Literature.

I have not been able to find in the literature any references to the immediate physiological action of chlorine. The most exact work on the effects of respiring mixtures of chlorine and air is that of Lehmann,² who placed animals during one to six hours in atmospheres containing from 1 part per 1,000,000 to 1 part per 1,000. He found that prolonged exposure to 1 part in 100,000 is sufficient to produce pulmonary hæmorrhage and oedema—which he regards as inflammatory—whilst exposure to 1 part in 10,000 causes in addition a fibrinous ("croupous") exudation in all the air passages, even the finest, and appears ultimately to produce asphyxiation. Numerous experimental pathologists, including Welch,³ S. Mayer,⁴ Sahl,⁵ and Löwit⁶ have endeavoured to elucidate the conditions of production of pulmonary oedema. These conditions have also lately been studied in the "surviving" lung in Magnus's laboratory at Utrecht by Modrakowski.⁷ In these last-mentioned papers the literature of the subject is given, but most of the work referred to has but little bearing upon the oedema produced by chlorine. Binz⁸ found in the frog that exposure to chlorine gas has no influence on the heart (as against Falk), nor on muscle and nerve, nor on the spinal cord, but believes that it is carried to the brain, in which, he states, it can be detected by the smell! I have not noticed any smell of chlorine *post mortem* in the animals I have dealt with.

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TREATMENT OF GAS POISONING IN TRANSVAAL MINES.

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CASES of poisoning by nitrous fumes are not uncommon in the mines of the Witwatersrand, and from published descriptions of "gassing" in the trenches the symptoms appear to be very similar.

"Gassing" as it occurs here is due

(1) To the inhalation of gases formed as the result of burning blasting gelatine. These gases consist mostly of the oxides of nitrogen.

(2) To the inhalation of gases formed by the more or less complete detonation or explosion of blasting gelatine. These gases consist chiefly of carbon dioxide and sometimes carbon monoxide.

The inhalation of (1), even in small quantity, is frequently followed by delayed symptoms, and, unless treated as described below, may cause death within twenty-four hours. This form of "gassing" is apparently very similar to that caused by the inhalation of the asphyxiating gases used by the enemy. The inhalation of (2) may cause no serious symptoms beyond the immediate ones of asphyxia and unconsciousness, and if the patient is rescued in time he recovers without having developed any secondary symptoms. The symptoms are due to the inhalation of CO and CO₂.

A man who has inhaled the fumes of burning gelatine may present very few symptoms beyond a little headache, cough and tightness of the chest, which soon pass off. In from six to eight hours he begins to have difficulty in breathing and coughs up large quantities of bloody serous fluid and dies of asphyxia and heart failure (delayed nitrous fumes poisoning).

Post mortem there is marked congestion and oedema of the lungs. The mucous membrane of the trachea and larger bronchi is injected. The abdominal veins are greatly enlarged and engorged with dark tarry blood. We have had the blood examined, and have never found any nitric compounds, nor has CO been found by spectroscopic analysis. The cause of the symptoms is the inhalation of nitrous fumes.

Treatment.

Every man who has been exposed to nitrous fumes must be kept under observation, in hospital if possible, for at least twenty-four hours. An emetic of copper sulphate gr. 8, followed by a large quantity of water, is given, or a hypodermic injection of apomorphine. It is imperative that vomiting should be freely induced. If an emetic is given early, any other treatment except rest in bed for a day is not usually required. The onset of dyspnoea and cyanosis is watched, and should moist sounds develop at the bases of the lungs bloodletting is employed, and as much as a pint may be withdrawn from a vein in the arm. This is usually difficult, as the blood is tarry, and coagulates very rapidly. We have transfused with saline to replace some of the blood withdrawn. Atropine may be given hypodermically. Inhalation of oxygen is also useful.

Should symptoms of general oedema of the lungs develop, the prognosis is extremely grave. Very few recover when this stage has been reached.

To illustrate the efficacy of treatment, the following case is quoted: In one of the mines under the care of one of us a case of gelatine was accidentally set on fire. The fumes spread through the workings, and fourteen men were "gassed." Thirteen of them immediately had an emetic, and the next day were all out of danger. One man disobeyed orders, and went home without receiving an emetic. He was sent for, but he had gone out for a stroll after having partaken of a hearty luncheon. He was found four hours after the accident, and came to hospital under protest, stating that he felt quite well. In the evening he developed general oedema and congestion of his lungs, and died before morning.

These notes are written in the hope that they may be useful to those treating cases of "gassing" in the trenches.

SOME RESULTS OF GERMAN GAS POISONING.

BY

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CASES of gas poisoning rarely arrive in this country until all the acute symptoms are over, but in the first days of the use of gas two men came under my care who had only been gassed a few days before. Their chests were full of fine moist râles, and there was great dyspnoea. Oxygen they did not like, but compound tincture of benzoin in a steam-kettle gave some relief. The thing, however, which did far and away most good was a big linseed poultice over the whole back. The men constantly asked for the poultice to be repeated.

On admission neither of these men had any albumin in the urine, but a few days later in one of them albumin appeared and rapidly increased in quantity, the urine becoming very scanty. Epithelial and granular casts were present in abundance. The legs became oedematous and the face puffy. There was no rise of temperature. All the moist sounds in the lungs had in the meantime cleared up. The man was in the hospital for two months with very little improvement in the renal condition, and the oedema was still present. He then wished to be transferred to a hospital near his home.

Since then I have seen three other cases of nephritis in men who had been gassed. One had uraemic headaches and early albuminuric retinitis. Obviously nephritis is one of the deadly sequelae of this gas poisoning.

In my other early acute case all physical signs in the lungs cleared up, and yet, when the man began to get out of bed, he was intensely short of breath. He was kept in hospital some time, being sent out in a bath-chair every day, but he could never walk more than about two miles an hour without getting out of breath. I have at present another man in hospital with the same dyspnoea and no abnormal physical signs, who was treated in France for gas poisoning. The condition is very similar to that of a case of South African miners' phthisis, which I saw some years ago, and probably fibrosis of the lungs has been caused by the irritant gas. The outlook for these men must be very bad.

In neither of these cases suffering from dyspnoea was