioxide. J. Appl. Physiol. 29(6): 847-851, 1970.

## $CO_2$ EFFECTS ON COGNITIVE PERFORMANCE

The increase in a person's $P_aCO_2$ is referred to as $CO_2$ retention. Therefore, the effect of an increased $P_ICO_2$ must be evaluated in light of an increased $P_aCO_2$. Henning et al., evaluated the behavioral effects of an elevated $P_ICO_2$ to clarify the risks due to $CO_2$ retention[11]. For this study, the subjects breathed 6% $CO_2$ 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 $CO_2$ retention. Furthermore, if a diver hypoventilates and if a high breathing resistance is suddenly added, the result can be sudden unconsciousness[12]. 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 $CO_2$ 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 $CO_2$ exceeded 3.8 mmHg (0.5% SEV)[13]. It should be noted that the divers in that study did not report any other symptoms even though the canister effluent $CO_2$ 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 $CO_2$ 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 $CO_2$ and its affect on ventilation. However, all the studies reviewed only found symptoms when the diver was exposed to very high resistances or a $P_ICO_2$ 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% $CO_2$ in 21% and 50% oxygen on cognitive performance during exercise and recovery on the surface[14]. They reported no deterioration in psychomotor and mental performance for the inhalation of up to 5% $CO_2$. The investigators noted that the short-term memory test they used detected effects due to strenuous exercise, whereas low levels of $CO_2$ did not cause a decrement in memory performance. Recalling Equation (1), if the $P_ACO_2$ is kept below 5% by appropriate ventilation for the level of $P_ICO_2$, the diver's cognitive performance would not be limited.

However, the MK 16 with a $PO_2$ 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 $CO_2$ and nitrogen narcosis[15]. This study used $CO_2$ 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 ($P_IN_2$) and the inspired $CO_2$ ($P_ICO_2$) rose simultaneously, their effect on performance was greater than the arithmetic sum of the changes induced by either gas alone. Allowing the $P_ICO_2$ to increase to 2% may potentiate the risk of nitrogen narcosis.

## $CO_2$ EFFECTS ON CENTRAL NERVOUS SYSTEM $O_2$ TOXICITY

An increase in the $P_ICO_2$ may reduce the time for the development of Central Nervous System (CNS) oxygen toxicity symptoms. The mechanism for this belief is the fact that $CO_2$ will cause a cerebral vasodilatation[16] resulting in an increase in brain oxygenation. Figure 6 illustrates the exponential rise in cerebral blood flow when the arterial $PCO_2$ rises.

As discussed earlier, $P_aCO_2$ 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[17]. Figure 7 shows 6 subjects' $P_aCO_2$ 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 $P_aCO_2$ 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.

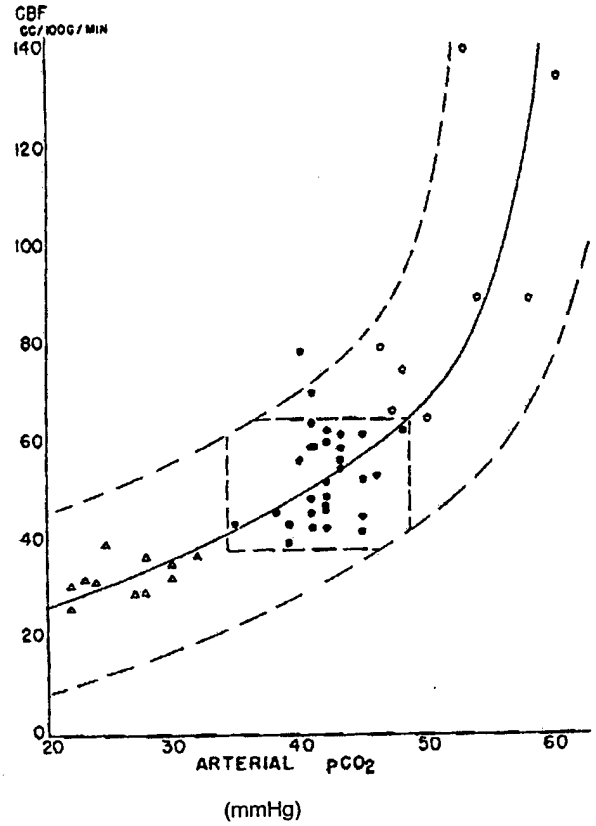


**Figure 6.** The relationship between cerebral blood flow and arterial $CO_2$ tension. The arterial $PCO_2$ varied from the normal (dots) by hyperventilation (triangles) or by inhalation of 5-7% $CO_2$ (open circles). The dashed lines bound 98% of the observations while the central polygon encloses 94% of the normal values.

Reprinted from: Kety, S. S. and C. F. Schmidt. The Effects of Altered Arterial Tensions of Carbon Dioxide and Oxygen on Cerebral Blood Flow and Cerebral Oxygen Consumption of Normal Young Men. Journal of Clinical Investigations, 1948, 27, 484-492.

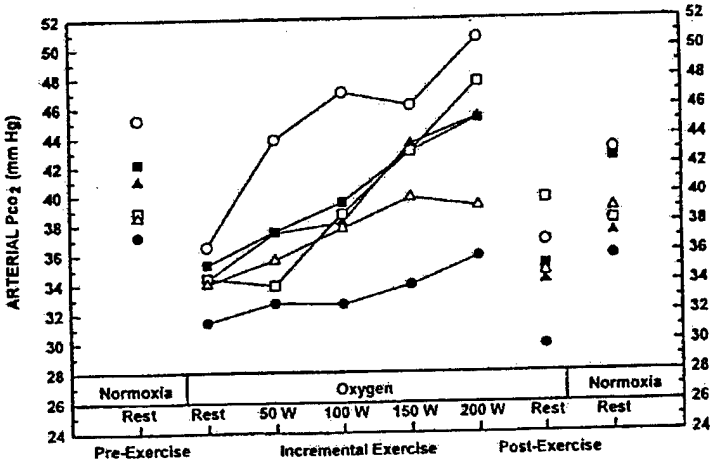


**Figure 7.** Individual arterial $PCO_2$ responses to incremental exercise while breathing $O_2$ at 2 ATA. Data are shown for 6 subjects who completed all 4 workloads.

Reprinted from: Clark J. M., R. Gelfand, C. J. Lambertsen, W. C. Stevens, G. Beck and D. G. Fischer. Human tolerance and physiological responses to exercise while breathing oxygen at 2.0 ATA. Aviat. Space Environ. Med. 1995; 66: 336-345.

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[18]. These studies simulated a variety of conditions: 1) $P_{O_2}$ of 1, 3 and 3.5 ATA while at rest; 2) $P_{O_2}$ of 2 ATA while exercising; 3) $P_{O_2}$ of 3.5 ATA with 2% $P_{CO_2}$ 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.

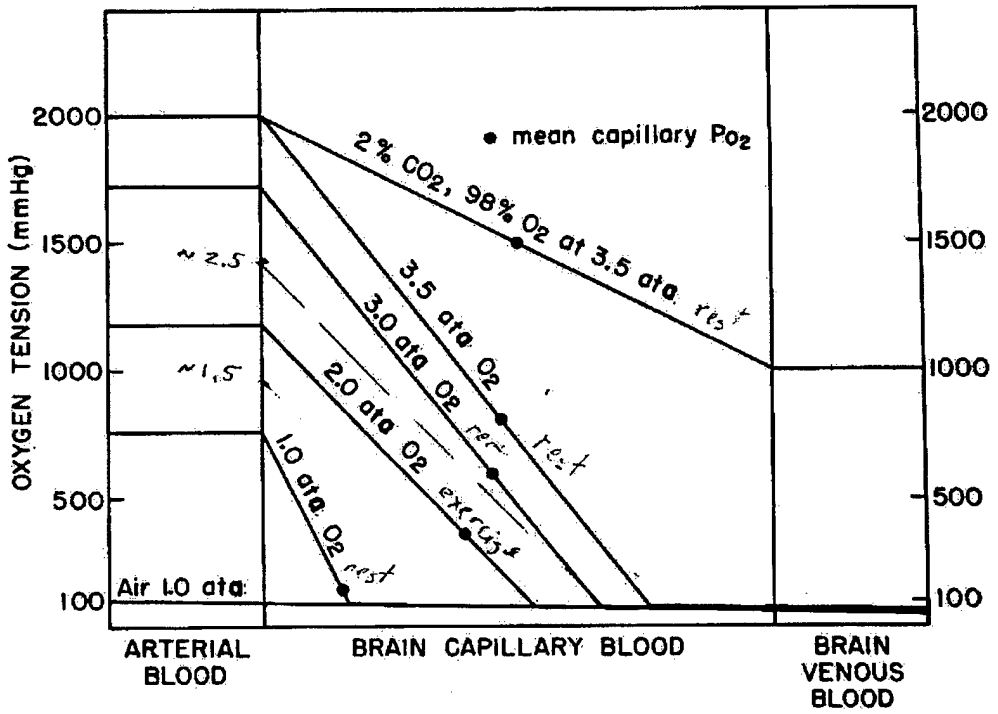


**Figure 8.** Effect of increased inspired $PO_2$ on the oxygen tensions of arterial, mean brain capillary, and internal jugular venous blood (average values in normal men). The graph illustrates for each of several levels of inspired $PO_2$ the manner in which oxygen tension across the mean brain capillary 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.

Reprinted from: Lambertsen, C. J. Effects of Hyperoxia on Organs and Their Tissues. In: Extrapulmonary Manifestations of Respiratory Disease, ed. Eugene Debs Robin. Vol. 8 of Lung Biology in Health and Disease, ed. Claude Lenfant. New York: Marcel Dekker, 1978.

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 $P_{O_2}$ of 2.5 ATA (100% oxygen at 50 fsw), and inspiring 2% $CO_2$, 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 $P_{O_2}$ 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 $P_ICO_2$ 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.

## $CO_2$ EFFECTS ON DECOMPRESSION SICKNESS

In 1908 Boycott, Damant and Haldane proposed that an elevated $CO_2$ 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 $CO_2$ 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, $CO_2$ 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 $CO_2$ 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 $CO_2$ exposure time for the decompression profile. Therefore, based on this article it's unknown if there's a dose-response relationship of $CO_2$ exposure to an increased incidence of DCS. The only conclusion is that an elevated $CO_2$ may result in an increased incidence of DCS.

## CONCLUSION

The literature suggests that a short duration exposure, 15 minutes, to a $CO_2$ level of 2% SEV may not result immediately in a catastrophic effect on a diver. Though there is a distinct relationship of $CO_2$ to CNS oxygen toxicity with a sustained elevated inspired $CO_2$, a momentary exposure would not greatly increase the risk of an oxygen convulsion.

Though the MK 16 diver using 0.7 $P_{O_2}$ 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 $P_{N_2}$ 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


1. Navy Experimental Diving Unit, *U.S. Navy Unmanned Performance Goals for Underwater Breathing Apparatus*. NEDU TECHMAN 01-94. Navy Experimental Diving Unit, 1994.

2. Hermann, J. T. *MK 15 MOD 0 UBA Canister Duration*. NEDU TM 88-05, Navy Experimental Diving Unit, July 1988.

3. Clark, J. M. R. D. Sinclair and J. B. Lenox. "Chemical and Nonchemical Components of Ventilation During Hypercapnic Exercise in Man". *J. Appl. Physiol.: Respirat. Environ. Exercise Physiol.* 48(6): 1065-1076, 1980.

4. Poon, C. S. "Effects of Inspiratory Resistive Load on Respiratory Load on Respiratory Control in Hypercapnia and exercise". *J. Appl. Physiol.* 66(5): 2391-2399,1989.

5. Demedts, M. and Anthonisen, N. R. "Effects of Increased External Airway Resistance During Steady-state Exercise". *J. Appl. Physiol.* 35(3): 361-366, 1973.

6. Lally, D. A., Zechman F. W., Tracy, R. A. "Ventilatory Responses to Exercise in Divers and Non-divers". *Respiration Physiology* 20: 117-129, 1974.

7. Kerem D., Melamed, Y., Moran, A. "Alveolar $P_{CO_2}$ During Rest and Exercise in Divers and Non-divers Breathing $O_2$ at 1 ATA". *Undersea Biomedical Research* 7(1): 17-26, March 1980.

8. Finkelstein, S., Elliot, J. C. and U. C. Luft. "The Effects of Breathing Low Concentrations of $CO_2$ on Exercise Tolerance". In: *Aerospace Medical Association, Preprints of Scientific Program*, 1970. Annual Scientific Meeting, St. Louis, April 27-30, 1970. Aerospace Medical Association.

9. Menn, S. J., R. D. Sinclair and B. E. Welch. "Effect of Inspired $PCO_2$ up to 30 mmHg on Response of Normal Man to Exercise". *J. Appl. Physiol.* 28(5): 663-671, 1970.

10. Craig, F. N. Blevins, W. V. and E. G. Cummings. "Exhausting Work Limited by External Resistance and Inhalation of Carbon Dioxide". *J. Appl. Physiol.* 29(6): 847-851, 1970.

11. Henning, R. A., Sauter, S. L., Lanphier, E. H. and W. G. Reddan. "Behavorial Effects of Increased $CO_2$ Load in Divers". *Undersea Biomed. Res.* 17(2): 109-126, 1990.

12. Warkander, D. E., Norfleet, W. T. Nagasawa, G. K. and C. E. G. Lundgren. "$CO_2$ Retention with Minimal Symptoms but Severe Dysfunction during Wet Simulated Dives to 6.8 Atm Abs". *Undersea Biomed. Res.* 17(6): 515-523, 1990.

13. Knafelc, M. E. *MK 15 MOD 0 Alternate Carbon Dioxide Absorbent Materials*. NEDU TR 11-87, Navy Experimental Diving Unit, July 1987.

14. Sheehy, J. B., Kamon, E. and Kiser, D. "Effects of Carbon Dioxide Inhalation on Psychomotor and Mental Performance During Exercise and Recovery". *Human Factors* 581-588, October 1982.

15. Hesser, C. M., Adolfson J., Fagraeus L. "Role of $CO_2$ in Compressed-Air Narcosis". *Aerospace Medicine* 42(2): 163-168, 1971.

16. Kety, S. S. and C. F. Schmidt. "The Effects of Altered Arterial Tensions of Carbon Dioxide and Oxygen on Cerebral Blood Flow and Cerebral Oxygen Consumption of Normal Young Men". *Journal of Clinical Investigations* 27, 484-492, 1948.

17. Clark, J. M., R. Gelfand, C. J. Lambertsen, W. C. Stevens, G. Beck and D. G. Fischer. "Human Tolerance and Physiological Responses to Exercise while Breathing Oxygen at 2.0 ATA". *Aviat. Space Environ. Med.* 66: 336-345, 1995.

18. Lambertsen, C. J. "Effects of Hyperoxia on Organs and Their Tissues". In: *Lung Biology in Health and Disease*, Ed. by Claude Lenfant, Vol 8, *Extrapulmonary Manifestations of Respiratory Disease*, Ed. by Eugene Debs Robin, 239-296, New York

19. Hodes, R. and M. G. Larrabee. "The Relation Between Alveolar Carbon Dioxide Tension and Susceptibility to Decompression Sickness". *Am. J. Physiol.* 147(4): 603-615, 1946.

20. Mang Y. and D'Arrigo, J. S. "Relationship Between $CO_2$ Levels and Decompression Sickness: Implications for Disease Prevention". *Aviat. Space Environ. Med.* 49(2): 349-355, 1978.