

HUMAN HEALTH RISK ASSESSMENT OF CO₂: SURVIVORS OF ACUTE HIGH-LEVEL EXPOSURE AND POPULATIONS SENSITIVE TO PROLONGED LOW-LEVEL EXPOSURE

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INTRODUCTION

The health of individuals near carbon transport and sequestration sites must be considered in site risk characterization. The lethal effects of high CO₂ concentrations are well known, but the literature also reveals cause for concern for both the survivors of high-level CO₂ exposure and individuals who experience prolonged low-level exposure. These effects are discussed below. This work is part of an on-going project to evaluate the adverse health effects of CO₂.

CO₂ TOXICITY

CO₂ has a continuum of effects that range from physiologic (e.g., ventilatory stimulation) to toxic (e.g., cardiac arrhythmias and seizures), anesthetic (significantly depressed CNS activity), and lethal (severe acidosis and anoxia). The effects of CO₂ in a specific individual depend on the concentration and duration of exposure as well as individual factors, such as age, health, physiologic make-up, physical activity, occupation, and lifestyle.

With high-level CO₂ exposure, the displacement of O₂ by CO₂ significantly contributes to toxicity. Signs of asphyxia are evident when the atmospheric O₂ is ≤16% [1]. Almost immediate unconsciousness leading to death occurs in humans at rest when the O₂ is reduced to 10 to 13%. Strenuous physical exertion increases the threshold [2].

In several studies, intoxication leading to unconsciousness was evident in ≤30 s in patients inhaling 30% CO₂ in 70% O₂. Some patients exhibited seizures that were characterized as decerebrate (no cerebral functioning) [3,4]. At this concentration, 71% of patients in one study had ECG abnormalities of atrial or nodal activity, including premature atrial and nodal beats, and atrial tachycardia [5]. Rhesus monkeys exposed to CO₂ in 21% O₂ exhibited arrhythmias at ~26% CO₂ and died at >60% CO₂ [6]. At the time of death, the ECG showed asystolic arrest, which is also reported to occur with a blood pH between 6.45 and 6.50 resulting from severe acidosis of origins other than that of inhaled CO₂.

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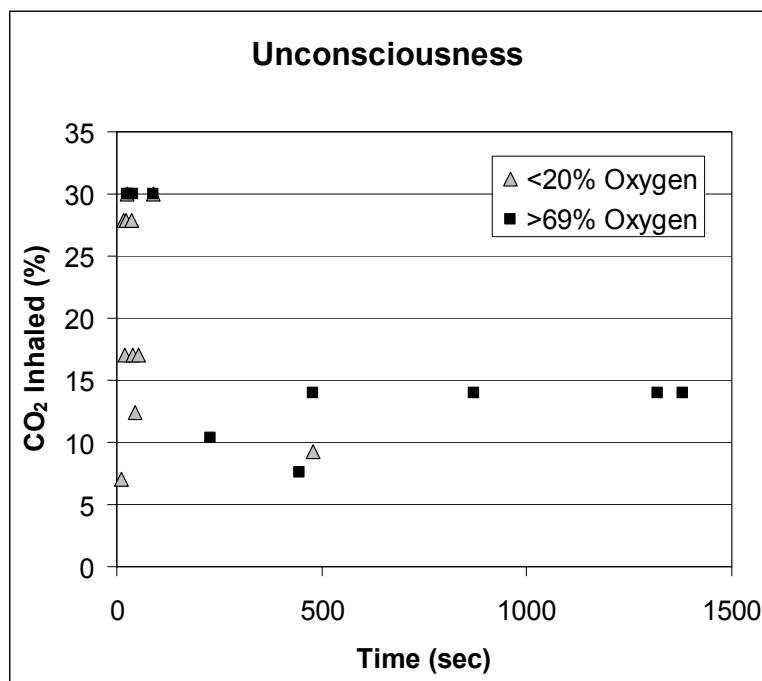


Figure 1. Time to unconsciousness in humans exposed to CO₂ in high and low O₂ environments [3,5,7-15].

HIGH-LEVEL EXPOSURE AT LAKE NYOS, CAMEROON, 1986

The Lake Nyos CO₂ release, where an estimated 1700 people died and a maximum 5000 survived, has provided insight into the morbidity and mortality resulting from a high-level CO₂ exposure of a large population [16-19]. Many survivors recount a rapid loss of consciousness. The atmospheric CO₂ was estimated to be ≥ 8 to 10% because oil lamps were extinguished [20]. Deaths were presumably caused by CO₂ displacing O₂ resulting in asphyxiation. Pulmonary congestion and/or edema were prominent findings in the few autopsies conducted. Postmortem changes, however, precluded other useful information.

The effects of CO₂ exposure were dependent on the distance from the lake and the duration of exposure. Survivors within 3 km of the lake experienced fatigue, light-headedness, warmth, and confusion immediately prior to unconsciousness, which lasted for up to 36 hr. Survivors 3 to 10 km from the lake reported shortness of breath, dizziness, and confusion prior to losing consciousness. These and other reported differences among groups of survivors are attributed to variations in the CO₂ and O₂ concentrations. The close proximity of survivors and the dead also strongly suggests that there is considerable interindividual variation in response to equivalent exposures of CO₂ and O₂.

Signs and Symptoms in Lake Nyos Survivors

Investigators examined local hospital records of walk-ins and admissions for two weeks following the release [18,19]. A total of 870 survivors were seen at the two local hospitals. The presenting signs and symptoms that were thought to be related to the exposure are shown in Table 1. All the survivors claimed to have lost consciousness for hours.

Table 1: Signs and Symptoms of Survivors Presenting at the Hospital

Signs and Symptoms	% (No. of Survivors)
Cough	31 (226)
Headache	26 (216)
Fever (malaria is endemic)	12 (104)
Weakness/Malaise	11 (95)
Limb swelling	10 (85)
Weakness of arms or legs	6 (51)
Dyspnea or Eye symptoms	5 (45)
Vomiting or Diarrhea	5 (46,44)
Hemoptysis (blood in sputum)	3 (23)

Table 2: Other Signs and Symptoms of Survivors

Other Signs and Symptoms	Incidence
Transient proteinuria	Fairly common
Pulmonary edema and consolidation	Fairly common
Unilateral loss of hearing	“Several patients”
Miscarriages under 20 weeks gestation	7
Stillbirths at 32 week of gestation	1
Stillbirths near term	3 of 6 known

No long-term health effects have been reported, however, there were no follow-up studies. Tests that could have identified CNS damage, which would be expected from severe hypoxia, were not performed.

OTHER ACCIDENTAL HIGH-LEVEL EXPOSURES

There are many reports of accidental and intentional exposures to CO₂ leading to intoxication or partial incapacitation and death due to hypercapnia, most often in the presence of severe hypoxia [21]. For most accidental exposures there is no record of the CO₂ or O₂ concentrations present in either the inspired air or the blood of the victim. Several cases show the relationship of CO₂ and O₂ concentrations to narcosis, unconsciousness, and death. In one report, air crewmen were partially incapacitated when the aircraft CO₂ fire extinguisher was accidentally discharged

resulting in an estimated concentration of 4 to 7% CO₂ [22]. The near collapse of other air crew members from the inhalation of CO₂ occurred during several flights due to the release of CO₂ from dry ice used to transport frozen foods [21-24]. In one case, it took only 10 min for an estimated 14.8% CO₂ to accumulate.

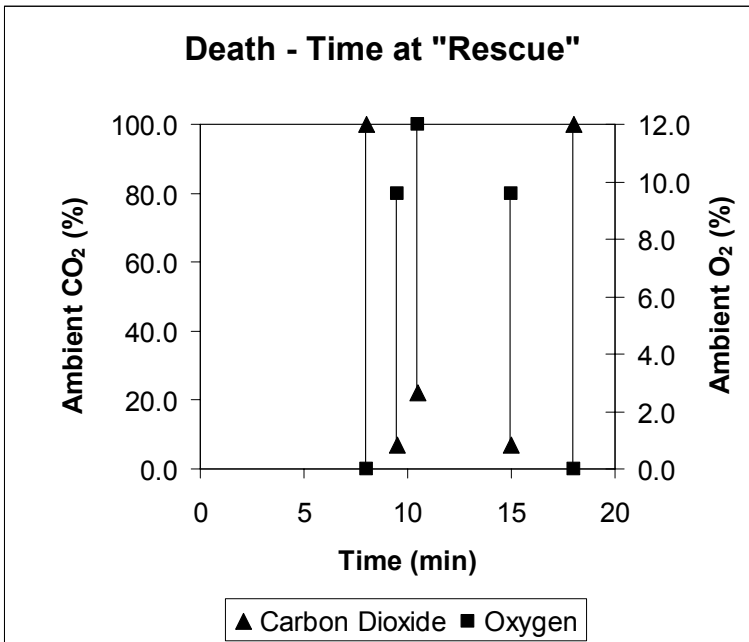


Figure 2.

The ambient CO₂ and O₂ concentrations measured or estimated where death occurred [7,25,26]. Times shown are the time at which the victim was removed from the CO₂ environment, and not the actual time to death.

Moderate to high concentrations of CO₂ are rapidly fatal in the presence of O₂ concentrations $\leq 12\%$. CO₂-related deaths have been reported for enclosed sites, such as grain silos, cargo holds of ships, composting and processing plants, deep wells, and mines. In grain silos, CO₂ may commonly reach 38% and cause asphyxiation by displacement of O₂ [20,27]. The cargo holds of ships have become filled with as high as 22% CO₂ from fermenting produce and fresh and putrefying trash and fish [25,28]. In these cases, O₂ is displaced and unconsciousness is rapid.

Signs and Symptoms of Short-Term, High-Level Exposure

Signs and symptoms among the survivors of short-term, high-level exposures include: unconsciousness, cyanosis, sluggish reflexes, rattling respiration, and excessive motor unrest. All these signs and symptoms cannot be attributed exclusively to CO₂, however, because O₂ was displaced by CO₂, which resulted in varying degrees of hypoxia in addition to hypercapnia.

A comatose man, rescued 10 min after he entered a well in which three of his co-workers died, remained comatose and completely unresponsive until his death 11 months later [29]. The autopsy, limited to the brain and eye, showed severe brain atrophy and major retinal abnormalities. The nonselective, widespread brain damage and the severity of the individual lesions contrasted with the expected findings from death caused by anoxia. It was concluded that the brain lesions were due to the histotoxic effects of CO₂. The effects on the eye were considered similar to those caused by anoxia.

Persistent health effects have been identified in those that recover consciousness after high-level CO₂ exposure in the presence of low-level O₂. In one report of CO₂ asphyxiation, two of three men regained consciousness after exposure in a well [30]. Both men experienced throbbing headaches, attacks of vertigo, poor memory and ability to concentrate, photophobia, difficulty sleeping, tinnitus, and double vision. The man exposed for the longest time also underwent a marked personality change and suffered from loss of eye movement as well as from visual field defects, enlargement of blind spots, and deficient dark adaptation.

High-Level Exposure Summary

Confined spaces appear to offer the greatest risk for adverse effects from the displacement of O₂ by CO₂ because little air mixing occurs. Spontaneous recovery from high-level, CO₂-induced coma has occurred when adequate O₂ was present during exposure and the individual did not become too acidotic.

PROLONGED LOW-LEVEL CO₂ EXPOSURE

Prolonged exposure to low CO₂ concentrations has rather subtle, seemingly benign effects. Only healthy, relatively young adults have been studied thus far, however, which raises a concern for these same effects in potentially sensitive populations. Table 3 lists some of the outcomes of low-level CO₂ exposure with the potential to cause immediate or long-term, adverse effects. These outcomes are briefly discussed with reference to potentially sensitive populations.

Table 3: Potentially Adverse Outcomes of Prolonged Low-Level CO₂ Exposure

%	Duration of Exposure	Potentially Adverse Outcome
0.85	20 days	↑ Lung dead space volume [31]
1.2	3 days	35% ↑ Cerebral blood flow [32]
1.2	5 days	~20% ↑ Blood pressure [33]
1.2	25 days	Significant ↓ Biomarkers of bone formation Slight ↑ bone resorption [34]
1.5	42 days	↑ Urine volume & Na, K, Cl excretion Slight ↑ Hct, RBC count, Hemoglobin [35]
1.5	42 days	Significant ↑ Lung dead space volume [36-39]
2.0	30 days	Slight ↑ Lung dead space volume [10]
3.0	8 days	Significant performance decrements Erratic, abnormal behavior [41]

Potential Consequences of Prolonged Low-Level CO₂ Exposure

Lung dead space volume is a volume of air where gas exchange does not occur. The larger the dead space, the less gas exchange, which can be important for those with pulmonary or cardiac problems. Patients with severe CO₂ retention (P_aCO₂ >95 mmHg) are invariably acidotic and will be at greater risk for profound acidosis with elevated ambient CO₂ concentrations because they cannot further compensate for elevations in P_aCO₂, except with limited increases in ventilation. In addition, although the CO₂-induced increase in the dead space volume was reported to be reversible in healthy subjects, it is not known if this reversal also occurs in patients with pulmonary disease.

Elevated systemic and pulmonary blood pressure is well tolerated by healthy individuals but can exacerbate preexisting systemic or pulmonary hypertension. Increased cerebral blood flow and increased intracranial pressure can put preterm infants at increased risk for intraventricular hemorrhage. Adults with brain injuries, tumors, bleeding, or increased intracranial pressure are also at risk with further increases in intracranial pressure.

Slightly decreased bone formation and increased bone resorption in healthy young individuals may have no long-term consequences but may be detrimental to persons with bone disease, such as osteoporosis. Another potential problem is increased blood calcium, which is related to changes in the bone. In guinea pigs and rats, focal renal calcification has been observed with prolonged exposure to concentrations of CO₂ ranging from 0.3 to 15% [42-48]. The authors characterized the kidney calcification as an adaptive disease. Studies of humans have not examined calcification in the kidney or other organs.

Low-Level Exposure Summary

Prolonged exposure to low-level CO₂ is not immediately life threatening, but it may have health consequences for healthy individuals as well as sensitive populations. Some of the observed effects in studies reported prior to the 1980s have been questioned for technical reasons, and only additional studies can determine the true potential of prolonged CO₂ exposure to induce adverse health outcomes.

SUMMARY

Acute high-level CO₂ exposure in the presence of low-level O₂ can produce significant persistent adverse health effects including headaches, attacks of vertigo, poor memory and ability to concentrate, difficulty sleeping, tinnitus, double vision, photophobia, loss of eye movement, visual field defects, enlargement of blind spots, deficient dark adaptation, and personality changes.

Prolonged low-level CO₂ exposure ($\leq 3\%$) in the presence of normal O₂ can produce relatively benign short-term effects in healthy, young adults. One effect, alterations in bone metabolism and related blood calcium concentrations, however, may have potentially longer-lasting adverse effects in both healthy and sensitive populations. The bone and calcium alterations in animals have been associated with renal calcification at CO₂ concentrations ranging from 0.3 to 15%. Other benign effects of CO₂ exposure in healthy study subjects may have more immediate

consequences for the health of sensitive populations. Additional studies must be conducted to define the actual risks for a given potentially sensitive population.

Risk characterization of carbon transport and sequestration sites must include the potential health effects from prolonged low-level CO₂ exposures as well as the better recognized effects of high-level CO₂ exposures.

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