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WAR OF CHEMICAL AGENTS: CHOKING GASES

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ABSTRACT

Choking gases are chemical warfare agents produced in large quantities that affect the person mainly on the respiratory system, are easily obtained by terrorist groups due to their use in industry, and are very difficult to control. Choking chemical warfare agents, which have been used since the First World War and caused heavy casualties, unfortunately still exist today and continue to be used as a tool of war. In CBRN incidents, all employees working in the pre-hospital area should know what the main choking gases are, their physical and chemical properties, and their toxicity mechanisms; It will help alleviate the effects of these gases, the use of which we cannot prevent. Although there is no antidote for choking gases, the use of corticoids is recommended. Treatment includes respiratory support with bronchodilators, high-dose corticosteroids, and prophylactic antibiotics. Treatment is applied based on symptoms. In case of exposure to choking gases, new treatment methods and antidote studies should be developed.

Keywords: *Chemical Warfare Agents, Choking Gases, Toxicity*

INTRODUCTION

In the historical context, particularly during the First World War, the extensive use of choking gases resulted in the deaths and injuries of numerous individuals, unfortunately continuing to be used today. The lack of knowledge among affected civillian

populations about what to do in case of exposure exacerbates the impact and therefore imposes significant responsibilities on all pre-hospital healthcare workers. This study aims to describe the basic characteristics, mechanisms of action and decontamination properties of choking gases, in order to ensure that intervention against these gases is carried out as quickly and effectively as possible.

SUFFOCATING GASES

Readily available, difficult to control and produced in large quantites, choking gases are known for their effects on the respiratory system (Corkins et al., 2013). Among gases that affect the respiratory system are ammonia, acid or alkali vapors, etc. Which are classified as choking gases (gases causing asphyxia and anoxia/pulmonary gases/lung irritants) (Sanghera et al., 2006). However, the main choking gases include:

• Chlorine (Cl)
• Phosgene (CG)
• Chloropicrin(PS)
• Diphosgene (DP)

Choking gases have been used in various wars throughout history and continue to be significant chemical warfare agents today, as well as being used in the industrial sector (Pitschmann & Hon, 2023).

Table 1. Boğucu Gazların Tarihte Kullanımı

Used Asphyxiant Gas	**Date**	**Place**	**Estimated Number Of People Affected**
Chlorine (Cl) Phosgene (CG)	1915-1918	Europe Middle East	More than 1.3 million people
Chlorine (Cl)	1923-1926	Morocco	Unknown
Chlorine (Cl)	1936-1940	Ethiopia	50.000-150.000 people
Chlorine (Cl)	1937-1945	Manchuria	More than 80.000 people
Phosgene (CG)	1963-1975	Yemen	>14.000
Chlorine (Cl)	2013- 2015	Syria	Unknown

Chlorine (Cl)

Chlorine was discovered by Swedish chemist Carl Wilhelm Scheele in 1774. It is a sharp – smelling, greenish-yellow gas that is easily detectable due to its odor threshold being below the toxic level. Chlorine is heavier than air. At concentrations in humans, irritation of the eyes, mouth and mucous membranes occurs at 1-3 ppm, the onset of pulmonary symptoms at 15-430 ppm, and concentrations of 430 ppm and above are lethal within 30 minutes. During World War I, it was used by the German army in Ypres, causing the deaths of 5000 soldiers. It has also been used in Iraq in 2006-2007. Additionally, it is encountered in industrial accidents (Allegaert & van den Anker, 2015; Ekvall & Ekvall, 2005).

Phosgene (CG)

Phosgene was synthesized by British chemist John Davy in 1812. It is obtained by exposing a mixture of carbon monoxide and chlorine to sunlight. Its name comes from the Greek words ‘’phos’’ (light) and ‘’gen’’ (born), hence ‘’phosgene’’ (Kleinman et al., 2004). The structural properties of phosgene include a molecular formula of $COCl_2$ a molecular weight of 98.92 g/mol, a boiling point of 8.2 °C, an odor threshold of 0.4 to 1.5 ppm and an irritation threshold of 3 ppm. In humans, concentrations of phosgene at 0.125-1.5 ppm result in odor perception, 1.5-3 ppm in odor recognition and concentrations above 3ppm cause irritation of the eyes, nose,throat and bronchi (Petersson et al., 2009).

During World War I, it was used by the German armies because it was more lethal than chlorine gas, causing the deaths of 120 people and injuring 1069. It was also used during the Yemen War from 1962 to 1970. Phosgene has a moldy and hay-like odor and is relatively easy to produce. It is heavier than air. While it affects the respiratory system, causing pulmonary edema and asphyxiation, its effects are not immediately apparent (up to 24 hours). Besides being a warfare agent, it is widely used in the industrial sector, with approximately 1 ton produced annually. Its preference for terrorist purposes lies in its ease of production and difficulty in treatment (Veiby et al., 2013;Williams et al., 2024). Phosgene is also known by various other names such as carbonyl dichloride, carbonic dichloride, carbonyl chloride, carbonyl dichloride and chloroformyl chloride(Ty et al., 2023)

Phosgene exposure routes to the gas:

• **Respiratory:** Inhalation is the primary route of exposure to phosgene. Olfactory exposure may provide inadequate warning. Even at low concentrations, exposure can

manifest its effects. Its irritant effect can be mild and delayed. Phosgene is heavier than air. Unventilated, low or enclosed spaces can lead to severe asphyxiations.

• **Skin/eye contact:** Phosgene gas can have an effect when it comes into contact with moist or wet skin or eyes.

• **Digestion:** It is not possible to ingest phosgene (Ayşegül & Avci, 2011).

Table 2. Phosgene Dose-Response Relationship (Ayşegül & Avci, 2011; Costi et al., 2022)

Phosgen Inhalation Dose	**Pulmonary effect**
<50 ppm-min	No clinical pulmonary effect
50 - 150 ppm-min	Subclinical pulmonary reactions. Edema not likely
300-150 ppm-min.	Pulmonary edema probability
300 ppm ve over -min.	Life-threatening pulmonary edema

Substances that react dangerously chemically with phosgene:

• t-Butyl azidoformate

• 2,4-Hexadiyn-1,6-diol

• Aluminum

• Alcohols

• Isopropyl alcohol

• Secondary amines

• Potassium

Chloropicrin (PS)

When inhaled, chloropicrin, which is highly toxic, was used alongside phosgene by the German armies against Italien soldiers in 1917. Chloropicrin serves as a mediator of toxicity between chlorine and phosgene. At lethal concentrations, chlorine causes injury to the upper parts of the respiratory tract, specifically the trachea and bronchi, while phosgenes affect the alveoli. Chloropicrin causes more damage (Pesonen & Vähäkangas, 2020).

Diphosgen (DP)

Diphosgen was initially developed as a pulmonary agent for chemical warfare, just a few months after the first use of phosgene. It was employed as a toxic gas by Germany during World War I. A significant factor in the development of diphosgen was its ability to easily penetrate the gas mask filters in use at the time.(Arpaci et al., 2018).

PHYSICAL PROPERTIES AND TOXICITY MECHANISMS OF CHOKING GASES

Table 3. The Physical Properties of Choking Gases (Costi et al., 2022)

Gases	Chlorine (Cl)	Phosgen (CG)	Diphosgen (DP)
Molecular Weight	70.9	98.92	197.85
Vapor Density	2.5	3.4	6.8
Liquid Density	1.393	1.381	1.65
Melting Point (°C)	-101	-128	-57
Boiling Point (°C)	-34	7.6	128
Vapor Pressure (mmHg, 25°C)	5.168	1.118	4.2
LCt50 (Respiration, mg-min/m3)	-	3.200	3.000-3.2000

Choking gases, when they come into contact with moist tissues such as the eyes, throat and lungs form hydrochloric and hypochlorous acid. Hypochlorous acid converts to hydrochloric acid, generating new oxygen in the process. Both hydrochloric acid and the newly formed oxygen damage lung tissue. This leads to an inflammatory response, causing damage to the alveolar-capillary membrane of the lungs and resulting in pulmonary edema (Heidari et al., 2019).

CLINICAL DESCRIPTION OF CHOKING GASES

The clinical effects of choking gases are described as consisting of an initial protective phase, followed by a symptom-free latent period and finally a terminal phase characterized by pulmonary edema:

• **Biopreservative phase:** During the initial stage of exposure, at high concentrations (> 3 ppm), individuals experience frequent and shallow breathing, decreased respiratory capacity and volume. An increase in arterial CO2 pressure and a decrease in blood Ph are observed. After exposure ceases, there is a tendency for the reflex syndrome to regress. Symptoms in this initial phase typically include shortness of breath, wheezing and coughing along with pain in the eyes and throat, chest tightness; hypotension, bradycardia and rarely sinus arrhythmias may occur.

• **Clinical latent phase:** Following exposure, the second stage, which typically lasts for several hours, is characterized by relatively mild clinical signs and symptoms. However,

histological examination reveals an increasing presence of pulmonary interstitial and alveolar edema on a microscopic level. This marks the beginning of swelling due to fluid accumulation. Damage to alveolar type I cells and an increase in hematocrit can occur. Individuals exposed to the gas are often unaware of these processes. Therefore, this phase is termed the ‘’Clinical Latent Phase’’. The duration of this phase varies inversely with the inhaled dose

• **Terminal phase:** At this stage, the toxicity of the choking gas leads to the accumulation of fluid in the lungs, resulting the pulmonary edema. Respiratory failure is indicated by tachypnea, dyspnea, tachycardia, cyanosis and decreased PO2. The severity of edema potentially increases, with fluid initially accumulating in the alveoli and gradually progressing towards the proximal parts of the respiratory tract. Severe edema can lead to an increase in blood hemoglobin concentration and occlusion of alveolar capillaries. Pathological findings include extensive degenerative changes in the trachea, bronchi, bronchiolar epithelium and focal pneumonia with hemorrhagic edema (Tripodi et al., 2023).

Table 4. The Short And Long-Term Effects of Choking Agent Exposure (Veiby et al., 2013)

Agents	**Thoracic**	**Dermatological**	**Cardiovaskular**	**Ophthalmological**
Short term effects	-Chest tightness -Cough -Grunt -Suffocation	-Dermatitis -Color change -Bubbles -Burning sensation -Chemical burns	-Bradycardia -Hypotension	-Blurred vision -Burning sensation -Excessive tearing
Long term effects	-Respiratory Failure -Chronic bronchitis -Emphysema	-Cyanosis	-Arrhythmia -Congestive heart failure	-Blindness -Glaucoma

Triage

In situations involving the use of choking gases, much like CBRN incidents, there will be a need for effective intervention with a large number of casualties and limited personnel and resources available. In such a scenario, appropriate triage is crucial to effectively utilize the existing capacity and potentially save many more lives. In cases of mass exposure to choking gases, the following guidelines should be followed: Asymptomatic patients, who experience only mild symptoms such as slight burning sensation in the nose, throat, eyes and respiratory tract (accompanied by mild cough) and whose symptoms are expected to resolve within 1 hour or less, should be considered the last priority for invention. However, if symptoms appear or reoccur,triage should be reassessed. If the number of individuals exposed to choking gases is low and includes small children, infants or individuals in high-risk groups, these individuals should be monitored in the emergency department for 6-12 hours. Patient experiencing persistent shortness of breath, severe cough or chest tightness should be admitted to the hospital and observed until symptoms resolve. Lung damage due to exposure to choking gases can progress over several hours. Therefore, if the responder suspects significant exposure to chlorine gas despite the patient's benign clinical appearance, admission for observation is warranted. Clinical symptoms of lung edema typically appear 2-4 hours after moderate exposure and 30-60 minutes after severe exposure.(Papoutsakis et al., 2013).

Decontamination

Decontamination is crucial for all individuals exposed to chemical agents. If the skin is suspected to have been exposed to liquid chemical agents, immediate disinfection should occur within 1 minute. Incidents involving chemical warfare agents have shown that time is of the essence in preventing injuries and deaths through decontamination. The importance of the decontamination tools used is less significant compared to time. Various tools such as talcum powder, soap, water or specialized decontamination kits can yield good results. Individuals exposed to choking gas but without any skin or eye irritation do not require decontamination. However, individuals exposed to choking gas may still pose a risk of secondary contamination and therefore personnel interving should wear Level A protective clothing to prevent contamination through contaminated clothing and skin. (Mcloud & Papoutsakis, 2013).

In the even of exposure to choking gases, the steps for decontamination are as follows:

• The decontamination team, equipped with appropriate protective clothing for the type

and level of chemicalagent used, sets up the decontamination system in a warm area.

• Injured individuals coming from the hot zone (contaminated area) are immediately removed and brought to the decontamination unit.

• The decontamination unit consists of two main sections. The first section is where injured individuals from the hot zone are accepted and the second section is where decontamination is completed and individuals transition to the cold zone.

• All injured individuals arriving at the decontamination unit are assumed to be contaminated and decontamination procedures begin.

• Clothing and accessories of individuals exposed to the chemical agent are removed and placed in sealed bag.

• If injured individuals wear contact lenses, they should be removed immediately and eyes should be washed with water or saline solution for at least 15 minutes.

• Decontamination begins with %0.5'lik hypochlorite solution or decontamination powders.

• Injured individuals are promprly moved to the second section and transported to hospitals located at least 2 km away.

• Upon arrival at the hospitals, individuals undergo measurement using CAM devices to check for contamination. If the results are satisfactory, patients are placed in designated areas within the hospital. If the results indicate contamination patients are taken to the hospital's decontamination unit for further decontamination. (Bacharier et al., 2008).

Table 5. Basic Isolation And Protection Areas (Hughes, 2017)

Chemical agent	**Minor Leakage**			**Major Leakage**		
	Isolation (m)	**Protection**		**Isolation (m)**	**Protection**	
		Daytime (km)	**Night (km)**		**Daytime (km)**	**Night (km)**
Phosgene	150	0.8	3.2	1.000	7.5	11.0+
Diphosgene	30	0.2	0.7	200	1.0	2.4

Minor Leakage: A minor leakage originating from a single small source (such as a barrel containing approximately 208 liters) or a small tube or a small amount of leakage from a large package.

Major Leakage: A significant leakage originating from a single large package or

intense leakage occuring simultaneously from multiple small packages.

Primary Isolation Area: The area downwind from the incident where exposure to a substantial quantity of the chemical agent and potential life-threatening situations may occur.

Protection Area: The area downwind where affected individuals have not yet been adequately protected and serious health effects could occur.

Emergency Room Care

The general approach to exposure to choking gases includes physical examination, assessment of vital signs, saturation, and arterial blood gases. Pre-hospital interventions encompass eye lavage as soon as possible in case of eye contamination, application of humidified oxygen, and administration of inhalative bronchodilators. In patients with retrosternal pain, an EKG, cardiac enzymes, and complere blood count should be performed. Routine chest X-ray is not recommended unless pulmonary edema is suspected. Inhalation of sodium bicarbonate after chlorine gas inhalation is among the debated topics. Depending on the extent of respiratory damage, patients may require airway management such as mechanical ventilation with continuous positive airway pressure, intubation/tracheotomy. Prophylactic antibiotics are generally not recommended. Extracorporeal membrane oxygenation (ECMO) may be considered in severe ARDS (Cook & Blinman, 2010). Interventions conducted at the scene exposure to choking gases will continue in the emergency departments of hospitals. Key considerations in the emergency department approach to choking gases include:

• Phosgene is a potent pulmonary irritant. Symptoms can be delayed up to 24 hours due to slow hydrolysis in the alveoli. Symptoms may even occur hours after severe toxic exposure.

• There is no specific antidote. Treatment is symptomatic and supportive. Treatment includes bronchodilators, high-dose corticosteroids, and respiraty support with prophylactic antibiotics.

• Intravenous administration of 1,0 mg methylprednisolone (or equivalent corticosteroid dose) is recommended. There is no evidence-based information on the use of corticosteroids.

• Oxygen support (preferably humidified) should be provided.

• Tracheal intubation or an alternative airway method may be used in cases of respiratory failure.

• Contact of choking gases with the skin can cause chemical burns. Adequate fluid

resuscitation, analgesics, maintenance of body temperature, and covering burns with a sterile dressing are necessary. Consultation may be sought in cases of severe thermal burns to the eyes.

• Heavy physical exertion should be avoided for 24 hours.

• Avoidance of exposure to cigarette smoke for 72 hours is recommended. (Bacharier et al., 2008)

CONCLUSION

Chemical choking warfare agents, known for their use in wars and industrial areas, have particularly affected numerous lives and led to significant losses by primarily affecting the respiratory system. Among these gases, chlorine, phosgene, chloropicrin and diphosgene gases have been the most preferred and have been used to affect masses. The general toxicity mechanism of choking gases is to damage the alveolar-capillary membrane of the lungs, leading to pulmonary edema. Understanding the dose-response relationship of different choking gases, which exhibit different effects at different concentrations, will guide us in determining the level of impact based on the doses measured in pre-hospital settings and will be effective in determining our intervention methods. Exposure to high concentrations of these gases may result in aprotective phase known as the initial phase, a symptomless latent period, and a terminal phase characterized by pulmonary edema. Decontamination is the most effective and crucial step in the intervention precess. Upon reaching the affected area, teams should promptly initiate decontaminaton procedures, determining isolation and protection areas according to the type of agent. After the initial intervention by pre-hospital units, intervention will continue in the emergencydepartment for transported individuals. Although there is no antidot efor choking gases, corticosteroid use is recommended. Treatment includes bronchodilators, high-dose corticosteroids, and prophylactic antibiotics for respiratory support. Treatment is symptom-based. New treatment methods and antidote studies should be developed for exposure to choking gases.

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