

EFFECTS OF NITROGEN NARCOSIS AND HYPERCAPNIA ON MEMORY

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INTRODUCTION

Divers are exposed to many occupational hazards when working at depth including those of N₂ narcosis and CO₂ retention. Conditions under which N₂ narcosis and hypercapnic stress are combined are considered a major threat to diver safety and have been blamed for the sudden loss of consciousness in some divers while working at depth^{1,2}. At lower partial pressures than those that induce unconsciousness both high PN₂ and PCO₂ levels have been shown to significantly impair cognitive function^{3,4}. Current evidence suggests that P_IN₂ and P_{ET}CO₂ are simply additive in their impairment on cognitive performance^{3,4}, however, this has yet to be demonstrated for higher cognitive functions that involve learning or memory processes.

The aim of this study was to examine the N₂ and CO₂ components of narcosis on immediate and delayed recall (IR and DR respectively) of a paired association task. Narcosis was induced by exposure to hyperbaric air at 6 ATA in a hyperbaric dry chamber. At both surface (1ATA) and 6 ATA ambient pressures three ranges of P_{ET}CO₂ were studied LOW (25 - 35 mmHg), MEDIUM (45 - 50 mmHg), HIGH (55 - 60 mmHg).

METHODS

Twelve healthy male volunteers (mean age 25.6 yrs, SD=0.6; mean mass 76.4 kg, SD=0.7), from the university population took part in the study. Each subject obtained medical clearance for diving and signed an informed consent release. All experiments were conducted in the Simon Fraser University Environmental Physiological Unit's (E.P.U.) hyperbaric chamber under dry conditions.

A semi-closed respiratory circuit was used to elevate and maintain P_{ET}CO₂ between the desired ranges for the duration of the experiment. The full experimental set up is described in detail elsewhere¹. Briefly, the subject inspired from a regulator (Conshelf 30, U.S. Divers) through a length of respiratory tubing connected to a mixing box. Expired gases were either vented to the chamber via a two-way valve in the exhalation line, (LOW P_{ET}CO₂ condition) or redirected to the mixing box for rebreathing (MEDIUM and HIGH P_{ET}CO₂ conditions). The level of hypercapnia for the MEDIUM and HIGH P_{ET}CO₂ conditions were induced by the introduction of appropriate lengths of 0.04m diameter tubing in the rebreathing circuit. Breath by breath gas samples were drawn from the mouthpiece through the chamber wall and analyzed for end-tidal and inspired CO₂ concentration (Ametek Applied Electrochemistry CD-3A CO₂ analyzer). Output from the CO₂ analyzer was passed to an IBM(PC) micro-computer via an analogue to digital converter (Tecmar Labpac) for sampling (rate = 33 Hz) and storage.

The paired association test involved learning 12 pairs of word and number combinations. A different set of 12 words were used during each testing session. All words were randomly drawn from the pool developed by Modigliani and Seamon⁵ and consisted of common one and two-syllable concrete nouns. The number associated with each word was randomly assigned from a list of numbers between 1 and 100. To minimize proactive interference between successive memory tests, a number, once it had been used, was omitted from inclusion in subsequent word/number lists until all numbers between 1 and 100 had been used. Word/number pairs were presented sequentially at 5 second intervals with the previous word/number pair removed from the field of view. Immediate recall was tested in the minute immediately following presentation of the last word/number pair and delayed recall tested approximately 15 minutes later. In between IR and DR tests subjects completed a number of other cognitive tests thus preventing rehearsal of the paired associations. Responses were recorded on a test paper containing only the rearranged words. A correct response was recorded when the correct number was matched with the corresponding word.

At the start of the study all subjects performed a block practice session (five practice trials) on the paired association test. In addition, immediately prior to conducting each experiment an additional practice trial was conducted in the laboratory at surface pressure. Presentation of the two pressure conditions and three P_{ET}CO₂ levels was counter balanced across the subjects according to a fully randomized and balanced design. Each subject completed all experimental conditions with only one condition performed on a given subject on any one day.

During experimental trials subjects breathed on the respiratory circuit for a **minimum** of 7 minutes before presentation of the word/number list. For most subjects this time period was sufficient to elevate their $P_{ET}CO_2$ levels to the desired range. If a subject's $P_{ET}CO_2$ rose above the desired range, it was lowered by opening the two-way valve on the exhalation side of the rebreathing circuit. This allowed exhaled concentrations of CO_2 to be vented to the chamber atmosphere, thus lowering the $P_{I}CO_2$ in the rebreathing circuit until $P_{ET}CO_2$ returned to the target range. During surface trials 100% O_2 was supplied to the mixing box at a flow rate of 0.5 liters/min to prevent inspired PO_2 dropping below normal levels during rebreathing. At 6 ATA the elevated $P_{I}O_2$ caused by the ambient pressure increase was considered sufficient to prevent hypoxic stress during rebreathing ($P_{I}O_2$ at 6 ATA = 1.26 ATA).

IR was tested during the minute immediately following presentation of the word/number list while the experimental conditions of pressure and $P_{ET}CO_2$ still prevailed. DR was tested 15 minutes later and 5 minutes after the subject had been taken off the respiratory circuit. In the 6 ATA experiments DR was tested at the first decompression stop (between 2 and 3 ATA) where $P_{I}N_2$ levels were considered to be non-narcotic.

RESULTS

Mean \pm standard deviations for the LOW, MEDIUM and HIGH levels of $P_{ET}CO_2$ averaged across the 1 and 6 ATA conditions for all subjects were 29 ± 4 mmHg, 47 ± 1 mmHg and 57 ± 2 mmHg respectively. Figure 1 shows the effects of increasing $P_{ET}CO_2$ levels and raising the ambient pressure from 1 to 6 ATA on IR and DR responses. A two-way repeated measures ANOVA on the total number of word/number pairs correctly recalled revealed that both N_2 narcosis and elevated levels of $P_{ET}CO_2$ produced significant decrements in IR scores ($p < 0.0001$ and $p < 0.05$ for N_2 narcosis and hypercapnia respectively). Delayed recall was significantly impaired following the 6 ATA experiments ($p < 0.001$) compared to performance following the surface trials, but was not significantly affected by the previous $P_{ET}CO_2$ level ($p > 0.05$). There was no interaction between N_2 narcosis and $P_{ET}CO_2$ levels on either the IR or DR responses ($p > 0.05$).

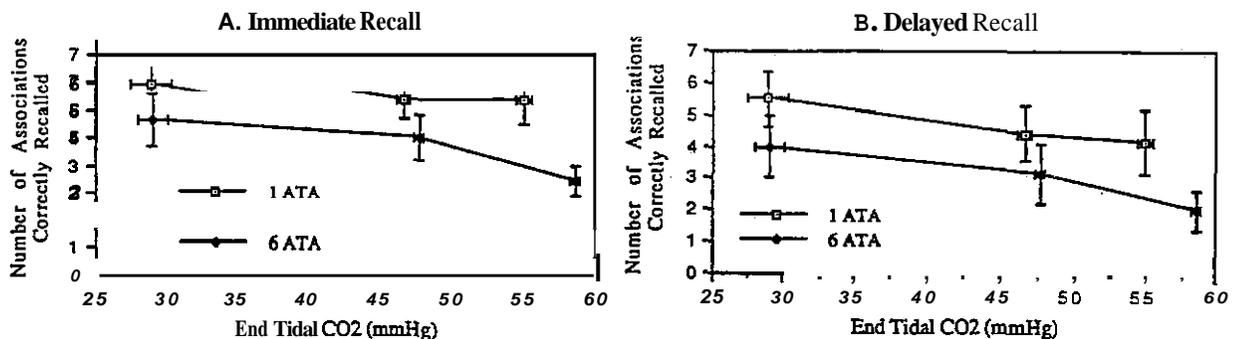


Fig. 1: The combined and separate effects of N_2 narcosis at 6 ATA and changes in $P_{ET}CO_2$ on IR and DR.

CONCLUSIONS

N_2 narcosis and high $P_{ET}CO_2$ levels were found to impair learning/memory processes in an additive fashion when both stressors were present in the hyperbaric environment. The fact that DR responses were impaired under non-narcotic conditions following the 6 ATA trials suggests that N_2 narcosis interferes with input of information into long term memory. This hypothesis does not however seem to account for the IR decrements seen under high levels of $P_{ET}CO_2$.

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