

Alcohol like Syndrome: Influence of Increased CO₂ Concentration in the respiration air

Yasser El-Nahhal

Dept of Earth and Environmental Science, Faculty of Science, The Islamic University-Gaza (IUGAZA). Tel.
00970599634708 ,

Email: y_el_nahhal@hotmail.com

Abstract

This article discusses the effect of CO₂ in losing sensation, Alcohol like syndrome (ALS) and in home sick syndrome (HSS), in poor conditions. The discussion revealed the formation of 2 moles of hydrogen ions as a result of elevated CO₂ partial pressure in the rebreathing air as a result of closed systems or covering heads with towels or blanket during sleep. This situation may result in a severe acidosis in the lung and transferring of Na⁺/K⁺ ions from the central nervous system to lung to react with excess of CO₃⁻. This situation may end by partial losing sensation as CO₂ level increased in the closed system which can be referred to Alcohol Like Syndrome (ALS). Recovery from ALS may occur immediately after rebreathing fresh air contains normal levels of oxygen, nitrogen and carbon dioxide. HSS may also appear in houses have bad ventilation and sun exposure during day.

Keywords: CO₂, Alcohol like syndrome, Home sick syndrome

1. Introduction

The sources of carbon dioxide (CO₂) in human body are metabolism of organic compounds, oxidation reduction reactions and inhalation from the atmosphere.

CO₂ levels have a diversity of effects on human body. For instance, Leibold et al. (2013) investigated the emotional and cardiovascular effects evoked by inhaling CO₂. They found that systolic and diastolic blood pressure rose with increasing CO₂ concentration, whereas heart rate results were less consistent. Moreover, Smith et al., (2013) evaluated the relationship between CO₂ inhalation and phonic respiration (breathing during speech) in respiratory protective devices. They concluded that Carbon dioxide (CO₂) rebreathing in respiratory protective devices has been highlighted as a key concern regarding respirator use. However, phonic respiration and low work rates contributed to significantly higher levels of CO₂ rebreathing. In addition, Gates et al., (2013) determined the effect of normoxic hypercapnia (10% CO₂ /21% O₂ /69% N₂) on outcomes of *Pseudomonas aeruginosa* pneumonia in BALB/c mice and on pulmonary neutrophil function. They found that mortality of *P. aeruginosa* pneumonia was increased in 10% CO₂-exposed compared to air-exposed mice. Hypercapnia increased pneumonia mortality similarly in mice with acute and chronic respiratory acidosis, indicating an effect unrelated to the degree of acidosis.

Furthermore, Javaheri and Dempsey (2013). Studied the central sleep apnea, (Central apneas occur commonly in high-altitude sojourn, disrupt sleep, and cause desaturation. Central sleep apnea also occurs in number of disorders across all age groups and both genders. Common causes of central sleep apnea in adults are congestive heart failure and chronic use of opioids to treat pain). They concluded that the mechanisms of central sleep apnea have been best studied in congestive heart failure and hypoxic conditions when there is increased CO₂ sensitivity.

In a different study, Vengust (2012) reviewed both the beneficial and adverse effects of permissive hypercapnic respiratory acidosis in critically ill newborn foals. He reported that partial carbon dioxide pressure (PCO₂) above the traditional safe range (hypercapnia), has beneficial effects on the physiology of the respiratory, cardiovascular, and nervous system in neonates. Even though adverse effects of hypercapnia have been reported, especially in patients with central nervous system pathology and/or chronic infection, critical care clinicians often artificially increase PCO₂ to take advantage of its positive effects on compromised neonate tissues. In the same sequence, Bleul and Götz (2013) studied the effect of lactic acidosis on the generation and compensation of mixed respiratory-metabolic acidosis in neonatal calves. They concluded that L-lactate is a more important factor in the pathogenesis of acidosis than pCO₂, and that the duration of metabolic acidosis exceeds that of respiratory acidosis in perinatal asphyxia of calves. Recent study (Tachibana et al. 2013) investigated the effect of neonatal

hypoxic hypercapnia on later functions in the hippocampus, which is a structure that has been implicated in many learning and memory processes. They concluded that Neonatal exposure to high concentration of carbon dioxide produced persistent learning deficits with impaired hippocampal synaptic plasticity. In a separate approach, Ohmori et al., (2013) investigated the anticonvulsant effect of carbon dioxide (CO₂) on Scn1a mutation-related febrile seizures. They reported that the Scn1a mutant rats demonstrated a higher hyperthermia-induced seizures susceptibility associated with respiratory alkalosis than the wild-type rats and the inhalation of 10% CO₂ demonstrated an elevated pCO₂ level and respiratory acidosis and fast-acting anticonvulsant effect against hyperthermia-induced seizures. In the same context, Noori et al., (2013) investigated the effect of pH on cardiac function and systemic vascular resistance in preterm infants. They found a weak negative linear relationship between pH and left ventricular output and a positive linear relationship between pH and systemic vascular resistance only during the post-transitional period. These relationships were maintained after adjustment for the degree of base deficit. Arterial CO₂ had effects similar to pH on myocardial function.

In a different study, Vadász et al., (2008) studied the mechanisms regulating CO₂-induced Na,K-ATPase endocytosis in alveolar epithelial cells and alveolar epithelial dysfunction in rats. They revealed that elevated CO₂ levels caused a rapid activation of AMP-activated protein kinase (AMPK) in alveolar epithelial cells, a key regulator of metabolic homeostasis. Furthermore, they provide evidence that elevated CO₂ levels are sensed by alveolar epithelial cells and that AMP-activated protein kinase mediates CO₂-induced Na,K-ATPase endocytosis and alveolar epithelial dysfunction. In a similar study,

Briva et al., (2007) examined the effects of increased pCO₂ on the epithelial Na,K-ATPase a major contributor to alveolar fluid reabsorption which is a marker of alveolar epithelial function. They found that short-term increases in pCO₂ impaired alveolar fluid reabsorption in rats and alveolar epithelial cells sense and respond to high levels of CO₂, independently of extracellular and intracellular pH, by inhibiting Na,K-ATPase function, via activation of PKC ζ which phosphorylates the Na,K-ATPase, causing it to endocytose from the plasma membrane into intracellular pools. As obvious effects of elevated levels of CO₂ to induced alcohol like syndrome or home sick syndrome were not discussed beside the fact that searching the database nothing about CO₂ and alcohol like syndrome or home sick syndrome were found. This paper discusses the influence of elevated levels of atmospheric CO₂ in the breathing and rebreathing air on the creation of Alcohol like syndrome and home sick syndrome.

2. Materials and methods

We collected recent published articles from the internet in the area of carbon dioxide and its effect on human health and analyzing the data in a way that support our concept. Furthermore, the author based on some observation of those who were sleeping with head covered with blanket to keep the body warm especially in winter time. In addition, observations on feeling a sleepy in meeting rooms have no ventilation condition. Further observations were also obtained in the Palestinian refugee camps and newly building projects or housing systems.

3. Results and Discussion

3.1 ALS

The author observed more than 40 cases of deep sleeping which took several mints to get them wake up. Those were sleeping under closed conditions or having covered their head with blanket to keep themselves warm during winter time. When they waked up it appeared to the author as if they were drinking alcohol, but in fact they were not.

With an open discussion with medical doctors in the UNRWA clinics. They mentioned that many Palestinian refugee patients came to the clinic without any infectious or communicable diseases but they felt sick.

The author provided scientific explanation to the above mentioned cases. However, as everyone may know that blood contains of hemoglobin and water, under this condition the atmospheric CO₂ that inter the lung through respiration reacts with water and form a kind of carbonic acid ionized as shown in Figure 1.

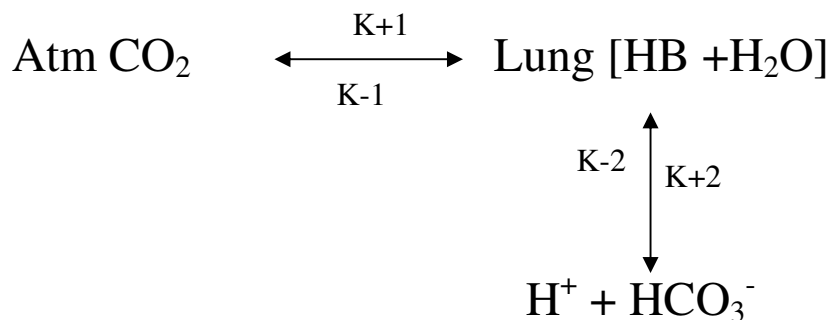


Figure 1. Interaction of atmospheric CO₂ with water in the blood in the lung during the normal respiration process. Atm CO₂ and HB, represent atmospheric CO₂ and Hemoglobin, respectively.

It is obvious from Figure 1 that one mole of CO₂ under atmospheric condition reacted with blood and produced carbonic acid which be ionized through kinetic reaction rate K+2 to produce one mole of hydrogen ion and one mole of Bicarbonate ions. These ions react again through K-2 to produce a one mole of carbonic acid which is excreted to the atmosphere as CO₂ through expiration process. This reaction occurs in the normal respiration process under open atmosphere. It is clear that one mole of hydrogen ion is produced at normal condition (Figure 1). This reaction is responsible of maintaining the blood pH in the range of 7.35-7.45. This acid-base reaction keeps the blood pH under buffered conditions.

Under closed rooms, concentrations of CO₂ tend to increase and O₂ tend to decrease in the close atmosphere due to the accumulation of CO₂ as a byproducts in the biochemical reactions and consumption of O₂ (Arthurs and Sudhakar 2005). Under excess CO₂ and reduced O₂ concentrations (Concentration above the atmospheric CO₂) such as in closed rooms, lecture halls, sleeping under closed condition like covering the head with towels or blanket specially in winter time to get warming up the body, especially in poor counties where population used multi-clothes, the reaction in Figure 1 goes further as shown in Figure 2.

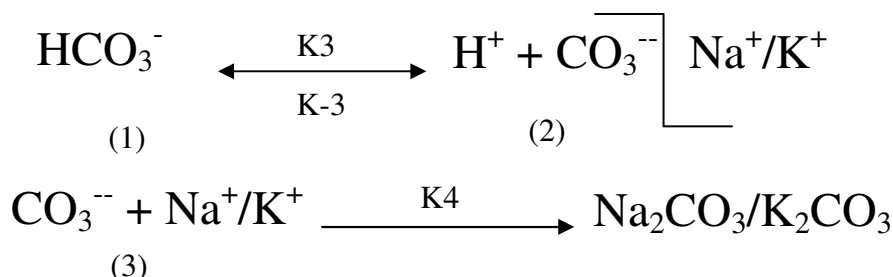


Figure 2. Ionization of bicarbonate ion under excess of CO₂ in respiration air.

It is obvious from Figure 2, that increasing CO₂ in the respiration air may result in further ionization of bicarbonate ion to produce one mole of hydrogen ion and one mole of carbonate ion, reaction 1 in Figure 2, kinetic reaction K3. As concentration of CO₂ is high in the respiration air, and/or in the rebreathing air, K3 is consequently high. Under this condition two moles of hydrogen ions are produced as shown in Figures 1-2. This may result in a sharp drop in the blood pH, consequently a sever blood acidosis may occur. This explanation is also supported by the results of Noori et al., (2013) who investigated the effect of pH on cardiac function and systemic vascular resistance in preterm infants and found that arterial CO₂ had effects similar to pH on myocardial function.

This acidosis may have negative effects on the blood functions or enzyme activity. This suggestion is also supported by the result of Briva et al., (2007) who examined the effects of increased pCO₂ on the epithelial Na,K-ATPase activity as a major contributor to alveolar fluid reabsorption which is a marker of alveolar epithelial function and found a short-term increases in pCO₂ impaired alveolar fluid reabsorption in rats.

Under this condition (high acidity) a healthy body starts a self recovery of this condition by transferring sodium/potassium from the central nervous system (CNC) or from other source in the body to react with carbonate ion as in reaction 3 Figure 2 to produce sodium/potassium carbonate to be excreted outside the body.

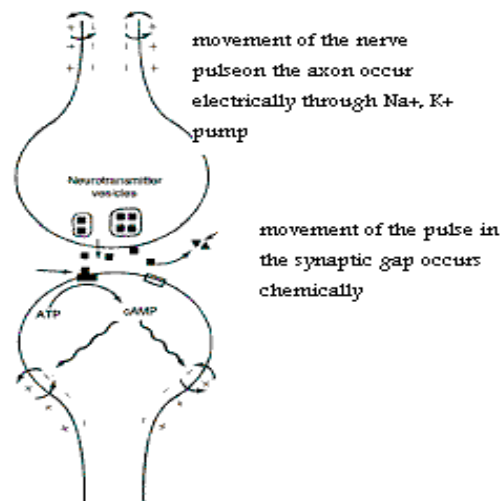


Figure 3. Movement of nerve pulse on the axon through Na⁺/K⁺ pump. Adopted from Williams et al 2000

As shown in Figure 3, the Na⁺/K⁺ ions have an important role in the central nervous system (CNS) as they are responsible for movement of nerve pulse on the axon electrically through Na⁺/K⁺ pumps and Na⁺/K⁺-ATPase.

As a result of transferring Na⁺/K⁺ ions from CNS or from other source to react with CO₃²⁻ (Figure 2) a loss or a delay of transferring nerve pulse would occur. Consequently, this would partially result in losing sensation. Accordingly, he/she would not positively respond to the external stimulants. This effect can be referred to as Alcohol Like Syndrome (ALS). As the value of K_a in Figure 2 is high as the ALS is very strong and needs longer time to be recovered. In the way around, the increased acidity, as a result of both reactions in Figures 1 and 2, may partially or totally inhibit the activity of Na⁺/K⁺ ATPase which is responsible in nerve pulse movement in the CNS. Accordingly a partial loss of sensation may occur and ALS would appear. Our suggestion is supported with the results of Briva et al., (2007) who found that short-term increases in pCO₂ impaired alveolar fluid reabsorption in rats by inhibiting Na,K-ATPase function.

This phenomenon, ALS, would be recovered if the respiration air comes back to the normal atmospheric concentration of CO₂ as in Figure 1. It is recommended to have open air condition to avoid occurrence of ALS or to get recovered from its effects.

3.2 HSS

In poor countries, in refugee camps and in unplanned cities, the buildings are much closed to each other without urban planning. This compact housing may result in losing aeration, sun exposure and accumulation of humidity and toxic gases such as CO, CO₂, NO₂ and SO₂ inside the building. Due to high population density, accumulation of high humidity may occur inside houses, especially those who are in the coastal zone. Due to the bad wastewater treatment in houses, some leaches may occur, accordingly hydrogen sulfide (H₂S) and nitrogen oxides (NO) may accumulate inside houses due to the above mentioned conditions. Under this condition, the atmospheric CO₂ and CO₂ from the expiration, NO₂, and H₂S may react with the humidity inside houses and produce a mixture of acidic humidity (Carbonic acid, nitric and hydro sulfurous acid in the humidity). Our suggestion is supported with the results from different countries and cases, for instance Norbäck et al., (2013) studied the effects of a CO(2) demand-controlled ventilation system in computer classrooms on perceived air quality and sick building syndrome and concluded that Use of a CO(2)-controlled ventilation system, reducing elevated levels of CO(2), may slightly reduce headache and tiredness and improve perceived air quality. Moreover, Sahlberg et al. (2012) examined the associations between biomarkers of allergy and inflammation, indoor environment in dwellings, and incidence and remission of symptoms included in the sick building syndrome and changes in the home environment. They concluded that the association between the

incidence of sick building syndrome symptoms and clinical biomarkers of allergy and inflammation suggests a common etiology between inflammatory diseases, including asthma, rhinitis, and sick building syndrome. Furthermore, Kanazawa et al., (2010) evaluated the associations between residential factors and sick house syndrome in a cold region and in a temperate region in Japan and concluded that the dampness state was associated with sick house syndrome and attributed it in the ventilation method In both groups. In addition, Sahlberg et al., (2009, 2010) investigated changes in sick building syndrome symptoms in follow-up period and in seasonal or regional variation and concluded that dampness in the dwelling is a risk factor for new onset of sick building syndrome.

It has also been reported that a high sick building syndrome score is observed in these 'urban poor' households because of inadequate ventilation and women and children indoors are most vulnerable to respiratory problems compared to other sexes (Kulshreshtha et al., 2008).

It was previously emphasized that changes in building industry during last 30 years ended in creating airtight and energy-saving constructions with reduced ventilation that resulted in accumulation of various chemicals such as CO₂, CO, NO₂, NH₃, and nicotine smoke (Bogacka 2002).

As discussed in the above mentioned reports that CO₂ and other gases are important elements in the indoor environment and sick building syndrome. However, those gases may react with the blood in the lung through breathing and rebreathing inside houses and produced various acids that may be ionized in the lung and produced severe blood acidosis and made the harmful effects mentioned above.

These acids may be ionized to produce 3 moles of hydrogen ions. A severe acidosis in the lung. Moreover, the acidic humidity that enter the lung through respiration air may result in a sharp drop in the blood pH in the lung and consequently a severe inhibition of Na⁺/K⁺-ATPase and or respiration enzymes (cytochrom-oxidase) may occur in the lung consequently a severe reduction of oxygen uptake and CO₂ exchange may occur. This explanation can be supported by the results of Briva et al., (2007) who found an inhibition of Na⁺/K⁺-ATPase as a result of elevated CO₂ in the rebreathing air.

This effect can be referred to as Home Sick Syndrome (HSS) in Gaza Palestine because homes are small and occupied by the same family. However, women and children may be the endangered generation because they spent large fraction of their time inside homes. However, they may go the nearest clinic for medical treatments and receive pharmaceuticals but they can realize later that it is useless to get medical treatment. The real treatment would be improving the air ventilation sun exposure or changing the living conditions of the house.

4. Concluding remarks

The rationale of this work comes from the real observation of the author and scientific analysis of the phenomena. ALS becomes clear that it is a result of losing sensation due to elevated levels of carbon dioxide in the lung and its consequent interactions with Na⁺/K⁺-ATPase or blood acidity. ALS can be recovered immediately after a period of time exposure to normal atmospheric air. HSS emerged from the bad environmental conditions at home, it has no medical treatment, but its effect may be medically treated but in general treatments should accompany with improving the environmental conditions at home.

5. Acknowledgement

Dr. El-Nahhal acknowledges Alexander von Humboldt Stiftung/ Foundation Fellowship Grant no IV-PAL/1104842 STP, Germany

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