CARBON DIOXIDE (CO_2)

NORMAL PHYSIOLOGY & HAZARDS AND RISKS

by Ted Schettler, MD, MPH, Science Director, Science and Environmental Network (SEHN)

CO_2 IN NORMAL PHYSIOLOGY

In humans, carbon dioxide (CO_2) is produced in cells as a byproduct of metabolism.[1] Then it circulates through the bloodstream, mostly within red blood cells, to be exhaled from the lungs as CO_2 and excreted from kidneys as bicarbonate (HCO_3^-).

CO_2 levels:[2]

- play a major role in regulating blood and tissue pH (acid-base balance)
- largely determine respiratory drive (rate and depth of breathing)
- influence oxygen attachment to hemoglobin

Internal physiologic processes tightly regulate CO_2 levels in the blood; abnormally high concentrations cause a variety of signs and symptoms. (See CO_2 toxicity below.)

Maintaining blood pH within a narrow range is essential for normal functioning of virtually all organs and physiologic systems. CO_2 plays a central role via these reactions:

$$CO_2 + H_2O \leftrightarrow H_2CO_3 \leftrightarrow HCO_3^- + H+$$

where H_2O is water, H_2CO_3 is carbonic acid; HCO_3^- is bicarbonate; H+ is hydrogen ion.

Humans and virtually every living organism (plants and animals) have evolved to produce an enzyme, carbonic anhydrase[3], which dramatically increases the speed of the first part of the above reaction, ($CO_2 + H_2O \leftrightarrow H_2CO_3$), making this an ideal system for very rapidly fine-tuning the regulation of CO_2 levels and blood pH through changes in the rate and depth of respiration.

CO_2 also reacts with water to form carbonic acid in a CO_2 pipeline, even in the absence of carbonic anhydrase, albeit much more slowly. Carbonic acid is highly corrosive to carbon steel.

The main drivers of respiratory rate and depth are normally bloodstream CO_2 levels and associated pH changes. These are maintained within healthy limits by chemoreceptors located in the medulla at the base of the brain (central chemoreceptors) and in the aorta and carotid arteries (peripheral chemoreceptors). Brain receptors detect changes in the CO_2 and pH, aorta receptors detect changes in CO_2 and oxygen, and carotid receptors detect changes in all three. Respiratory rate and depth increase primarily in response to an increase in CO_2/decrease in pH to reduce CO_2 and maintain pH within normal limits.

CO_2: AN ASPHYXIANT AND TOXICANT

CO_2 is colorless, odorless, non-flammable, and heavier than air. As a gas in air, concentrations will be higher near the floor or ground. It can be compressed at high pressures into a liquid or frozen at very low temperatures into a solid (dry ice).

CO_2 is classified as a hazardous substance by the Occupational Safety and Health Administration (OSHA) and National Institute for Occupational Safety and Health (NIOSH)[4]

Workplace exposure limits:

OSHA: Airborne permissible exposure limit (PEL) is 5000 ppm (0.5%) averaged over 8 hours.

NIOSH: Recommended airborne exposure limit (REL) is 5000 ppm averaged over 10 hours and not to exceed 30,000 ppm (3%) over any 15 minute period.

Ambient air contains about 0.04% (400 ppm) CO_2, 20% O_2, 79% nitrogen, and small amounts of other gases.

This air level of CO_2 has no direct adverse health effects in humans. As a potent greenhouse gas, however, this steadily-increasing atmospheric concentration is a major driver of climate change, which has wide-ranging direct and indirect health impacts globally.

Inhalation exposures to concentrations of CO_2 higher than OSHA and NIOSH exposure limits are hazardous in two ways, both of which contribute to signs and symptoms (see also table next page):

- Gaseous CO_2 is heavier than air and when released in concentrated amounts it flows downhill, collects in low-lying areas—indoors or outdoors—or confined, poorly-ventilated spaces such as basements, storage tanks, dry-ice refrigeration systems, fermentation areas, or mines. It displaces ambient air so that oxygen levels can fall to dangerously low levels. For that reason, CO_2 is well-known as an asphyxiant.

- CO_2 also has toxic properties in addition to causing oxygen-deprivation. Inhalation of high concentrations of CO_2 can sharply lower the pH of blood and tissues (acidosis) causing acute effects on the respiratory, cardiovascular, and central nervous systems.

The response to excessive CO_2 inhalation varies even among healthy individuals[5] so that the timing of onset of signs and symptoms of toxicity can vary from one person to another. This may be due to underlying health status, age, or variability in chemoreceptor sensitivity and respiratory response to acute CO_2 exposures.

Symptoms depend in large part on the concentration of inhaled CO_2 and the length of time a person is exposed. Since CO_2 is odorless and does not cause irritation, unlike some other gases, it gives no warning and people may be unaware of excessive exposures until they experience troubling, dangerous symptoms.

CO_2 TOXICITY [6]

CO_2 Concentration	Health Effect	Timing
2% (20,000 ppm)	Respiratory center stimulated causing increases in breathing (tidal) volume	Rapid
4% (40,000 ppm)	Increase in breathing rate becomes distressing; development of respiratory acidosis	Immediately dangerous to life and health (IDLH) [NIOSH] *
5-10%	Dimmed sight, sweating, tremor, increased heart rate and blood pressure; can →unconsciousness**	Within a few minutes
More than 10%	Can cause convulsions; coma (less than one minute)	Death within 10 minutes
20-30%	Loss of consciousness; death	Within one minute

* NIOSH considers this level to be immediately dangerous to life and health because it can cause confusion and impair ability to respond and get to safety. Signs and symptoms resulting from low to moderate exposures are generally reversible when a person is removed from a high CO_2 environment.
** Symptoms are due to a combination of lower oxygen levels and CO_2 toxicity.[7]

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