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Investigation of Chlorine Gas, Phosgene Gas, and Mustard Gas: Development, Use, Effects, and Treatments with a Focus on World War I

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Abstract

The German attack in Ypres on April 22, 1915 ushered in a new era of warfare. For the first time in military history, large amounts of weaponized gas were being deployed. The first attack utilized chlorine gas while subsequent attacks used phosgene gas, mustard gas, or a combination of them. Scientific and medical communities scrambled to combat the new threat, leading to great advancements in the understanding of the pathophysiology and treatment relating to chemical gas attacks. New ways to bypass chlorine gas, phosgene gas, and mustard gas effects were proposed and tested and already existing medical interventions proved useful. Venesection and alcohol became part of the new protocol for treatment of gas victims. Sodium bicarbonate, subcutaneous oxygen injections, cocaine, and adrenaline were among the preexisting methods incorporated into the new protocol as they were being used to treat the exact same symptoms rooted in other causes.

INTRODUCTION

On the afternoon of April 22, 1915, more than 150 tons of chlorine gas were released from the German trenches, creating a dense cloud of asphyxiating gas along the northern arc of the Ypres salient. Within minutes, more than 1,000 French and Algerian soldiers were dead and another 4,000 were injured. Shortly after, the Germans released weaponized gas on Canadian troops near Saint-Julien. The use of such a large-scale gas attack in World War I sparked great outrage among Western leaders, particularly those on the Allied side, who considered its use inhumane and in violation of the rules of war. Although irritant vapors from liquid ethyl bromo-acetate were used in war before by the French as early as 1914 in what were called "cartouches suffocantes," the chlorine gas attack on April 22, 1915 marked a great escalation of the use of chemicals in warfare (1). The attack on Allied troops at Ypres was notable since it was the first time a gas as toxic and debilitating as chlorine was used in combat. Also, the shear amount of gas successfully released had not been achieved previously and was the main reason why the attack was so effective as an agent of war, leading the many horrific injuries and death (1).

The development of chlorine gas, phosgene gas, and mustard gas in the immediate years before and during World War I, and their initial deployment in the years 1915-1917 during the war, depended on academic and industrial scientists who increasingly served the military needs of the state (2). Germany's chemical program, the world leader in academic and industrial chemistry at the time, was led by Fritz Haber and based at the Kaiser Wilhelm Institute in Berlin. Haber is responsible for the development of ammonia by discovering a process, now known as the Haber-Bosch process, to convert atmospheric nitrogen to fertilizer. Without Haber, the world would not have enough food to support the billions of people currently on Earth. Haber also, however, developed and championed chemical warfare. He not only developed it, he insisted on

its use, saying “To win the war, wage chemical warfare with conviction” (3). Haber was on the front lines in Belgium directing the first use of chlorine gas in World War I. He returned to Germany feeling vindicated for his successes (3).

Other nation’s followed Germany’s chemical advances in turning industrial substances into weapons used by the military. Great Britain opened thirty-three laboratories to test 150,000 compounds as being suitable for chemical warfare between 1914 and 1918. The United States organized the National Research Council, a group of scientists, to work on offensive and defensive aspects of chemical war. In 1918, the United States Army integrated the National Research Council into its new Chemical Warfare Service. Other countries followed suit with similar organizations. Observers refer to World War I as “the chemist’s” war” (4). By the end of the war in 1918, the use of chemical weapons such as chlorine, phosgene, and mustard gas accounted for more than 1.3 million casualties and 90,000 deaths. More than 124,000 tons of gas were produced by the end of the war (2). None of these gases had been weaponized or used in war until the German Empire realized their potential as a debilitating agent to gain an advantage in trench warfare. They were previously only used in industry.

Carl Wilhelm Scheele discovered chlorine in 1774 and its bleaching properties resulted in its subsequent industrial-scale production (5). Early chlorine use in the United States in the 1900’s was responsible for potable water and sewage disinfection. Not until the mid 1910’s did Haber and the Germans weaponize chlorine gas. Haber initially chose chlorine gas when developing a chemical weapon because it was a commercially available chemical that could be sourced easily and cheaply from existing suppliers. Chlorine was widely used in Germany as a bleaching agent and was involved in the production of paper, textiles, and dyestuffs. Before 1914, German companies were making 40 tons of chlorine a day (6). At the turn of the

nineteenth-century, chlorine was being produced via the electrolysis of brine, a sodium chloride (NaCl) solution. In water, NaCl disassociates to form a sodium ion and a chloride ion. Oxidation of the chloride ion occurs at the anode to produce Cl_2, or chlorine gas (7).

Chlorine gas can be cooled and pressurized to become a liquid. In liquid form, it is easier to store and transport. When liquid chlorine is exposed to air, it becomes a gas that, because it is more dense than air, stays low to the ground and spreads rapidly (8). This liquid form was how the Germans deployed chlorine gas during World War I and why predicting wind patterns was very important to a successful attack. Chlorine gas has a strong odor similar to bleach and appears yellow-green in color (8).

Chlorine becomes especially harmful to the human body when elemental chlorine reacts with water, such as the moist tissues of the eyes and respiratory tract, to produce hypochlorous and hydrochloric acid. The acids react once again to create oxygen free radicals which damages cell walls. The acids and free radicals interact with sulfhydryl groups of some amino acids and enzyme systems thus disrupting their function (9). When exposed to dangerous concentrations of chlorine, symptoms may include blurred vision, burning and blistering of the skin, nose, throat, and eyes, coughing, chest tightness, difficulty breathing, fluid in the lungs, nausea, and vomiting (8). As with most noxious gases, the effects of chlorine gas are worst when exposed in high concentrations. At 40-60 parts per million (ppm), lung injury including toxic pneumonitis may occur as well as pulmonary edema. Death occurs after 30 minutes when exposed to chlorine gas at 430 ppm and becomes fatal only after a few minutes of being exposed to 1,000 ppm (10). Unfortunately for the soldiers in Europe, chlorine gas was not the deadliest gas deployed on the battlefield. That honor belongs to phosgene gas.

Phosgene gas was first used by the Germans in World War I in 1915. Phosgene gas is specifically used for industrial purposes in the dyestuffs, organic chemical, and pharmaceutical industries. In addition, it is involved in the manufacture of metallic oxides (11). Phosgene gas is much deadlier than chlorine gas and became the Allies primarily used chemical weapon during the war. Industrially, phosgene gas is manufactured by reacting carbon monoxide, CO, with chlorine gas, Cl_2, using an activated carbon catalyst to produce $COCl_2$, or phosgene gas (12).

At room temperature phosgene is a gas but, like chlorine gas, with cooling and pressure can be turned into a liquid to be more easily stored and transported and turns back into a gas upon exposure to air (13). When in gas form, phosgene has a characteristic odor of newly mown hay. Among the effects of phosgene gas exposure are blurred vision, coughing up white to pink-colored fluid, nausea, vomiting, difficulty breathing, pulmonary edema, low blood pressure, heart failure, chronic bronchitis, and emphysema. Contact with skin may produce lesions similar to those caused by frostbite or burns (13).

Another of the major chemical innovations during the First World War was the introduction of mustard gas by the Germans in 1917. It had been known as a blistering agent for decades but had never been weaponized on such a large scale. The Germans discovered its effects from using it on prisoners. They found that the oily liquid would remain on the ground for days and the gas would lacerate the conjunctiva of the eye. Among those affected from mustard gas in World War I was Adolf Hitler (14). Like phosgene gas, mustard gas is a derivative of chlorine gas via the disulfur dichloride intermediate. Disulfur dichloride is produced by mixing chlorine gas into molten sulfur (15). Mustard gas is then produced by way of the Levinstein process which involves bubbling dry ethylene gas, C_2H_4, through the disulfur dichloride, S_2Cl_2, to produce bis(2-chloroethyl)sulfide, or $C_4H_8Cl_2S$ (16).

Exposure to mustard gas, however, is typically not fatal. Less than 5% of soldiers who were exposed to mustard gas and who received medical care during World War I died (17). Although it was less fatal, mustard gas victims required skilled nursing care to overcome its effects. Mustard gas is a powerful irritant and blistering agent that impairs the skin, eyes, and respiratory tract. It damages DNA, especially in the bone marrow, which may lead to a decreased production in blood cells called aplastic anemia or a decrease in red and white blood cells and platelets called pancytopenia (17). When exposed in excessive concentrations, mustard gas may produce extensive burning on the skin, chronic respiratory disease, blindness, and lung cancer (17).

The military needs and search for a tactical advantage of countries involved in World War I led, for the first time in history, to the widespread wartime use of gases commonly used in industry such as chlorine and phosgene as well as the chlorine-derived mustard gas. Prominent German chemists such as Fritz Haber spearheaded the German chemical program that found a way to successfully weaponize these gases and initiated their military use. After the release of more than 150 tons of chlorine gas at Ypres, both the Allies and the Central Powers scrambled to understand its effects and treatments. Primary sources from the first half of the twentieth-century will be used to analyze contemporary medical thought regarding the effects and treatment of injuries caused by chlorine gas, phosgene gas, and mustard gas. Their use, development, and production will also be discussed.

LITERATURE REVIEW

Chlorine Gas

As the Allies hurried to understand the gas the Germans were unleashing on the battlefield, reports from the media warned of the dangers of gas attacks and their effects they had on soldiers combating the German enemies in the trenches. In an article titled "The German Use of Asphyxiating Gases" published by *The British Medical Journal*, or *The BMJ*, on May 1, 1915, the effects of chlorine were documented by a correspondent observing the effects of chlorine gas in hospitals. The correspondent in the Northern French town of Boulogne, less than 80 miles west of Ypres, Belgium, described the devastating effects seen on April 28, a mere six days after the initial attack. Of those arriving at the Boulogne hospital, some were already deceased and others suffered from physical symptoms.

Around the larynx, there was a region of acute inflammation and there was great irritation of the trochlea. The kidneys presented as cyanotic, characteristic of asphyxiation brought on by acute bronchitis. In some of the more severe cases, pulmonary edema was present. The signs and symptoms were those generally seen in acute bronchopneumonia. According to the correspondent, if the gas is highly concentrated as it enters the trenches, soldiers experience burning of the eyes, violent coughing, and asphyxiation. The correspondent believed chlorine to be the culprit due to symptoms demonstrated by the hospital. The means of protection, he said, is to saturate a handkerchief with a sodium bicarbonate solution and place over the mouth and nose (18). This was a quick and easy way of protecting the respiratory tract, thus acting as a respirator. Furthermore, Sir John French described how the Germans had made use of facilities to produce large amounts of gas in an obviously deliberate attempt to create a chemical weapon. He believed this was a blatant violation of the Hague Convention (18). This article in *The British*

Medical Journal pleads for the deployment of respirators on the front lines and informs medical personal about the effects of chlorine gas exposure and how to identify it.

An article published in the *Scientific American* magazine on May 15, 1915 titled "Chlorine Gas on the Battlefield" resembled the sentiment published in *The British Medical Journal* weeks earlier regarding chlorine as the gas used in recent German offenses, including in Ypres. According to the article, the yellowish-brown cloud, distinct odor, and high density indicate chlorine gas (19). As a means of protection, the article stated that a towel or other absorbent material should be soaked with water or a basic substance such as sodium bicarbonate, or baking soda, and placed over the face. These measures may help the Allied troops overcome the "barbarous application of scientific knowledge" (19). The article, like the one in *The BMJ*, outlined the best measures of protection, including the use of a basic substance to offset the harmful effects of chlorine gas. Since chlorine gas, and all other gases, disperses and flows readily with the wind, these measures could be taken until the cloud has passed. The article, too, condemns the use of gas in war in the name of advanced military tactics.

As the Germans ushered chemical warfare into World War I, it was imperative that effective measures of protection continued to be developed to neutralize the threat of gas attacks. An article in *The BMJ* written on June 12, 1915 by Rushton Parker, J.D. Mortimer, and W.S. Syme entitled "Poisonous Gases" detailed the new suggestions themselves and a renowned scientist and chemist named Sir Henry Brunner had pertaining to the treatment of victims of chlorine gas. He stated that the inhalation of vapors from fabric immersed with alcohol is an efficacious treatment. According to Brunner, this could be done with whiskey, brandy, rum, pure alcohol, or ether (20). Brunner used his knowledge as a chemist to search for a crafty new way to alleviate the effects of chlorine gas. He considered how alcohol is highly reactive with chlorine.

The reaction, he said, would produce compounds that were less harmful and some that were even already being used in the medical community. Chloroform, and chloric ether are among the products of the reaction between alcohol and chlorine gas in the lungs (20). For Brunner, the use of chemical knowledge was a matter of using chemical advances not for the production of deadly weaponized gases, but rather for the development of a more effective treatment against them.

Rushton Parker, a professor of surgery at the University of Liverpool, pitched his idea of subcutaneous oxygen injections being a remedy for asphyxiation to a surgeon who had been on the front lines. The surgeon stated how it has already been tried and had seemed to be effective. The idea came from its use by those suffering from dyspnea, or difficult or labored breathing (20). The recognition that the effects of chlorine gas are similar to those of other medical conditions opened up a new realm of possibilities for the treatment of chlorine gas victims – subcutaneous oxygen injections being one of them.

An anesthetist at the Royal Waterloo Hospital for Children and Women by the name of J.D. Mortimer offered another solution for the respiratory troubles experienced by those exposed to chlorine gas. He suggested administering cocaine and adrenaline to alleviate the difficulty in breathing and to treat the subsequent damage caused to the larynx, trachea, and bronchi. The combination of cocaine and adrenaline act as a local anesthetic on the inflamed passageways. This method had already been used in throat clinics in the treatment of those who suffered from asthma, bronchitis, and bronchiectasis (20). Mortimer's suggestion was yet another example of the many ideas to attempt to successfully apply preexisting medical interventions to those victimized by gas attacks.

J.C. McWalter, a medical doctor from Dublin, responded to Rushton Parker's recommendation that alcohol be used as a remedy in July 15, 1915, in an article published in *The*

BMJ titled “The Prevention of Gas Poisoning.” Per Parker’s suggestions, he conducted a few experiments. He found that the inspiration of alcohol through an inhaler allowed subjects to freely breathe chlorine without the usual detrimental effects of chlorine gas inhalation (21). McWalter proved that the production of chloroform from reacting alcohol with chlorine gas in the lungs is indeed a great way to relieve and prevent harm to the respiratory tract.

While the effects that chlorine gas had on the body were observed and understood, the exact mechanism of action was not. Sir Edward Schafer, a professor of physiology at the University of Edinburgh uncovered chlorine’s deadly work in his article “On the Immediate Effects of the Inhalation of Chlorine Gas” published in *The BMJ*. He explained that chlorine must only have local effects in the lungs, since chlorine would instantly combine with the many components of blood and thus have less of an effect. Furthermore, when the bodies of those who were killed by chlorine gas are opened and examined, the muscles are responsive upon artificial nerve stimulation and the only visible effects are on the lungs (22). Schafer confirmed that chlorine is not effectively carried through the body via the blood and thus only reacts and produces harm to the tissues of the respiratory tract.

He continued his investigation and concluded that chlorine gas “may directly affect the bronchial musculature or the vascular musculature; it may stimulate the mucus-secreting mechanism of air tubes; it may influence the coagulability or viscosity of the blood within the pulmonary vessels; or it may produce reflex effects by exciting the endings of afferent nerves within the lungs” (22). In any case, he attributed the cause of death to obstruction in the pulmonary vessels, thereby impeding the ability of the blood to freely pass to the left auricle and left ventricle of the heart from the lungs (22).

In addition, Schafer examined the common belief that fatality results when chlorine induces constriction of the bronchioles. His findings suggested that obstruction of the pulmonary vessels is responsible for death, not constriction of the bronchioles, and may result in complete stasis of the respiratory vessels. Among his evidence were comparative microscopic evaluations of the lungs of animals exposed to chlorine gas. Upon examination, the animals had pulmonary capillaries that were engorged with blood and edema of the interstitial tissues of the lungs was present (22). Schafer's contributions to the understanding of chlorine's method of harm allowed researchers to have a better grasp on the properties of chlorine and aided medical personnel in the diagnosis and treatment of chlorine gas poisoning.

In a follow-up article titled "Treatment of Chlorine Gas Poisoning" published in *The BMJ* on May 27, 1916, Sir Edward Schafer suggested that extensive venesection be performed on patients presenting with chlorine gas poisoning, noting that the method had not yet been tried. Since obstruction of the pulmonary vessels causes death, venesection, removing blood from circulation, otherwise known as blood-letting, was seen by Schafer as a potential solution that could be very easily applied and produce immediate results (23). With new insight into the dangers of chlorine gas, Schafer's suggestions caught the attention of medical professionals, some of who sought to legitimize his proposed treatment.

Among those who tested Schafer's assertion was A. Stuart Hebblethwaite, a medical graduate in the Royal Army Medical Corps. He published his findings in *The BMJ* on July 22, 1916 in the article "The Treatment of Chlorine Gas Poisoning by Venesection." Hebblethwaite's writing came not even two full months after Schafer offered his hypothesis. As the German gas offensives were still ongoing, Hebblethwaite, as he wrote, "had the opportunity of treating a number of cases" (24). For those patients who were in grave condition and suffering from

cyanosis, he tried venesection. Since chlorine creates obstruction of the pulmonary circulation, cardiac resistance, or pulmonary vascular resistance, increases. Obstruction in the pulmonary vessels generates a high-pressure system which forces the right ventricle of the heart to work harder to force the blood through the system.

Venesection, according to Hebblethwaite, has two notable effects in theory. First, venesection lowers cardiac resistance proportionately to the amount of blood removed from circulation. Second, it removes the fluid that causes the obstruction which also decreases cardiac resistance and reduces strain on the heart. Practically speaking, his results suggested the following: relief of cyanosis, alleviation of pulmonary congestion, alleviation of severe headaches, and increased sleepiness (24). He found that exactly when the venesection was performed proved important. In ten cases of cyanotic patients, five received venesection with positive outcomes, two died, and three received venesection later on and did not fare as well. He concluded that the less amount of time the heart is strained with high cardiac resistance, the greater the long-term outcome for the patient (24). Whether due to venesection itself or the physical manifestations associated with venesection, Hebblethwaite proved Schafer's theory that venesection is a useful treatment for patients exposed to chlorine gas, at least among those presenting with cyanosis.

The use of chlorine as a weapon in World War I enraged leaders on the Allied side and engaged the chemical and medical communities, which worked fervently together to bring about new techniques to prevent and treat chlorine gas poisoning. Fabric immersed in the basic solution of sodium bicarbonate was quickly identified as an effective first line of defense against the chlorine gas attack. Sir Henry Brunner and J.C. McWalter used their advanced knowledge in chemistry to establish alcohol as a remedy for those implicated in a chlorine attack. Now, there

was not only a way to prevent inhalation of chlorine gas but also a way to neutralize the harmful effects of chlorine gas once exposed to the respiratory tract. Rushton Parker and J.D. Mortimer furthered the search for medical interventions to bypass the effects of chlorine and pioneered the use of existing treatments on gas victims by looking at what was already being used to treat almost the exact same conditions rooted in other causes. These included subcutaneous oxygen injections to supply oxygen to the tissues as well as administration of cocaine and adrenaline to ease difficulty in breathing by acting as a local anesthetic on the inflamed passageways. Sir Edward Schafer made large strides in the understanding of how chlorine works in the body and was the first to propose venesection, a treatment which was proved effective by Hebblethwaite. By the end of the war, chlorine gas was nowhere near as potent as a debilitating and fatal agent of war due the great advances made by the chemical and medical communities.

Phosgene Gas

Since chlorine gas was the first to be used during World War I, many of the treatments developed to protect against chlorine gas attacks were also used to combat phosgene and mustard gas attacks, both being chemically derived from elemental chlorine gas. In addition, the investigation of the effects of phosgene gas and mustard gas largely took place after the war ended in November of 1918. What made the study of the harm inflicted by phosgene gas difficult to determine was the fact that the Germans began combining multiple chemicals in one artillery shell that would be fired at the Allied troops. In an article dated August 10, 1918 in *The BMJ* called “War Gas Poisoning,” It was stated that in the attacks from April to August 1916, phosgene gas was combined with chlorine gas. In doing so, the Germans attempted to create a

severely irritable mixture of gases, thereby compelling opposing soldiers to discard their respirators and thus be exposed to deadly phosgene gas (25).

Phosgene became the Allies chemical weapon of choice as industrial production ramped up. An article in *Scientific American* entitled "United States Chemical Warfare Service – II" and dated April 12, 1919 detailed the construction of a plant for the manufacturing of phosgene gas shells at Edgewood Arsenal in Maryland. At Edgewood Arsenal, carbon monoxide and chlorine were passed through a carbon catalyzer at high temperatures to generate phosgene gas. The phosgene was then liquefied by subjecting the gas to condensers placed in refrigerated brine. The plant was responsible for filling shells and shipping containers to Allied troops in wrought-iron drums capable of holding 1,700 pounds (26). To ensure that the phosgene gas remained a liquid during production, conveyors brought the shells through rooms at zero degrees Fahrenheit and were filled in facilities that were refrigerated well below the gas' boiling point. "The filling machine … is arranged to fill six shells at a time and the filling is done within a glass-enclosed cabinet" (26). The Allies quickly utilized facilities to produce phosgene gas and shipped it to the front lines to be used against the Germans in a bid to greatly diminish the advantage the Germans had in initially being the first and only side to employ gas.

The noted effects of the gas included impairment of the alveolar epithelium, acute edema, and thrombosis of the pulmonary capillaries, the exact cause of death via chlorine as determined by Sir Edward Schafer years earlier. Of the other effects mentioned were rapid respiration, irritation of the pharynx, larynx, and bronchi, secondary bronchitis, and bronchopneumonia (25).

Later in the twentieth-century, more information released detailing the effects of phosgene gas. Since phosgene gas is important in industry, some of the breakthrough in the understanding of phosgene gas came from industrial incidents. The article "Toxic Gases in

Industry: Phosgene and Arsine" published in *The BMJ* on December 23, 1939 detailed these incidents. Phosgene is much more deadly than chlorine gas, with air containing a concentration low enough that its smell and lacrimation are barely noticed by those affected proving fatal. Since immediate symptoms are rarely noticed, those who are exposed may not even know they are being exposed, leading to more severe outcomes. Profound lung injury may occur when exposed to a concentration of only one part per thirty-thousand (11). These sources demonstrate that phosgene gas has very similar effects on the body but is deadlier when exposed to even small concentrations.

In 1941, an article called "Diagnosis and Treatment of Gas Casualties" published in *The BMJ* described the effects of phosgene gas exposure and the protocol of triage that was present at the time. The article suggested that cases be categorized as either mild, moderate, or severe. The mild cases presented with a reddish and flushed face, increased respiration, pain in the chest area, and coughing. For those categorized under mild, rest, fresh air, and nursing were the prescribed. Moderate cases experienced a larger amount of respiratory difficulties, pulmonary edema, and cyanosis. Oxygen supplementation and venesection were used in moderate cases. The most severe patients suffered from very shallow breathing, cyanosis, copious edema and frothing at the mouth, and, in some cases, pneumonia. These patients received oxygen supply, rest, and heavy surveillance (27). The use of venesection and oxygen supplementation in the treatment of phosgene gas exhibits the continuation of the use of preexisting medical thought. Sir Edward Schafer originally proposed venesection to treat chlorine gas poisoning back in 1916 and Rushton Parker introduced the idea of subcutaneous oxygen injections in preventing asphyxiation. Since the harm caused by phosgene gas strongly resembles that of chlorine gas, the same treatments used for chlorine gas proved effective against phosgene gas exposure.

Mustard Gas

Mustard gas is similar to phosgene gas in that much of what is known regarding its specific effects came later on after World War I, with some shocking delayed manifestations occurring later in life for soldiers exposed to mustard gas during the First World War. Mustard gas has some of the same effects on the body as chlorine and phosgene but is less deadly. In the article "War Gas Poisoning" from *The BMJ* on August 10, 1918, some details surrounding the use of mustard gas are discussed. The first noticeable trace comes from its characteristic odor, described in the article as that of mustard. Like the other gases used in war, mustard gas resulted in laryngitis, pharyngitis, tracheitis, bronchitis. When exposed directly to the skin, burns were experienced. More harsh symptoms appear later on and included conjunctivitis, photophobia, swelling of the eyelids, vomiting, and epigastric pain (25). Mustard gas seemed to be much more hazardous to the eyesight compared to chlorine and phosgene.

Reginald E. Bickerton, an ophthalmic surgeon at St. Dunstan's, a convalescent hospital, described the cases he was seeing regarding the residual effects of mustard gas on the eyes in his article "New Cases of War Blindness Due to Mustard Gas" published in *The BMJ* on October 27, 1934. Early conjunctivitis caused great edema of the conjunctiva and Bickerton noted that the lower fornices of the conjunctiva were very inflamed, seemingly as much as they would have been in the years around the attacks in 1917, 1918, and 1919. Delayed symptoms included partial blindness via corneal destruction signified by "thinning, ulceration, and bulging of the weakened substantia propria of the cornea" (28). Bickerton identified the severe long-term effects that mustard gas had on the eyes in soldiers who were victims of gas attacks during World War I.

Some of the information regarding the effects of mustard gas was not gathered until the World War II era closer to the mid-twentieth century. “War Gas Cases: First Aid Treatment” written in May 1942 by Alden H. Waitt in *The American Journal of Nursing* documented the effects of mustard gas exposure. Waitt wrote that as little as one part in fourteen million for one hour will cause injury to the eyes. Loss of sense of smell occurred as well as blistering, and ulcerating after direct exposure to the skin. When inhaled, the gas caused coughing and severe chest pain and sometimes resulted in nausea and vomiting. An effective measure of protection, according to Waitt, was to eliminate the gas within five minutes of exposure. Gasoline proved to be a good way to remove liquid mustard gas from the skin, as it acted as a solvent (29).

Although it was years following the Great War, the symptoms that resulted from the Germans employment of hazardous mustard gas continued to be studied and understood. Notable of the later symptoms was partial blindness. Harm to the respiratory tract and ways to prevent and treat it came largely from those used to treat chlorine gas and phosgene gas victims. The focus was to understand the effects on the eyes.

CONCLUSION

On April 22, 1915, The Allied forces watched in horror as a yellowish-brown cloud of chlorine gas advanced from the German trenches. Chemical warfare was officially in full force and the medical and scientific communities worked together to study the new weapon and devise ways to overcome the threat. As both sides launched gas attacks during the First World War, inventive as well as preexisting methods to treat the symptoms of the deadly gas were utilized. Soon after the attack in Ypres, phosgene gas and mustard gas were employed. Being chemically derived from elemental chlorine, the effects of phosgene and mustard were, for the most part,

very similar and in some instances required the same exact treatment that was developed for the remedy of chlorine gas poisoning earlier during the war. By the end of the war, the German threat and tactical advantage of a chemical attack was greatly reduced after the use of effective preventive and treatment measures and the use of chemical weapons by the Allies themselves

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