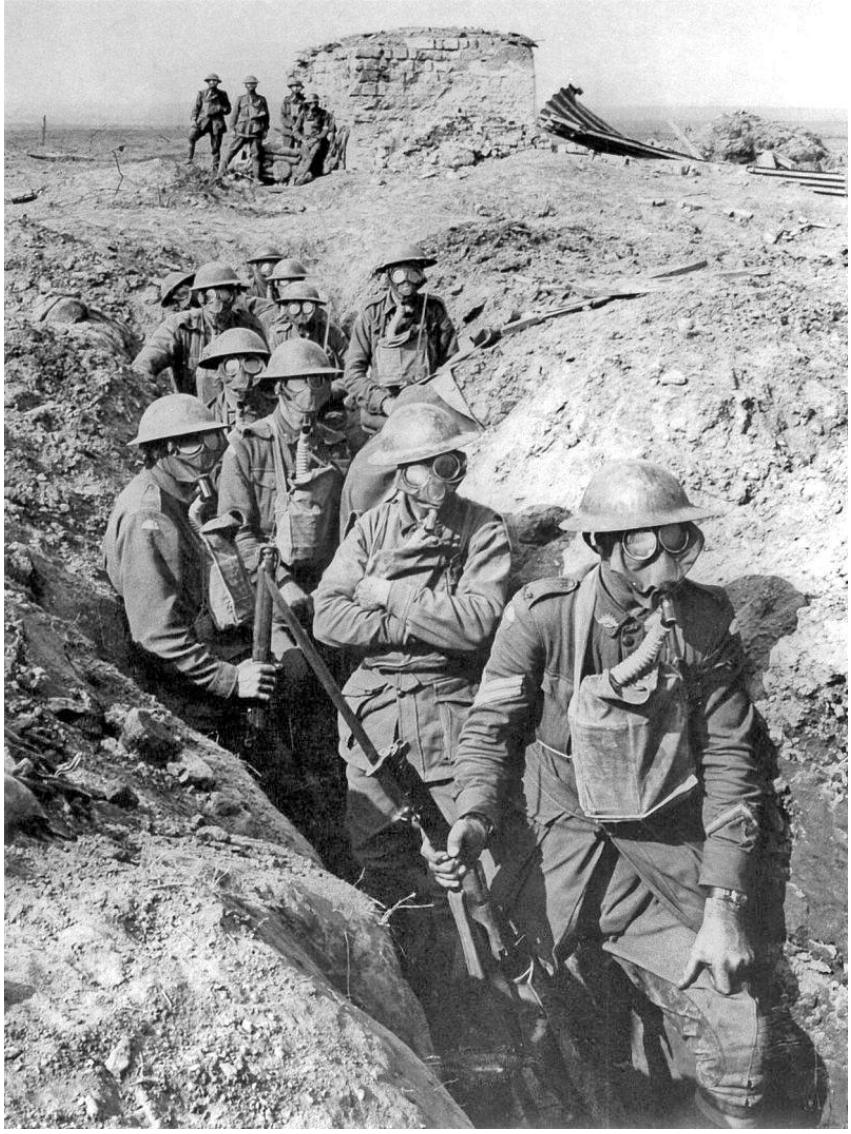
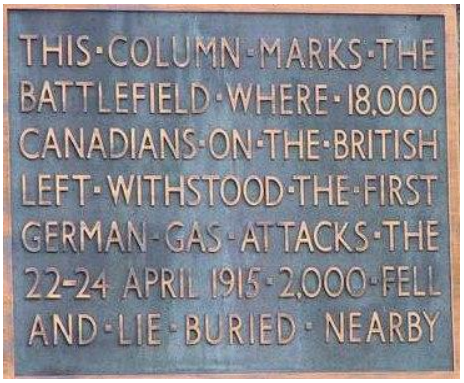
THE NORTHERN HOSPITAL

CHLORINE GAS INHALATION



"We knew there was something wrong. We started to march towards Ypres but we couldn't get past on the road with refugees coming down it. We went along the railway line to Ypres and there were people, civilians and soldiers, lying along the roadside in a terrible state. We heard them say it was gas. We didn't know what the hell "gas" was. When we got to Ypres we found a lot of Canadians lying there dead from gas the day before. Poor devils, it was a horrible sight. I was only twenty at the time and it was very traumatic. I've never forgotten it, and never will forget it".

Private W. Hay of the Royal Scots who arrived in Ypres just after the chlorine gas attacks on 22nd April 1915.



Preparing for a gas attack. Australian infantry of the 45th Battalion, 4th Division at Garter Point, Ypres sector, 27 September 1917. (Right) Plaque on the Canadian War Memorial Ypres, France

Photographer Frank Hurley

The German Army was the first to use poison gas in warfare. The gas used was Chlorine, which was unleashed onto the Allied trenches at Ypres on the 22nd April 1915. The British Army retaliated with a gas attack on the German lines later on in the same year on 25th September. The wind, however, blew the gas back into the faces of the advancing troops with fearsome consequences. This problem was solved in 1916 when gas shells were produced for use with heavy artillery. This increased the range of attack and helped to protect their own troops when weather conditions were not "ideal". By the end of the war there were an estimated 100,000 deaths on all fronts from the effects of chemical gas attacks; countless more would suffer chronic respiratory illnesses

CHLORINE GAS INHALATION

Introduction

Chlorine gas is a mucosal irritant that causes acute damage to the eyes and the upper and lower respiratory tract.

History

Highly concentrated chlorine gas was the first chemical weapon used in war, being unleashed during the First World War at Ypres, France in 1915.

Properties

- Chlorine is a greenish-yellow, noncombustible gas at room temperature and atmospheric pressure.
- In addition, the density of the gas is greater than that of air, causing it to remain near ground level and increasing exposure time.
- It has a pungent odor. The odor threshold for chlorine is approximately 0.2-3.5 parts per million (ppm); however, distinguishing toxic air levels from permissible air levels may be difficult until irritative symptoms are present.

Pathophysiology

Chlorine is moderately soluble in water and is a strong oxidizing agent that forms both hypochlorous and hydrochloric acid on contact with the water of moist mucous membranes.

Elemental chlorine and its derivatives, hydrochloric and hypochlorous acids may cause biological injury.

The chemical reactions of chlorine combining with water is:

$Cl_2 + H_2O \leftrightarrow HCl$ (hydrochloric acid) + HOCL (hypochlorous acid)

Hypochlorous acid may then decompose into hydrochloric acid and oxygen free radicals.

$HOCl \leftrightarrow HCl + (O^-)$

Hydrochloric acid and hypochlorous acid are both highly soluble in water. These and the free radicals cause cellular damage by the disruption of cellular proteins.

The predominant targets of the acids are the epithelia of the ocular conjunctivae, upper and lower respiratory tracts.

Potential sources of exposure to chlorine gas

1. Terrorist activity and chemical warfare.

2. Industrial exposures including the chemical, paper, and textile industries and in sewage treatment.

3. Accidental transport and storage facility leaks.

4. Household exposures, especially accidents involving the inappropriate mixing of hypochlorite cleaning solutions. These are usually caused by prolonged exposure to irritant gas in a poorly ventilated area. The most common mixtures of cleaning agents are sodium hypochlorite (bleach) with acids or ammonia. Potential irritants released from such mixtures are chlorine gas, chloramines, and ammonia gas.

Clinical Features

The early response to acute chlorine exposure will depend on:

1. Concentration of chlorine gas.

2. Duration of exposure.

3. Water content of the tissues exposed.

4. Individual susceptibility.

The immediate effects of chlorine gas toxicity include acute inflammation of:

1. The conjunctivae.

2. The upper respitatory tract, nose, pharynx, larynx and trachea.

    - Laryngospasm and upper airway edema is the most serious complications.

3. The lower respiratory tract, involving bronchi and lung parenchyma.

    - Bronchospasm.

    - Plasma exudation results in a non-cardiogenic pulmonary edema, (ARDS)

        ♥ This is the most serious complication, leading to severe hypoxia.

Exposure thresholds and estimated clinical effects are as follows: [1]

0.2 - 3.5 ppm	Odor detection
1 - 3 ppm	Mild mucous membrane irritation may be tolerated for up to 1 hour.
5 - 30 ppm	Moderate irritation of the upper respiratory tract.
40 - 60 ppm	Toxic pneumonitis and pulmonary edema.
430 ppm	Lethal over 30 minutes

1,000 ppm	Fatal within minutes

Investigations

Blood tests:

All patients should have pulse oximetry.

ABGs, if clinically indicated:

- Abnormalities include hypoxia and metabolic acidosis.
- The metabolic acidosis may be hyperchloraemic (nonanion gap).
- A postulated mechanism for the production of this acidosis is the absorption of hydrochloric acid following the reaction of chlorine gas with water.

CXR

- To look for a pneumonitis or an acute ARDS.

ECG

- Especially in the elderly or those with significant co-morbidity.

Management

1. Immediate attention to any ABC issues.
2. Nebulized salbutamol for any bronchospasm.
3. Oxygen therapy for any hypoxia.
    - CPAP should be instituted for pulmonary edema.
4. Eye and skin exposures require copious irrigation with saline.
    - In cases of suspected ocular injury, determine initial pH using a reagent strip. Continue irrigation with 0.9% saline until the pH returns to 7.4.
    - Topical anaesthetics may be required to help symptoms and allow for patient cooperation in irrigation of the eyes.
    - Treat any corneal ulceration with antibiotic drops and ointment.
5. Nebulized 1 % lignocaine in normal saline may be considered for severely irritant upper airway symptoms.
    - The benefit of this must be weighed up against possible compromise of airway reflexes however.

6. There is no evidence of benefit for IV steroids. Nebulized steroids (eg budesonide) may be of benefit to relieve symptoms, however, again there is no proven benefit.

7. Prophylactic antibiotics are not routinely required.

Disposition

The threshold for admission should be low for:

- Those with persistent symptoms (greater than 6 hours).
- Children and elderly.
- History of severe exposure.
- Significant underlying lung or cardiac disease and smokers.

No hospitalization is required for mild chlorine exposure or for patients who remain asymptomatic.

Prognosis

- Resolution of pulmonary abnormalities in most individuals occurs over the course of one week to one month following acute exposure.
- Most acute exposures do not usually result in any long-term disability. Chronic disability is more closely related to chronic exposure, or very severe acute exposure.

"Gassed" oil on canvas, John Singer Sargent 1918

References:

1. Medical Toxicology 1st Ed Ellenhorn and Barceloux 1988, p. 878

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