An Address ON GAS POISONING.

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BY

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THE reprobation of the use of poison gases by the Germans in war does not lie so much in their actual use as in the fact that the German Government broke, in this as in so many other respects, their word, and secretly prepared before the war this method of offence.

All the propellants now in use set free enormous volumes of gases. The late Professor Vivian Lewes calculated that one of the 15in. guns on a super-dreadnought, with its charge of 400 lb. of cordite, gives off about 2,500 cubic feet of carbon monoxide gas each time the big gun is fired. In every battle hundreds of thousands of cubic feet of this gas must be produced, and yet so great is the diffusive power of the atmosphere that no poisoning from it can be traced. Nevertheless, carbon monoxide is a gas so poisonous that the breathing of 1 per cent. quickly renders a man unconscious. The high explosive nitre compounds such as picric acid and tri-nitro-toluol set free, when exploded, not only carbon monoxide but nitric oxide gas, and the latter when breathed has an irritative effect on the lungs closely comparable to that of chlorine.

The fumes of high explosives set free in close spaces, such as cellars and the interior of warships, where the ventilating power of the atmosphere is absent, may poison those who are not actually put out of action by explosive violence.

Gas poisoning, then, to a limited extent, occurred in modern warfare before drift gases and asphyxiating shells were introduced by the Germans. Much has been said of the suffering produced by gas poisoning, and truly dreadful to behold is the fight for breath of the strong man poisoned by chlorine; but we cannot suppose that the sum of suffering produced by gas is greater than that inflicted by shell wounds with the attendant sepsis, lockjaw, and permanent maiming of individuals.

There are poison gases which kill by cutting off the supply of oxygen—for example, nitrogen, hydrogen; these dilute the atmospheric oxygen below a viable amount—and carbon monoxide, which by combining with haemoglobin prevents the carriage of oxygen to the tissues. There are other gases like cyanogen, hydrogen sulphide, and hydrocyanic acid, which when breathed become absorbed into the blood and paralyse the respiratory centre. There is still another set of poison gases which acutely irritate the respiratory passages, causing exudation of lymph therein, which drowns the subject. This last set of gases the Germans have made use of, and for two reasons—first, because they put a man out of action when breathed for a shorter time and in greater dilution than any other poison gas; secondly, because they are heavier than air, and so suitable for drifting with the wind.

DIFFUSION IN THE AIR.

The molecules of gases, unrestrained by cohesion, are able to intermingle freely, and this diffusive process is very greatly quickened by convection currents set up by differences between the soil and air temperature, by currents due to evaporation of moisture, and still more so by winds and the eddies produced by the friction of wind against the soil. Owing to the immense ventilating power of the atmosphere, and the concentration of the poison gas required, the problem of effective use is very difficult. The drift gas must be considerably heavier than air, or diffusion will disperse it—gases intermingle at a rate which is inversely proportional to the square roots of their densities; it must not be too heavy or it will sink to the foot level; the wind must be of the right strength and direction; the gas must poison in a concentration of at least 1 in 10,000, or the quantities required will be unmanageable. It must be borne in mind that a man can hold his breath for at least half a minute, and that the poison gas cloud must therefore last long enough to enforce breathing, and this breathing must continue long enough to put the man out of action. Owing fortunately to the enormous ventilating power of the atmosphere there is no reason to fear that Zeppelins will drop poison bombs on London. The German High Staff know perfectly well that no real frightfulness can be effected in this way. The scare about poison bombs which was prevalent in London some months back was unreasonable, and the public ought to have been warned against the purchase of respirators constructed to be worse than useless in a real emergency.

GERMAN EXPERIMENTS BEFORE THE WAR.

Examination of the back volumes of the *Archiv für Hygiene* shows that poison gases were investigated in Germany for years by Lehmann and his pupils from the ostensible point of view of making safe dangerous trades. From a critical survey of these papers the conclusion is inevitable that if any gases were used in warfare they would be chlorine or bromine. They alone come up to the requirements, namely, (1) that a 1 in 10,000 concentration rapidly puts a man out of action—by asphyxiating him, owing to its intense irritative property; (2) they are much heavier than air; (3) they are manufactured in huge quantities in trade processes; (4) they are easily compressible into cylinders for convenience of transport and handling. Moreover, a respirator is easily contrivable to protect the person who manipulates the brigade gas attack. It is obvious that no drift gas can be used offensively from which the users are unprotected. The density of the various asphyxiating gases which at first were suspected of being used are: sulphur dioxide, 2.21 times heavier than air; nitrogen peroxide, 3.17; chlorine, 2.45; phosgene, 3.49; bromine vapour, 5.53. The power of liquefying a gas by cold or pressure, or a combination of the two, enables the chemist to get into a convenient form large quantities of these asphyxiating gases, but the turning of these liquids back into gases may be troublesome because the heat withdrawn during volatilization may be so great as to freeze the nozzle and stop the outflow.

Special devices are required to produce the expulsion of the gas some distance in front of the trench and to prevent the retardation of flow by freezing. Sulphur dioxide irritates the eyes and air tubes in concentrations of 1 in 2,500; it is liquefied by a pressure of three atmospheres, chlorine by six atmospheres. Of the two, chlorine is a far more powerful asphyxiant, being unbearable in a concentration of 1 in 10,000. Nitrogen peroxide can be liquefied below 26° C. In comparison with chlorine, used in weak concentrations, it has a delayed irritative action on the lungs, and therefore, owing to its want of stopping power, is far less suitable for use. Firemen are sometimes exposed to fumes of nitric acid—for example, after the bursting of carboys; they are unaffected at the time but develop a fatal inflammation of the lungs during the next twelve hours. As the oxides of nitrogen play so important a part in the manufacture of explosives, it is unlikely that the peroxide should be used as a drift poison gas.

BROMINE.

Bromine vaporizes at atmospheric pressures and boils at 59° C. It is far heavier than and as powerful an asphyxiant as chlorine. Germany produces almost the whole of the European supply. It has been said that certain bromine organic compounds have been extensively used by the Germans in asphyxiating and lacrymating shells. The vapours of these substances in concentration as little as one part in several millions of air are said to put a man out of effective action by causing watering of the eyes and inability to open the eyes, so specifically irritating are they to the conjunctiva. They are said also to cause in greater concentrations irritation of the respiratory mucous membrane.

CHLORINE.

Chlorine can be made very easily by heating a mixture of hydrochloric acid and black oxide of manganese, or by electrolytic processes. It can be stored in lead-lined cylinders. The gas above the liquid chlorine exerts a pressure of at least 90 lb. per square inch, so that all that the Germans required to project chlorine was a long tube projecting in front of the trench parapet and a valve. The spray turns into a yellowy-greenish vapour, and owing to its weight drifts with the wind along the ground. Any one who has watched smoke from a weed bonfire drift over a

field will see how far the chlorine vapour may be carried in poisonous concentration. It will sink into trenches, shell-pits, mine-craters, cellars, and dug-outs. To produce a concentration extending 10 ft. up of 1 in 10,000 during a period of ten minutes in a wind moving uniformly four miles an hour, over 1,000 cubic feet of gas are required for each hundred yards of front. This is leaving out of account diffusion and the ventilating power of the atmosphere. It is clear, then, how large a volume of gas is required for an attack, and how any gas which does not come up to the 1 in 10,000 standard must be ruled out.

Lethal Doses.

To estimate the lethal dose of chlorine or bromine special methods have to be devised because these gases combine very readily with the hair of an animal, turning this into a gummy substance. My fellow worker, Dr. Benjamin Moore, found that hair dissolved in bromine into a gummy red-black mass, from which the bromine could be washed away, leaving a white friable substance. This bromo-protein compound gave an intense violet biuret reaction, and on addition of strong nitric acid yielded up its bromine. The effect of 1 in 10,000 chlorine is such that no man would endure breathing it who could escape from its influence. The eyes and the mucous membranes of the respiratory tract are intensely irritated and a watery exudation takes place, the inevitable effort which the living tissues make to dilute so irritant a poison.

Effect on the Lung Structure.

Just as lymph is poured out after a superficial burn of the skin, or the application of a blistering fluid, or in a septic wound under the influence of bacterial toxins or antiseptics, so does chlorine produce an exudation of lymph in the lungs. The epithelial lining, both that of the mucous membrane and of the capillary wall, is damaged by the poison. The osmotic pressure of the damaged tissue is raised—the celloidal lining complex becoming killed and disintegrated with the setting free of crystalloidal substance. Thus fluid is pulled out by osmotic forces, while through the damaged capillary wall, too, the plasma may actually leak away. The classical first symptoms of inflammation thus appear, ending in stasis of the corpuscles in the capillaries owing to exudation of the plasma. In the earliest stage the salivary glands in the mouth and the mucous glands in the air tubes are stimulated to secrete, just as the tear glands flood the eyes. It is this pouring out of the fluid in a vain effort to ward off the poison which causes the asphyxial symptoms of chlorine poisoning and finally drowns the man. He is as surely drowned by the exudation as he is when he breathes water into his air tubes. The mucous membranes of the nose and mouth, wet with secretion, at first act as a protective respirator, catching much of the poison and preventing it entering the lungs. That this is so is seen by the greater celerity with which serious symptoms arise in an animal when chlorine is administered through a tracheal cannula instead of through the nose and mouth. It is a remarkable fact that while 1 in 10,000 is unbearable to breathe, and 1 in 100,000 is distinctly irritative, yet we find it takes a concentration of as much as 1 in 3,000 of chlorine dissolved in water to stop the movement of the cilia in a preparation of ciliated epithelium observed microscopically. Chlorine is much more toxic when it comes in contact with the moist living membrane in a gaseous state than when in watery solution.

Sir Edward Schäfer[1] has drawn attention to this. "From the chemical nature of chlorine," he writes, "it seems evident that its immediate action must be local. For it is scarcely possible to imagine that it can exist in the free state in such a fluid as blood, which contains many bodies with which it would immediately combine, and which would—unless it were introduced in immense quantities—at once render it innocuous." When 10 c.cm. of Ringer's solution saturated with the gas were injected by Schäfer into the jugular vein of a rabbit, in a period of 20 seconds there occurred a quite temporary fall of blood pressure and increased depth of respiration. Only in one case when the same amount was injected rapidly and with, therefore, less perfect admixture of blood, did oedema of the lungs and congestion result in the pulmonary vessels, producing a fatal result. The irritative effect of the dissolved chlorine is spent on the blood, or the lung, the first tissue it comes in contact with. When inhaled the chlorine spends its effect on the air passages and lungs, and we have no evidence that free chlorine or any poisonous chloro-protein complex is formed, which, conveyed by the blood, poisons other tissues. Major Walter Broadbent,[2] in a note concerning nephritis following chlorine poisoning, says: "It looks as if in some cases the chlorine or bromine damages the lung epithelium so severely that it does not allow absorption into the general circulation, while in others the gas passes through the lungs without affecting them permanently, but then sets up an acute nephritis."

It is not possible to uphold this theory. Chlorine gas in every case expends its fury on the lungs. The nephritis, I believe, is due to the intense and prolonged dyspnoea and the struggles for breath. Albuminuria is a common result of the very temporary dyspnoea which athletes suffer in a race. It results in such case from the want of oxygen in the kidney, just as it does when the renal artery is temporarily occluded. It is, I believe, the want of oxygen which produces the increased acidity of the blood observed by Mr. Barcroft in a few cases of chlorine poisoning, including a dog experimentally poisoned by us. No doubt the products of the damaged pulmonary tissue, absorbed during the days subsequent to the poisoning, have a toxic effect, particularly as the damaged lung becomes infected.

Symptoms.

We are told[3] that a typical case on admission is cold with a subnormal temperature, conscious but restless, with pulse slow and full (except in the collapsed cases). The face is cyanosed, intensely so in many cases, and the expression strained and anxious. The posture varies. In some cases the patient sits propped up, with head thrown back, gasping for breath; in others, he lies on his side, with his head over the edge of the stretcher in an attempt to aid expectoration. The respirations are jerky and hurried, often numbering 40 a minute, and are associated with a choking cough, accompanied by a varying amount of frothy expectoration. With each inspiration the chest is expanded to its fullest, all the auxiliary muscles being brought into play just as in an asthmatical paroxysm.

This is the first or asphyxial stage, which, if the patient survives, gradually passes off after some thirty-six hours. Can we wonder that such long-lasting intense dyspnoea should produce nephritis, accompanied as it is with convulsive breathing which just maintains the cerebral circulation within viable conditions at the expense of the abdominal circulation? Major Broadbent records a case in which he believes a cusp of the aortic valve was ruptured in the struggles for breath.

"After the first stage the patient falls into a sleep, and awakes feeling much better. But after a few hours of comparative quiet symptoms of bronchitis begin to manifest themselves. In the majority of cases these are not severe"—because, no doubt, nearly all the severe cases die in the first stage. "In the cases which are kept alive with difficulty there is a short quiescent stage followed by intense bronchitis. The frothing gives place to greenish muco-purulent expectoration, consciousness to delirium, the temperature rises from subnormal up to 104° F., the pulse becomes of small volume, with its rate increased perhaps to 160, the respirations are less choking but more shallow, and number up to 70 per minute before death."

Pathology.

Post-mortem examination in the acute cases shows an intense congestion of the mucosa of the trachea and larger bronchi. These tubes are filled with a thin, light yellow frothy secretion, some of which escapes from the mouth and nose when the cases are first laid on the table. The fluid is highly albuminous, solidifying on heating. The larger bronchi only can be traced, the smaller being lost in a condition of intense congestion and oedema which affects the lungs as a whole. The lungs do not collapse in these acute cases, but appear like a solid cast of the thoracic cavity, and are greatly increased in weight. On incision, the lung tissue appears of a deep maroon red colour, and the exudation flows from the cut surfaces in abundance. Light grey patches are to be seen on the surface of the lungs amidst the congested areas. They were found to be due to emphysema. So intense is the obstruction to the entry of air, and so violent the efforts of respiration, that emphysema is produced in these least poisoned parts where air can still enter. We can picture how the violence

of the respiratory efforts, brought to bear on a relatively few small parts of the lungs, distends and breaks down the walls of the alveoli, expelling the blood into surrounding congested areas.

The parts of the lung tissue not affected by emphysema show the intense congestion of the capillaries, and many of the alveoli are seen filled with exudate which takes on the eosin stain. Into some alveoli red corpuscles escape; larger patches of haemorrhage may occur. The heart in these acute cases is congested, particularly on the right side. The stomach shows a condition of catarrh, the mucosa being covered with a thick yellowish mucus and haemorrhages being visible in the submucosa. These changes may conceivably be due to the swallowing of saliva and exudate from the nose and expectorated fluid in which chlorine is dissolved. The venous congestion of the stomach and other abdominal organs and of the brain is due to the asphyxial character of the death.

Experiments made on animals make quite clear the stages of toxic effect. Using bromine in concentration of 1 in 1,000 approximately we find that the mucous membrane of the windpipe, killed by the poison, may be stripped off by the violence of the respiratory efforts, so that drawn down into the large bronchi it forms a tree-like cast therein, suffocating the animal. Chlorine, in our experience, causes a greater exudation of fluid than bromine. Chlorine (1 in 1,000) breathed through a tracheal cannula may shortly cause in a cat such an exudation of fluid that it fills up the trachea. By compressing the chest many cubic centimetres of the fluid can be squeezed out of the lungs into a basin. It is a clear serous liquid, containing plenty of coagulable protein. That this fluid drowns the animal may be seen by the relief which is given after squeezing it out.

Professor Schäfer, experimenting with very high concentrations of chlorine gas—for example, 1 to 2 per cent.—has concluded that death is brought about by stasis in the pulmonary vessels. If this be so for the high concentrations used by Sir Edward, it is not so in the case of the weaker concentrations, such as are breathed on the battlefield.

We have put the matter to the test in two ways: (1) We manipulated the animal so that we could artificially respirate one lung with pure air, the other with air containing chlorine. Recording the blood pressure we first of all proved that artificial respiration of either lung sufficed to maintain the circulation in undiminished vigour. We then gave air *plus* chlorine to the one lung, and observed the gradual production of congestion, oedema, and lessened expansion of that lung, leading to symptoms of asphyxia and failure of the circulation. On carrying out the respiration of the other lung with pure air we observed the complete and immediate recovery of the circulation. On now squeezing the fluid out of the first lung and changing the respiration to that we see that the circulation may continue, the asphyxia no longer being complete.

In the other set of experiments we had the co-operation of Dr. Kuno, of the Physiological Institute, University College, one skilled in the particular technique required. The circulation was confined by Dr. Kuno to the heart and lung preparation and the technical arrangements made so that the output of the heart could be measured at any period of the experiment. The thorax was widely open and the lungs exposed to view. On giving chlorine the first and immediate effect was a very evident diminished expansion, due, we thought, to contraction of the bronchial tubes. Congestion and oedema followed, appearing first in patches on the surface and then spreading; as these grew marked the blood became more and more venous; the output, it is true, was then diminished, but whatever stasis there was in the pulmonary vessels did not markedly affect it.

It is well known that a very large part of the pulmonary vessels can be ligatured and yet an adequate circulation be maintained through the remainder. A very small portion of lung suffices, too, to keep up the oxygen supply to the heart.

These experiments made clear to us how artificial respiration keeps alive the gas-poisoned animal. If fluid be forcibly squeezed out of the lungs of a chlorined cat, struggling for breath, its condition is greatly improved. If air is forced by artificial respiration into the lungs emphysema in places may be produced, but the heart is kept going.

Symptoms and Lesions of the Lungs in Experimental Animals.

Animals exposed to chlorine exhibit first of all profuse watering of the eyes and salivation; they make efforts to escape, and if the chlorine in the chamber is not mixed by a fan but sinks to the lower parts, they hold up their heads as high as possible to escape breathing the more concentrated lower stratum. The respiration soon becomes quickened, and then, as the oedema of the lungs and exudation into the air tubes increases, the respiration becomes slower and laboured. The obstruction to the entry of air becomes great, and in consequence the lower ribs are drawn in with each inspiratory gasp. The mouth gapes open and a frothy secretion hangs round the orifices of nose and mouth. The whole effort of the animal is given up to breathing; finally, it falls over exhausted, the breathing becomes rarer and shallower, and it dies. If in the stage of laboured breathing the animal be removed from the poison, it generally dies during the next twenty-four hours, but may live longer to die within the next few days. One of our animals died as late as a fortnight after the exposure.

Examination of the lungs of those which die in the first twenty-four hours shows an intense congestion of the lungs; they are dark red in colour; a more or less solid oedema prevents their collapse on opening the thorax. The air tubes contain frothy exudation which, on cutting the lungs, exudes in large quantities. When the lung of a rat was kept in a covered dish it shrank like a blood clot, exuding serous fluid, till it floated in it.

The animals which die in the later days show more or less extensive patches of red hepatization. Those parts of the lungs which were least poisoned appear relatively normal, but are more rosy in colour.

Microscopic examination was carried out on the animals poisoned by us by Professor William Bulloch. The sections show intense congestion and small haemorrhages in places, and an oedema which fills the hardened alveoli with an eosin-stained homogeneous coagulum, reminding one of the appearance of the thyroid alveoli filled with colloidal secretion. The coats of the arteries are enormously distended with exudate, giving a most remarkable picture. The epithelium of the air tubes is in many places detached. The animals die in the early stages from asphyxia, and in the later stages from pneumonia with consequent absorption of toxins and exhaustion.

In those animals which are less severely poisoned the laboured breathing gradually passes away; those that recover appear quite normal at the end of a fortnight. Their fur, which was made sticky, and looked, so to speak, burnt at the ends, becomes glossy again, the damaged hairs being shed. These animals, if killed during the process of recovery, generally show small pneumonic patches. The lungs seem very sensitive to further injury during the period of recovery; inhalation of chloroform may cause acute oedema, and drown the animal.

There seems no reason why recovery of the lungs should be any less perfect after chlorine poisoning than it is after bronchopneumonia. The damaged and shed epithelium of the air tubes can be replaced, and the pneumonic patches resolved by the absorptive action of the phagocytes, until repair is complete.

I have no evidence to offer as to the state of the lungs at any long period after the poisoning.

Irregular Distribution of Lesions.

The remarkable fact that some parts of the lungs are far more severely damaged than others requires an explanation. When chlorine of, say, 1 in 1,000 is driven into the lungs by artificial respiration, and the lungs are exposed and observed from the start, it is evident that the poison reaches and severely damages certain parts in the surface, while other points remain normal. In these experiments the current of air from the pump was driven through chlorine water and then passed into the windpipe. We must suppose either that the chlorine does not uniformly mix with the air, or that certain air tubes are shut up by contraction of the bronchial muscles, and so prevent the poison reaching the alveoli they supply.

There is no doubt that the first effect of concentration such as 1 in 1,000 is to cause contraction of the bronchial muscles and diminish the expansion of the lungs. Using enormously strong concentrations, Sir Edward Schäfer finds no evidence of such contraction—probably the concentrations he used rapidly killed the lining membrane of the air tubes, including the muscle. Some experiments conducted by F. J. Twort and myself on the oxygenation of the blood, in subjects breathing in a shallow way, suggested to us that parts of the lungs may then not be expanded, nor the blood oxygenated in these parts, and that the bronchial muscles may regulate to which part of the lungs the air goes on each inspiration. The results of our chlorine experiments seem to confirm this view.

TREATMENT.

Expulsion of Fluid.

For the severe cases of chlorine poisoning the one object of treatment must be that of getting rid of the exudation in the air tubes which is drowning the victims. Experiments on animals show that the frothy fluid can be easily squeezed out of the lungs and trachea by rhythmic compression of the thorax, and that the dyspnoea which is threatening life can be greatly, if only temporarily, eased by this means. Artificial respiration is reported to have given good results in several of the cases on which it was tried. It must be repeated as often as the dyspnoea becomes excessive. The case is recorded of one man, almost moribund, who was treated in this way on four successive occasions, and ultimately recovered. After squeezing out the fluid, air may be blown into the lungs by mouth to mouth artificial respiration to overcome the resistance of the froth in the smaller tubes and expand enough lung to keep the patient alive. It is true that emphysema may be caused by so doing, but if it is a question of just carrying a man through the threatened asphyxia, one cannot hesitate to get air into the lungs by these means.

I took over to Flanders an apparatus constructed by Messrs. Siebe Gorman called the "Vivator," in which there is a foot-pump which feeds a face-mask through a flexible tube. By each downstroke a measured volume of air or oxygen is pumped into the lungs, by each upstroke a valve is opened which allows the air to escape from the lungs by the elastic recoil of the thorax and lungs. With this apparatus respiration can be kept going in the collapsed or unconscious cases, the fluid now and again being evacuated by squeezing the thorax and by posture.

The inverted posture will help to drain out the fluid. I was told that several of the patients of themselves hung their heads down over the side of the stretcher or table, in order to aid their expectoration.

Emetics.

Emetics have proved very useful in giving relief to the less critical cases. Half a pint of salt and water or 8 grains of copper sulphate, followed by large draughts of lukewarm water, are recommended. A brush or the patient's finger put to the back of the throat will initiate the vomiting without delay. The act of vomiting is reported to cause the expulsion of a large quantity of the frothy liquid.

Oxygen.

Administration of oxygen relieves the cyanosis and improves the condition of the subjects.

Not only does the percentage of carbonic acid in the blood rise in the suffocative condition, but other acids such as lactic acid increase in quantity owing to the lack of oxygen. When the blood is oxygenated by breathing of oxygen these other acids do not appear, and the acid intoxication is therefore so far eliminated. Tests of the power to hold the breath show that a higher percentage of carbon dioxide can be borne when oxygen rather than air fills the lungs. To give oxygen to a man who is struggling for breath and needing to expectorate is no easy matter. It is difficult to get any kind of close-fitting face-mask tolerated. The ordinary clinical method of administering oxygen through an open funnel held near the mouth and nose is of relatively small value; nearly all the oxygen is wasted by escaping into the atmosphere; just at the period of inspiration the stream which reaches the mouth and nose is not enough, so that the air drawn into the lungs is very slightly enriched. I have found the oxygen in my alveolar air increased by only 1 or 2 per cent. when oxygen was administered to me by a sister in a London hospital. If a loose kind of face-mask be made out of a towel, and the oxygen tube be led under that, and the oxygen sent in sufficient stream to blow away the exhaled carbon dioxide, then 70 per cent. of oxygen can easily be obtained in the alveolar air. Down Bros. have made a transparent face-mask to my design, fitted with a curtain which drapes the face, by which oxygen can be effectively given on this plan, but not economically. A 20 ft. cylinder is soon blown away by these methods. To give oxygen economically a well-fitting face-mask, breathing bag, and cartridge for absorbing the exhaled carbon dioxide must be used. The subject breathes through the cartridge in and out of the breathing-bag, which is filled with oxygen from the cylinder as required. The cartridge is loosely packed with small pieces of caustic soda-coke; to prepare these the coke pieces are heated red-hot and dipped into strong caustic soda. They offer a splendid absorbing surface and no appreciable resistance to the breathing. The apparatus—made by Messrs. Siebe Gorman—I took over for use in Flanders. The difficulty in using such lies in keeping the mask over the face of a man who wants to struggle and expectorate. Oxygen breathed between the periods of expectoration will undoubtedly give him relief, and with the above apparatus a 20-ft. cylinder will give a supply lasting many hours.

Compressed Air.

Experiments on animals have shown us that compressed air relieves the dyspnoea to the same extent as oxygen does. On placing a patient in a medical air-lock, such as is used in compressed-air tunnel works, and compressing him to two atmospheres, he would breathe double the concentration of oxygen and at the same time would be able to expectorate and struggle as he pleased. The compression of the air when first applied would halve the size of the air bubbles in the frothy liquid which obstructs the air tubes, and this should give relief by lessening obstruction. Artificial respiration could be applied in the compressed-air chamber and the subject be kept in it for several hours, and then slowly decompressed. The medical locks are fitted with air-locks, through which the medical officer can enter or leave. The difficulty, of course, lies in the provision of such medical locks—heavy cylindrical boiler-like structures, each of which would hold only four or five patients, with the necessary oil-driven compressor engine. A small medical lock and engine would go on a 3-ton lorry, but it is a serious thing to hamper the transport of the army with such a provision. There is another way in which oxygen might be administered without the use of a mask, and that is by drawing over the stretcher containing the patient a cylindrical balloon, say 10 ft. by 4 ft., tying up the end, and then distending the balloon with oxygen.

Atropine, etc.

The giving of atropine has been extolled on the theory that it lessens secretion of fluid and dilates the bronchial tubes. In severe cases of poisoning we have not found it of the least service.

It is claimed that the inhalation of stramonium vapour from cigarettes relaxes the bronchial muscles; this may afford relief in the mild cases, which recover whether so treated or not.

CONCLUSION.

The chlorine poisoning of the lungs is comparable to extensive burns of the skin, and the same general treatment to support strength and lessen shock is required. Just as septic infection of the skin is the sequel of the burn, so pneumonia and bronchitis follow chlorine poisoning. In our experimental animals severe poisoning has in every case had this end, and we know of no means of preventing it. Warmth and good nursing might pull a man through; these conditions are difficult to apply to animals.

REFERENCES.

[1] BRITISH MEDICAL JOURNAL, August 14th, 1915. [2] BRITISH MEDICAL JOURNAL. [3] Black, Glenny, McNee, BRITISH MEDICAL JOURNAL, July 31st, 1915.