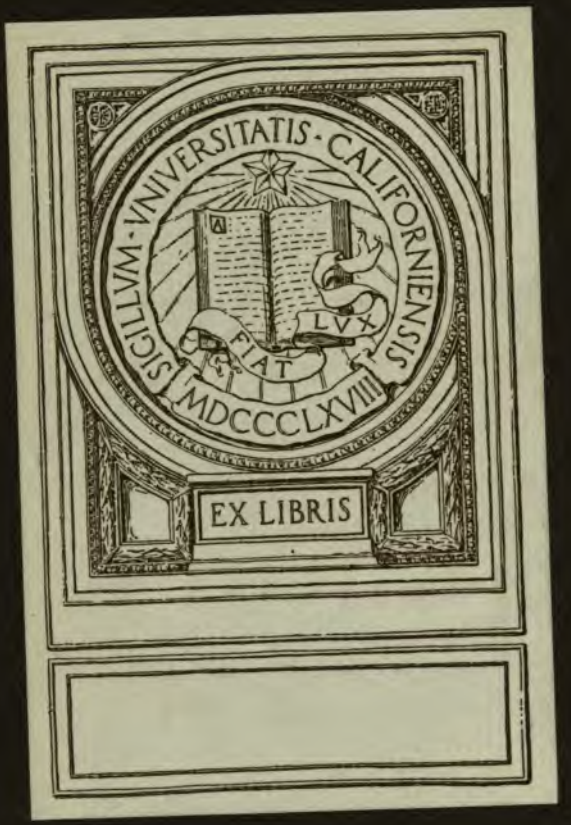
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MEMORANDUM ON

GAS POISONING IN WARFARE

WITH NOTES ON ITS

PATHOLOGY AND TREATMENT

NOTE.—This Memorandum has been drawn up by a Committee of Consultant Physicians and Physiologists for the information of Medical Officers. IT IS TO BE TREATED AS CONFIDENTIAL, AND SHOULD ON NO ACCOUNT BE TAKEN INTO THE TRENCHES.

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The following Memorandum on Gas Poisoning in Warfare, with notes on its pathology and treatment, is published for the information and guidance of all concerned.

[2599035, A. G. O.]

BY ORDER OF THE SECRETARY OF WAR:

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Major General, Acting Chief of Staff.

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GENERAL CONSIDERATIONS.

The employment of poisonous gases as a means of offensive warfare has made it imperative that medical officers should have some knowledge of the action of the various gases that are likely to be met with and of rational lines of treatment which may be adopted in cases of gas poisoning.

Poisonous gases have been used in warfare with the intention of putting men out of action, hampering Artillery, preventing supports from advancing, and inspiring general terror, so that an advance may be made with a minimum of opposition and some military advantage secured. Apart from any question of an advance, poisonous gases have been used merely as a means of causing a certain number of casualties on the opposing side and of inspiring terror.

Besides this deliberate use of gas for offensive purposes, poisonous gases are liable to be encountered under other circumstances—e. g., during mining operations, or as the result of the imperfect detonation or burning of explosives.

The Mode of Action of Poisonous Gases.

Poisonous gases that are liable to be met with can be grouped roughly, according to the main physiological action that they produce on the animal body. It must, however, be clearly understood that it is impossible to draw absolute distinctions, since many of the gases partake of the characters of at least two of the groups.

I. GASES WHICH ACT AS LUNG IRRITANTS.

The main characteristic of gases of this group is that they cause the onset of a profuse pulmonary edema. The accumulation of fluid in the lungs interferes with the respiratory exchange, causing intense cyanosis and death by asphyxia. In addition to this these gases are irritating to the respiratory passages, some of them intensely so, causing great pain and discomfort in the chest. They may also cause marked irritation of the eyes and lachrymation. In some cases circulatory failure is a very marked feature, and the cyanosis may then give place to the leaden pallor of collapse. With some gases of this class mild poisoning may lead to a condition somewhat akin to asthma.

Type 1—Chlorine.—A concentration of 1 in 100,000 can be respired for a considerable time, though this may give rise to subsequent bronchitis.

A concentration of 1 in 10,000 is barely respirable by man and experimentally is not borne for more than two or three breaths. It produces irritation of the eyes, spasm of the glottis, a feeling of choking, and severe cough. If the inhalation is continued, the struggle for breath becomes more acute and the man will be rapidly incapacitated. In such a concentration edema of the lungs commences very soon and develops steadily.

Type 2—Nitrous fumes, i. e., nitric oxide and nitrogen peroxide.—Sensory irritation of the eyes and upper respiratory passages is slight as a rule, and passes off in a short time after leaving the dangerous atmosphere. For some time after this the man may feel perfectly well and go about his ordinary business, but after the lapse of a few hours acute symptoms of pulmonary edema suddenly come on and progress with alarming rapidity.

In contradistinction to chlorine poisoning, there is, therefore, a very definite delay before the onset of pulmonary edema, and the slightness of the symptoms shown by the man while he is in the dangerous atmosphere would not naturally lead one to anticipate the disastrous effects which may develop later.

Type 3—Phosgene.—In certain characteristics this gas holds an intermediate position between chlorine and nitrous fumes. Though it is a more intense eye irritant than chlorine, it is not so immediately irritant to the upper respiratory passages and there is some delay before the onset of pulmonary edema. Circulatory failure is apt to be a prominent feature and cases exhibiting a leaden pallor and collapse are the rule rather than the exception. Phosgene incapacitates a man more slowly than chlorine but is a more serious poison.

II. GASES WHICH ACT AS EYE IRRITANTS OR LACHRYMATORS.

The main characteristics of this group of gases are that in exceedingly small concentrations they exert an intense irritant effect on the eyes and cause so profuse a flow of tears and often so much pain that vision may be impossible. In stronger concentration they may also act as acute lung irritants.

Type—Benzyl bromide.—A very minute concentration causes marked lachrymation. A high concentration, when the eyes are protected, rapidly causes respiratory discomfort, irritation of the throat, and nausea.

III. GASES WHICH ACT AS DIRECT POISONS OF THE NERVOUS SYSTEM.

The characteristics of this group of gases are that in sufficient concentration they act on the central nervous system with great rapidity, causing a cessation and finally a total abolition of its functions. Some of these gases cause delayed pulmonary edema in cases which have not succumbed rapidly from the effects on the central nervous system.

Type—Hydrocyanic acid.—Minute concentrations are practically innocuous. High concentrations cause immediate unconsciousness and death in a few seconds.

Animals if taken out of the poisonous atmosphere as soon as they have fallen down paralyzed, may recover perfectly in a short time.

IV. GASES WHICH ACT BY INTERFERING WITH THE RESPIRATORY PROPERTIES OF THE BLOOD.

Under this heading may be grouped cases which cause destruction of red cells, alter the hemoglobin, or combine with the hemoglobin to the exclusion of oxygen, so that severe symptoms arise because the oxygen-carrying power of the blood is interfered with. Certain cases may, for instance, lead to destruction of red cells, accompanied by hemoglobinuria or jaundice. Again, nitrous fumes in strong concentration may cause the alteration of hemoglobin into methemoglobin in addition to producing the intense irritation of the lungs mentioned above.

Carbon monoxide.—This gas owes its poisonous action to the fact that it combines with hemoglobin to form a dissociable compound and thereby takes the place of oxygen. Carbon monoxide has about 300 times the affinity for hemoglobin that oxygen has. If therefore a small proportion of carbon monoxide is present in the air breathed, the hemoglobin in the body will divide itself between the two gases, the final partition being determined by the relative concentration of the two gases. The oxygen-carrying power of the blood is progressively diminished as the hemoglobin becomes more and more saturated with carbon monoxide, and symptoms arise owing to the interference with the oxygen supply to the tissues from this cause.

CIRCUMSTANCES UNDER WHICH GAS POISONING MAY BE MET WITH IN WARFARE.

Some poisonous gases of the foregoing groups are well adapted for use as offensive agents in warfare, while others are unsuitable owing to various causes, such as difficulty of manufacture or of storage, chemical instability, limited toxicity save in high concentrations

or after prolonged exposure, or insufficient density. Though the acute lung irritants as a whole must be classed among the more powerful methods of destruction, nitrous fumes do not lend themselves to deliberate offensive use. They may be met with during warfare accidentally under certain circumstances. Carbon monoxide, too, is not adapted to offensive use, though it may prove a very serious factor indeed when met with during mining operations.

THE USE OF GAS FOR OFFENSIVE PURPOSES.

(*a*) *Cloud gas.*—The rate at which the cloud gas is carried over the ground intervening between the trenches of the two opposing forces depends entirely on the velocity of the wind. With winds at the rates of 3, 9, and 15 miles per hour, the gas cloud would move forward at the rate of 90, 270, and 450 yards per minute, respectively; and with a wind of 9 miles per hour, the distance of 100 yards would be covered in about 20 seconds. These figures indicate how short may be the time available for warning men in the front trenches of the commencement of a cloud gas attack and emphasize the extreme importance of training troops thoroughly in the use of protective devices.

(*b*) *Gas shells and gas bombs.*—In this case the poisonous substance is contained in liquid form in the shell or bomb and is converted into a cloud of vapor on the explosion. As a rule these shells are employed to produce a toxic effect at a distance over a limited area and are most frequently used against batteries, or in the form of a "tir de barrage" to interfere with the movements of supports during an engagement.

The commonest gas shell used by the Germans contains the lachrymators, benzyl, and xylyl bromides, but shells containing acute lung irritants have also been employed.

A cloud gas attack necessitates a favorable wind blowing toward the enemy's position, but gas shells can be used even if the wind is blowing toward the user, since if the gas is blown back, it will have been so diluted with air as to be harmless to him. A cloud gas attack may be preceded or accompanied by a gas shell attack, and gas shells of different forms may be fired not only on points behind the trenches but also on the front line and support trenches as well.

Medical officers must, therefore, remember that more than one method may be used at the same time during a gas attack and that different chemical substances may be simultaneously employed. They must in consequence be on the watch for differences in the symptoms presented by gassed cases so that they may report the facts to headquarters and adopt suitable treatment.

CIRCUMSTANCES UNDER WHICH POISONOUS GASES OTHER THAN THOSE USED DELIBERATELY FOR OFFENSIVE PURPOSES ARE LIABLE TO BE ENCOUNTERED.

(*a*) *Mine gases.*—During mining operations the most dangerous gas met with is carbon monoxide. This arises from the imperfect detonation or combustion of the explosives used for charging the mine or for blasting. The galleries may be flooded with the gas after the explosion of either our own or a hostile mine, as the result of the explosion of a hostile camouflet designed to blow in our galleries, or as the result of the explosion or burning of a blasting charge. The gas is liable to be driven into the ground disturbed by the explosion and to come welling out later, especially if the barometer commences to fall after the explosion, or it may be met with in the form of pockets of gas when new galleries are driven through ground disturbed by a previous explosion. Carbon monoxide has even been known to be driven out of the shafts into the adjacent trenches after the explosion of a mine.

Besides carbon monoxide, nitrous fumes may arise if a charge does not explode properly, and especially if it burns instead of exploding. Nitrous fumes are quickly absorbed by moist ground, and so cases of nitrous fumes poisoning are seldom met with unless men are exposed to the fumes immediately after the explosion or the burning of the charge.

(*b*) *Fumes from high-explosive shells.*—Nitrous fumes have so far given rise to no cases of poisoning after the explosion of high-explosive shells. Cases of carbon monoxide poisoning have been known to arise under special circumstances—e. g., when a shell has penetrated into a deep dugout before bursting. Such cases are, however, unlikely to arise unless the shell explodes in a confined space, since either a high concentration or a prolonged exposure is required for the production of symptoms.

(*c*) *Fumes from the firing of guns.*—In closed machine-gun emplacements, owing to the blowback from the gun, enough CO sometimes gets into the air to give rise to symptoms. Symptoms of CO poisoning are also sometimes caused in gun pits owing to the blowback on opening the breech when certain forms of powder are used.

(*d*) *Burning of nitro explosives.*—If nitro explosives are accidently set on fire and continue to burn without explosion, there is great danger of poisoning from nitrous fumes. Carbon monoxide poisoning may also occur.

(*e*) *Burning buildings.*—It must be remembered that when combustion takes place in a limited supply of air, as in the interior of a burning building, there is a considerable risk of carbon monoxide poisoning.

(*f*) *Warming of billets.*—A burning coke brazier in a small, ill-ventilated billet is very likely to give rise to cases of carbon monoxide poisoning.

Poisoning by Gas Cloud.

The gas used by the Germans in attacks made on the English trenches by poisonous clouds drifting forward on the wind was in the early summer of 1915 chiefly, if not solely, chlorine. Later it became apparent from the change of symptoms in casualties that a different form of gas was being employed and this is believed, though not proved, to be a mixture of chlorine with varying proportions of carbonyl chloride or phosgene gas. It is, therefore, necessary to describe separately the action of each type of cloud gas.

CLOUD GAS OF SUMMER OF 1915.

The following account of the pathological changes is based entirely upon observations made on men injured by German cloud gas in 1915, and these observations coincide with what has been noted in laboratory experiments with chlorine. The effects may be classified under the following headings:

Irritation and inflammation of the respiratory tract.
Interference with the circulation.
General toxic effects.

RESPIRATORY TRACT.

Chlorine is very irritant. Sensory nerves are violently stimulated, causing immediate pain and uncontrollable reflex movements, such as coughing or spasmodic gasping, which interfere with respiration. Lachrymation is relatively slight. A heavy concentration of the gas seems almost irrespirable, but the spasmodic check to breathing ultimately gives way, and no case of death from asphyxia immediately upon exposure to the gas has been proved to have occurred in the English lines. Breathing continues, but it is of a spasmodic character and accompanied by violent coughing which, as will be described later, has a very harmful effect on the lungs.

The irritation of the sensory nerves, however, is not the fatal factor. An inflammatory reaction, with congestion of the vessels, edema of the tissues and an abundant discharge of serous effusion through the

dying epithelium occurs all the way down the respiratory tract. This begins at the back of the throat; the larynx, with its resistant epithelium, escapes injury, but the damage to the surface tissues increases progressively down the trachea, the bronchi and their finer branchings, and ultimately attains its maximum in the air sacs. The bronchioles are rapidly filled with a serous exudate which passes up into the trachea and is coughed out.

A universal obstruction in this way of the bronchioles alone would suffice to cause death by simple blockage of the airway and asphyxia. But the injury is not confined to the bronchial tree, and probably the effusion in the tubes alone is not dense enough to hinder the passage of some air up and down, though even this thin fluid may constitute a very serious mechanical obstruction when it is churned up into a continuous foam by violent respiratory efforts. At first there may be so much spasmodic contraction of the muscles of the smaller bronchioles that the gas is denied access to many areas of the lungs. Sooner or later these relax, and the irritant vapors then destroy the pulmonary epithelium, while the air sacs rapidly fill up with inflammatory exudate and become utterly useless for the purpose of respiratory exchange. The edematous effusion must ultimately compress the capillaries and cause great hindrance to the flow of blood through the lungs. The irritation spreads so widely that a blood-stained serous effusion soon accumulates in the pleural cavity, but this is never so abundant as to require aspiration.

In addition to this inflammatory edema, which chokes the circulation and prevents gaseous exchange, there is often some actual damage to the structure of the lungs. The violent coughing and inspiratory efforts which may result from inhalation of the gas may be so forcible that the partitions between the alveoli are torn and air may even burst its way out into the areolar tissue and escape along the hilum of the lung and up into the subcutaneous tissues of the neck. Such disruptive emphysema adds at once to the disability of the sufferer, since it throws out of action that part of the lung which is not yet submerged in the rising flood of edema fluid, and it may lead to permanent shortness of breath if the man survives.

There are, therefore, four factors which develop in rapid succession and prevent exchange of respiratory gases through the lungs:

(1) Narrowing of the bronchioles by spasmodic contraction of muscles in their walls.

(2) Disruptive emphysema.

(3) Obstruction of the bronchial tubes by exudate.

(4) Flooding of the pulmonary air sacs by serous effusion into them.

The result of these changes in the lungs, of which (3) and (4) are the most important, is that the intake of oxygen and the elimination of carbon dioxide are seriously interfered with, and the man passes into a state of asphyxial cyanosis in which mental failure and unconsciousness may rapidly supervene. During this state the breathing is labored, rapid, and interrupted by spasmodic bouts of coughing. The blue color of the face shows the urgent need for oxygen, and every muscular effort made by the body and by the heart increases that need. Coughing is helpful in so far as it may dislodge exudate from the bronchi, but the violent effort is prodigal of such oxygen as can be supplied and it tends to increase the disruptive emphysema. The increased respiration is caused both by the accumulation of carbon dioxide and by the want of oxygen and not by the absorption of any poisonous substance from the gas. In itself, this increased breathing does no harm and it helps in eliminating CO_2 and increasing the intake of oxygen which are the chief needs of the moment.

Interference with circulation.—The ordinary phenomena in asphyxia of mechanical origin are that the blood pressure rises and that the heart soon loses its full driving power because its muscle can not maintain this increased effort when it is working with a scanty supply of oxygen. Consequently the pulse rate quickens, the right heart dilates and the blood tends to be pooled up behind it in the great veins. If this failure proceeds apace, a patient who at the beginning showed congestive cyanosis of the face with a full pulse will gradually assume a gray pallor while the pulse accelerates and falls off in power. These changes are present in cloud gas poisoning and they are augmented by the edema of the lungs, which directly obstructs the pulmonary circulation and causes an earlier failure of the right side of the heart.

If the patient during this critical time tries to carry on his work and remain standing, he will use up still more rapidly the little oxygen that he is receiving, extra work will be loaded on to a heart which is already overstrained and the circulation will be likely to fail still more speedily on account of the difficulty of maintaining compensation in the upright posture.

There is no interference with the respiratory properties of the blood which can at once take up any oxygen that reaches it through the lungs.

General toxic effects.—Experiments indicate that chlorine is not absorbed from the lungs and that the effects produced by this gas are only a direct and local inflammation with secondary results which are either of a mechanical nature or caused by oxygen want, or due to a nervous reflex from the seat of injury. Changes of a general nature do undeniably appear in the nervous, circulatory, alimentary, and renal systems of a man who has been poisoned by cloud gas, but these are probably the result of the asphyxia rather than of the direct action of the gas. Thus in the mild cases there is a sense of fatigue and of being altogether done up, and in the serious cases even unconsciousness, but these features are not in excess of what may result from oxygen want. The retching and vomiting that generally occur in the first stages of poisoning may be due only to direct irritation of the back of the throat and of the stomach by the gas, or may be the direct sequence of violent bouts of coughing;, while the diarrhea, which sometimes is an early feature, may be that of emotion.

Microscopic examination of the kidneys in death, a few days after gassing, reveals very definite inflammatory and destructive changes, and such kidneys may be swollen and enlarged as though with a parenchymatous nephritis. This change, however produced, rarely leads to any clinical feature of renal trouble. In the first few days the urine contains neither sugar nor albumen nor many casts, and it is very unusual for albuminuria to develop later.

The circulation may fail with unexpected rapidity and the patient soon present the aspect of a leaden gray cyanosis. But in general terms the clinical picture with chlorine may be summed up as that of a man suffering from intense irritation of the respiratory tract and dying by asphyxia from the fluid that has drowned his lungs. It does not suggest a deeper toxic action.

Post-mortem findings.—In a case of death at 24 hours after gassing, the trachea and bronchi are purple red and congested, while a thin serous exudate wells up into them from the lungs. The latter organs are heavy and edematous, while aerated islets of emphysematous overdistention alternate with depressed purple patches of collapse. On section, serous fluid drips abundantly from the lung tissue. Air that has escaped from ruptured vesicles is seen in chains of bubbles on the surface of the lungs, along the interlobar fissure and even penetrating the tissues of the mediastinum. In some of the earliest cases the most intense disruptive emphysema was observed, destroying the air sacs and interfering with the circulation in their walls.

Petechial hemorrhages appear on the surface of the lungs, on the heart, and also on the inner surface of the stomach. All the veins are greatly distended and the abdominal viscera are engorged with dark blood that clots very early after death. The heart itself may fail to show right-sided dilatation, for this does not of necessity appear post mortem in cases of asphyxial death.

If the man succumbs at a later date, inflammatory complications appear in the lungs. There is superficial pleurisy, scattered broncho-pneumonia, and a purulent secretion in the bronchi. The serous exudate will then be found to have disappeared and no fluid drips from the cut surface of the lungs.

CLOUD GAS OF 1916.

The symptoms tend to differ from those produced by chlorine in three main details.

(1) Subjective respiratory irritation is much less in evidence. The men do not suffer with such violent coughing when first exposed to the gas. There is consequently less disruptive emphysema of the lungs, and subcutaneous emphysema of the neck is rare, while post-mortem examination in early cases often fails to find anything more than a little escape of air along the inter lobar fissure.

(2) The poisonous effects may appear speedily with cyanotic asphyxia, but sometimes they are more insidious in their onset. A man may feel able to carry on his work for an hour or two with only trivial symptoms; then he rapidly becomes worse and passes into a state of grayish white collapse, with progressive edema of the lungs that may soon be fatal. It has even been reported from the trenches that men who have passed through a gas attack and seemed to have suffered but slightly have died abruptly some hours later upon attempting some bodily effort.

(3) Features suggestive of general collapse are more in evidence. There is a much greater tendency to circulatory failure. Many of the cases that die on the first day show a leaden gray tint of the face rather than a purple red cyanosis; the pulse is rapid and of poor tension. Mental confusion or mild delirium with restlessness and unconsciousness become more prominent in the severe cases.

No case has been reported in the armies of death immediately upon exposure to the gas, although men have gone through an attack of gas in high concentration without wearing a mask at all and succumbed in consequence an hour or more later.

The action of phosgene ($COCl_2$) differs from that of chlorine in certain respects. Upon meeting moist surfaces it is broken up and hydrochloric acid is liberated. It excites less spasm than does chlorine in the upper respiratory tract, and so can penetrate to the innermost recesses of the lungs, where it causes an irritant edema which may be a little delayed in its development, like that caused by the acids formed from nitrous fumes, although the delay in the latter case is more prolonged. Experiments do not show that the products of phosgene are absorbed from the lung and act as general poisons apart from their local action.

POST-MORTEM FINDINGS.

Two hours after exposure to gas.—In a case of death by asphyxial cyanosis where inflammatory effusion had developed with great rapidity the lungs were smaller than normal, heavy, uniformly airless and purple, so that each resembled a big spleen. There was no disruptive emphysema. Thin serous fluid ran abundantly from the surface when cut across. Each pleural cavity contained about 15 ounces of serous effusion.

Second and third day.—There is no striking difference between the cases which had shown pallid collapse and those which succumbed in extreme cyanosis. The condition of the heart is found to be variable, but there is always evidence in the viscera of vascular engorgement from failure of circulation. The lungs never show voluminous emphysema, and indeed the earlier the death the greater is the serous edema in their substance. On the second day the fluid does not drip quite so freely from the cut surface as on the first, and toward the beginning of the third day the general aeration of the lung is everywhere greater, while relatively large islets of well-aerated and slightly emphysematous lung may be present between areas of edema or of collapse. This aerated condition appears at first in the lower lobes of the lungs where they are in contact with the diaphragm, while edema persists longest and is most profuse in the upper lobes.

A day later and no serous fluid at all escapes from the cut surface. At this date inflammatory complications tend to appear in a surface pleurisy, and areas of the lung are found to be slightly friable and entering into a condition of broncho-pneumonia.

It may be that cases of extensive and overwhelming edema succumb at once, while those in which on the second or third day larger islets of aerated lung alternating with edematous patches are seen, had from the beginning been in that state, so that they

succumbed later than the completely edematous group because the injury to the lung was less. But the general evidence favors a more hopeful view, namely, that the edema fluid is rapidly absorbed from the second day onward, and that the later post mortems illustrate the stages in this recovery. The chief fact in support of this view is that patients who had been deeply cyanosed at first, with the usual signs of extensive pulmonary edema, and so asphyxiated as to be unconscious for a couple of days, may yet recover so completely that eight or nine days after exposure to gas it is difficult to discover any physical signs of edema in the lung.

SYMPTOMS.

Onset.—Upon exposure to chlorine alone, a man feels immediate respiratory distress. He coughs violently and speech is made impossible by his spluttering gasps. With the later forms of drift gas, the onset is slightly altered. There is some lachrymation. The throat feels gripped and the chest tight. Breathing is difficult but not impossible. Coughing develops a quarter of an hour or more later. Nausea and vomiting appear quickly, so that a man who was slow in getting protection may vomit inside his gas mask. Headache and throbbing sensations in the body are experienced.

Development.—Coughing and retching increase. The respiration becomes very hurried and labored, though shallow. The patient's face assumes a cyanotic hue; he may lose muscular power and consciousness and die in an hour or two. Those who survive longer show the following features:

Headache, pain behind the sternum and in the epigastrium. Extreme restlessness and anxiety, or a semicoma with a muttering delirium, from which as a rule they can be roused to answer questions. Varying cough, sometimes slight, sometimes reiterant with a croupous rattling from exudate in the trachea. There is practically no laryngitis. A cyanotic blueness in the lips and ears, which may accompany a flushed lividity of the face or the grayish-yellow pallor of collapse. Extremely rapid respiration, from 40 up to even 80 a minute, of a shallow type on a distended chest, and often marked by a jerking grunt of expiration. A pulse of about 100, which may rise to a higher rate and fall to a very low pressure in the gray examples of collapse. The skin is dry, and either hot or cold in correspondence with the state of collapse. Expectoration may be very slight, though in others there soon develops an abundant discharge of thin watery fluid, often streaked with blood, which simply flows from the mouth as the dying patient loses power

to expel it. After death, the foam from this fluid may dry to a white efflorescence around the mouth. The percussion note is slightly flattened over the lungs behind, where the breath sounds are much weakened, but otherwise unchanged in quality. Fine rales are heard behind and in the axillæ. There are no tubular breath sounds. In front there may be extremely little change beyond harshness in the breath sounds. The physical signs fail altogether to indicate the extent to which the lungs are damaged, for in any area examined there is always some aeration of the bronchioles and alveoli which suffices to produce relatively normal sounds on auscultation.

The color, the pulse, and the rate of respiration are the chief guides to prognosis.

Progress.—Four-fifths of the deaths occur in the first 24 hours. Very few succumb after the third day. A man, who at first seemed to be lightly gassed, may, toward the end of the first day, develop cyanosis and die; but from the end of the second day onward, there is no danger to be apprehended for the less grave cases. On the second day the sputum becomes less abundant, more viscous and yellow tinted. The dyspnea persists and the temperature is raised. If complications develop subsequently from infections of the raw respiratory tract, they will be shown by persistence of fever, by a purulent sputum, and by signs of broncho-pneumonic consolidation.

But as a rule the patient recovers rapidly after the third day, and at the end of a week he is fully convalescent. Cough, pain in the chest, which is often very severe beneath the rib margins, shortness of breath, loss of appetite with gastric pain, and general lassitude persist longest of the symptoms. There are no serious after results to be apprehended. A man who has been badly gassed requires a long rest; but the majority, if free from neurasthenic symptoms, are fit for light duty in a very few weeks, provided that they are allowed sufficient rest at first. The heart and circulation are severely strained by gas poisoning. Convalescents who show tachycardia must be carefully watched lest too heavy physical effort early in the first month of recovery induces further strain and lead to the condition of irritable soldier's heart, from which recovery will be long delayed.

RARITIES.

Brain.—In some cases that died after two or three days of persistent cyanosis and unconsciousness the white matter of the brain was found to be peppered with tiny petechial hemorrhages. These are the direct outcome of the asphyxial state and have little clinical

significance. Large cerebral hemorrhages have, however, been noted, occurring on the first or second day in cases of plethoric cyanosis.

Vascular obstruction.—Occasionally the peripheral arteries to the limbs may become occluded. As a rule, the threatening gangrene clears up in these cases.

Stomach.—Petechial hemorrhages and a slight superficial ulceration are often seen post mortem over the inner surface of the cardiac fundus. The stomach has occasionally at autopsy been found to be full of blood from an extension of this ulcerative process, but only in one case has death occurred with hematemesis. The gastric derangements, which generally persist during convalescence, are not of a type suggesting the result of an ulcerative process.

Kidney.—Clinical nephritis is rare, and its occurrence may in each case have been a chance coincidence.

TREATMENT.

GENERAL CONSIDERATIONS.

REST IS THE MOST IMPORTANT POINT OF ALL IN THE GENERAL TREATMENT OF GAS CASUALTIES.

Men, and especially officers, should be warned beforehand that if lightly gassed they must refrain from moving about or shouting out orders. Physical strain after gassing may easily involve the loss of a life that might otherwise have been restored to the fighting line in a short time. The principle of attaining complete rest as soon as possible underlies the detailed advice for dealing with gas casualties that is given below, and is the reason for their detention at the casualty clearing stations. All kit that hinders the play of the respiratory muscles, especially belts and suspenders, should be undone. Sleep brings improvement, and restless excited cases should be quieted by morphia. It is important that arrangements should be planned beforehand at each casualty clearing station so that even a large number of gas casualties can be handled with such discipline and control as will at once introduce a sense of order and quietude, and by separating those who are more dangerously ill from the remainder, enable the less severe cases to get to sleep at once.

Next in importance to rest comes the use of oxygen, protection from cold, special stimulants or drugs, venesection, and methods for removing serous exudate from the lungs.

Bronchial spasm does not seem to be a serious danger with the present form of cloud gas. Life or death is decided by the degree

of pulmonary edema and asphyxia with circulatory failure. The edema fluid tends to be absorbed quickly, and if the patient can be carried alive through the first two days, he should recover. Precautions in the meantime need to be taken to lessen the chance that secondary respiratory infections may develop as a later complication.

Oxygen, if rightly administered, will generally lessen cyanosis, and therefore improve the patient's chance of life. But the lung surface available for absorption is so small that the oxygen must be given in high concentration. The simple admixture with air obtained by open flow from a funnel or a tube placed in the patient's mouth is useless, and since it wastes valuable oxygen it should be forbidden. Given as the pure gas from a bag with a valved face mask, as described in Treatment at Casualty Clearing Stations (see p. 22), say for 3 or 4 minutes every quarter of an hour, an oxygen cylinder of 20 feet capacity will last about 4 hours. By this means life can undoubtedly be saved in some of the apparently desperate cases. The administration must be continued night and day, so as to hold cyanosis in check. The consumption of oxygen by this method is so large that all care must be taken to economize cylinders, the provision of which, under active service conditions is necessarily limited by considerations of transport. Many casualties are so severely poisoned that their condition is seen in the first few hours to be hopeless. Some selection of the cases for oxygen treatment must therefore generally be made, and it is especially with the intermediate group who are surviving into the second day that oxygen has the best chance of acting with ultimate advantage. It is quite unnecessary to use it for relatively mild cases. Deep cyanosis, whether of the congestive or pallid type, is the indication of need, and the lividity can always almost be lessened if the face-mask is properly applied.

The subcutaneous injection of oxygen is useless.

Warmth is needed in many of the severe cases, especially at the outset when they are collapsed and their skin is cold. To attain this, it may even be necessary to transfer them temporarily indoors. Otherwise the fullest supply of fresh air is needed for all cases. When outside, care should be taken to protect against chill, which might lead to a subsequent broncho-pneumonia.

Special stimulants and drugs.—Ammonia is very useful as an inhalation from the small ampoules supplied. It is necessary to warn orderlies that these ampoules are to be held at some distance from the patient's mouth, and that their use should not be persisted in

where they seem to aggravate the difficulty of breathing. The action is more as a stimulant than in chemical antagonism to the chlorine.

Brandy and water is a good restorative when given in small sips. Cases of gray collapse with a rapid pulse of poor tension do not react well to stimulant drugs and are almost hopeless when they develop this condition in the first day. Pituitrin, 0.5 cc. hypodermically every three hours, gives some aid to a failing heart. The benefit of digitalin and strychnine is more doubtful.

Venesection gives real relief to men with deep cyanosis and a full pulse. The headache is lessened, the breathing feels easier, and the patient may soon fall into a sleep that conserves his strength. It appears to be harmful in collapsed cases with a poor pulse, and it should not be postponed until the patient passes into this dangerous state.

Methods for aiding the discharge of exudate from the lungs.—(1) Emetics have been given at various times in the hope that the lungs would be partly emptied in the act of vomiting. The effort is very exhausting and the results do not justify the treatment in late cases. But in the first few hours, before the patient is gravely ill, vomiting is probably good and retching may be encouraged by simple means. Later, it is better to try and allay gastric pain or retching by the use of drinks with sodium bicarbonate. Beef tea is not to be recommended in view of the possibility of slight gastric ulceration.

(2) In the first day, if there is much fluid expectoration, good results may be obtained by postural treatment, such as by turning the head of the patient sideways and then raising the foot end of the stretcher two or three feet, or even higher, for a few minutes at a time.

(3) Schafer's artificial respiration has occasionally proved of service in expelling fluid from the chest, but it is necessary to watch its effect on the patient very closely lest disaster ensue.

(4) Expectorants in large doses, such as 10 or 15 grains of ammonium carbonate, are probably harmful at the beginning, for they will cause nausea and an irritant cough which will interfere with sleep and may augment the tendency to disruptive emphysema of the lungs. Patients who are not seriously ill should be given a simple mixture such as amm. carb. gr. v and vin. ipecac. m. x. 6 hourly on the second day. Later treatment might comprise ordinary medicinal measures such as potassium iodide, atropine, steam tents with tincture benzoin compound, etc., for symptoms as they arise.

Regulations for Treatment of Gassed Cases.

TREATMENT BY REGIMENTAL MEDICAL OFFICERS.

1. All ranks should be warned of the need for seeing that the gas masks of wounded men are kept properly in position until the danger from gas has passed away, and also of the importance to lightly gassed cases of remaining absolutely quiet.

While the gas cloud is concentrated, all ranks should refrain as far as possible from movement, so that they may breathe slowly and keep the current of air through the gas mask at a low velocity.

2. After the order has been given for removal of masks, ammonia inhalations from the capsules should be given by stretcher bearers to all gassed men with difficulty in breathing, but their use should not be persisted with in those special cases where the ammonia seems to increase the discomfort of the patient.

3. Clothing over the chest should be loosened, the suspenders and belt undone in front and equipment removed.

4. Vomiting at first is beneficial and it may be encouraged by drinks of tepid water, either alone or with salt (1 tablespoonful to half a pint or more of water), and the back of the throat should be tickled shortly after the drink has been taken.

5. Experience has shown that atropine is of no use in the early stages of gas poisoning.

6. All gas casualties must be evacuated as soon as possible to dressing stations. All except the lightest cases should, as far as possible, be evacuated lying down, and walking cases should be warned to seek assistance in going back along the trenches so as to avoid physical effort as much as possible. Special attention should be paid to men who complain of feeling collapsed, though they show no manifest features of having been gassed, since these may develop later the serious form of delayed poisoning.

Rest is the one point that is essential from beginning to end of the treatment of gas casualties. Gassed men should not be allowed to carry their own equipment back from the trenches. It is suggested that arrangements for the disposal of kit should be made regimentally in advance.

TREATMENT IN DRESSING STATIONS.

1. Casualties should be kept lying down in the open air, so far as weather permits. Suspenders must be undone and clothing over the chest loosened, so as to give freedom for breathing.

2. Ammonia inhalations should be given. Experience has shown that atropine injections are of very doubtful value.

3. If the patient still feels inclined to vomit, he may be given tepid drafts of salt and water, and the back of the throat tickled.

4. Restless cases, if fully conscious, may be given one quietening injection of one-fourth grain morphia. The time of injection and the dose must be recorded on the diagnosis tag.

5. Severe cases fall into two groups: The "collapsed" with a poor pulse and leaden gray tint, and the "cyanotic" or "blue" with a full pulse and general asphyxial lividity.

6. Collapsed cases may be retained at the dressing station for a short time, and their state of collapse combated by stimulants such as external warmth, hot drinks, brandy, pituitary extract, or other drugs. If there is much fluid flowing up through the mouth, the feet of the stretcher should be raised to a height of about 4 feet and the patient's head turned slightly sideways. This position should not be maintained for more than a few minutes and it should be abandoned if it aggravates the cyanosis or fails to increase the discharge of fluid.

7. Blue cases of severe cyanosis require continuous treatment with oxygen in high concentration. They should therefore be evacuated as soon as possible to the nearest casualty clearing station or field hospital, unless special apparatus for their treatment is available at the dressing station. If there is unavoidable delay before transference the treatment recommended at the casualty clearing station, paragraphs 7 and 8, should be tried.

8. All cases except the mildest should be evacuated lying down, and the severe cases, with the exception of those of grave collapse, should be transferred first of all.

TREATMENT AT FIELD HOSPITALS AND CASUALTY CLEARING STATIONS.

1. Serious cases should at once be separated from the slight, so that the latter may lose their anxiety and get to sleep.

2. Ammonia inhalations may be useful on admission at any time up to 24 hours after exposure to gas.

3. Open air treatment is best, if the weather permits, and complete rest is essential for the first two days. The clothing and suspenders should be loosened, and the worst cases should be undressed and put to bed wherever this is possible.

No patient should be allowed to leave his bed or stretcher for any purpose whatsoever.

4. Restless cases, if not unconscious, may be quieted by one injection of one-quarter grain morphia.

5. Collapsed cases should be treated with warmth and stimulants as described in dressing station 6, and oxygen should be used in addition. Atropine is harmful to these patients.

6. Attention should be paid to the posture of unconscious cases, both to aid discharge of any fluid that may be escaping from the lungs and to avoid injury to nerves or skin by pressure, e. g., from a clasp knife or the edge of the stretcher, if the patient is not undressed and in bed.

7. Cyanosis can be lessened by the administration of oxygen in high concentration. For this purpose the flow from a simple funnel or a tube in the mouth is inadequate and wasteful of the gas. An apparatus should be used with a valved face mask, such as that for nitrous oxide anesthesia, the valves being so set that the patient breathes in oxygen from the bag and breathes out into the open air. The treatment should be continued hour after hour with brief intervals until the edema of the lungs clears up. Cases may recover after being cyanosed and unconscious for two days.

8. Venesection, performed slowly, to the amount of 15 or 20 ounces may be of use to "blue" cases if practiced early and before the pulse begins to fall.

9. *Expectorants.*—Drugs such as ammonium carbonate and vinum ipecac are recommended for use in expectorant doses from the second day onward.

10. The diet should be light and simple. Measures should be taken to open the bowels.

11. As far as circumstances permit, no case should be evacuated to the line of communication until definite cyanosis or serious symptoms have disappeared. A note should be sent down with those cases which have passed through a condition of gravity, so as to guide the medical officer on the line of communication as to the future treatment of the case.

LINES OF COMMUNICATION.

The methods of treatment advised at casualty clearing stations are applicable for use in hospitals on the line of communication when circumstances necessitate the early evacuation of cases.

A very large proportion of the casualties sent to the base as "gassed cases" are likely to become fit for duty after a short rest. Wastage by transfer to home territory is to be avoided, and medical officers should exercise the greatest care in selecting cases for evacuation to home hospitals.

As a rule, only cases in which there was a clear history of grave cyanosis or collapse, or in which secondary infections of the respiratory tract have developed, should be sent farther than the base. It may be taken for granted that all casualties which had been detained in a casualty clearing station for five days were of a serious nature, whatever their condition on arrival at the base hospital.

Rest stations.—Rest, with the possibility of lying down on a bed at any time of the day, must be provided during the first two or three weeks for all cases except the very mildest. Arrangements should, when possible, be made at the base to give accommodation, if the patients can not be retained in the main hospitals, for an easy life of this nature which is not permitted in the usual routine of convalescent depots.

Convalescent depots.—Gas casualties, who show neurasthenic or cardio-vascular weakness after three weeks' treatment at the depot, should be transferred to home hospitals.

INDEX TO DRUGS WHICH HAVE AT VARIOUS TIMES BEEN USED.

Ammonia.—The benefit of occasional inhalations of ammonia during the first day is universally admitted. Spiritus ammon. aromat. in 4 cc. doses with plenty of water is useful as a direct stimulant, but it does not ease breathing in the same way as does vapor.

Ammonium carbonate.—In large doses of gr. xv as an emetic has proved harmful and never done good in the early stages. As an expectorant it should only be used in small doses of gr. v.

Apomorphine.—In hypodermic doses of gr. $\frac{1}{10}$ has been recommended as an emetic. The vomiting often fails, the drug is depressant, and its use had never proved to be of the least value.

Atropine has been supposed to be useful in the early stages of poisoning, both to relieve bronchial spasm and to check the secretion of edema fluid. The drug has been extensively tried, both in the French and British medical services, and no conclusive evidence of such benefit has been found. On the other hand, atropine does tend to accelerate the heart beat, and a result of this nature is an actual disadvantage to an asphyxiated patient. Its use in the first two or three days should therefore be discontinued.

Camphor in conjunction with ether forms a useful stimulant in cases of collapse. The Japanese in the Russian War used as a general stimulant a hypodermic injection of 10 or 15 minims of camphor 1 part, ether 4–5 parts, olive oil 4 parts. It is possible that this might also be of value in cases of gas poisoning associated with collapse.

Digitalin, grain 1/100, hypodermically, has so slight an action that it probably is not worth using in the two or three critical days of acute pulmonary edema.

Ipecacuanha has been recommended as having a specific action in promoting the absorption of edema fluid. For this purpose, 20 to 30 minims of the Vinum have been given two-hourly. Nausea and emesis are not aimed at. The evidence as to its action in this sense is inconclusive, though it is useful in smaller doses as an ordinary expectorant. The drug should not be given as an emetic, because it produces depression.

The extractive, *Emetine*, has been tried hypodermically in repeated doses of gr. $\frac{1}{3}$ and without any apparent benefit whatsoever.

Morphia is most useful as a sedative and may be given even to deeply cyanosed patients for extreme restlessness. The dose should not be large, gr. $\frac{1}{4}$, followed, if need be by gr. $\frac{1}{6}$, or 15 to 20 minims of tinct. opii.

Phenacetin and *Aspirin* have been given to relieve the headache caused by mine-gas poisoning, and they were found to produce a dangerous state of collapse in these patients. It is therefore inadvisable to use them for the headache that follows the inhalation of cloud gas.

Pituitrin 0·5 cc. into the muscles or hypodermically increases cardio-vascular tone for a short time and gives aid to a failing heart. If a second injection is given 15 to 20 minutes after the first, it may cause the pressure to fall. An interval of three hours should be observed if the dose is repeated.

Sodium lactate taken by the mouth every four hours in 2 drachm doses of a 50 per cent solution has been employed with a view to augmenting the alkaline bases of the blood, fixing some of the excess of carbon dioxide, and thereby tending to lessen the rate of respiration. The main features of gas poisoning are not influenced by this method of treatment.

Sparteine has erroneously been supposed to be a cardiac tonic and a useful drug in the place of digitalin. Actually it is a poison which slows and weakens the heartbeat.

Strychnine, one-thirtieth grain, may be employed in the early stages of pallid collapse.

Nitrous Fumes.

Though no cases of nitrous fumes poisoning have hitherto been recorded, the possibility of their occurrence owing to exposure to the fumes of burning explosives should not be lost sight of.

The great danger of nitrous fumes arises from the fact that in the concentrations usually met with there is comparatively little irritation of the eyes or upper respiratory passages, and a man working in such an atmosphere will not recognize its deadly nature. Air which contains enough nitrous fumes to cause feelings of irritation in the nose or air passages must be regarded as very dangerous. Nitrous fumes are very soluble in water, and the gas may be readily removed from the atmosphere by means of a water spray, whilst a few folds of a handkerchief or a towel wetted with water and tied over the nose and mouth will give efficient protection in the absence of a mask. The possibility of the simultaneous occurrence of carbon monoxide in atmospheres containing nitrous fumes must be remembered.

Owing to the delay in the onset of serious symptoms, it is essential that any man who is suspected of having been exposed to nitrous fumes should be sent to hospital with as little delay as possible and detained there for 24 hours for observation.

Pathological changes.—The pathological changes found post-mortem in a fatal case of nitrous-fumes poisoning are identical with those described above as characteristic of chlorine poisoning. If the concentration of nitrous fumes to which the case has been exposed is very high, the blood may be somewhat chocolate colored owing to the formation of methaemoglobin.

Symptoms.—If the gas is in very great concentration, rapid fatal asphyxiation takes place, but in the concentrations that are usually encountered, the characteristics which distinguish this from chlorine poisoning are the slightness of the initial symptoms due to irritation of the upper respiratory passages and delay in the onset of acute pulmonary edema.

The typical sequence of events is—

(1) Slight irritation of the nose and throat, feeling of constriction of the chest, headache and slight smarting of the eyes and coughing while actually exposed to the fumes.

(2) On leaving the poisonous atmosphere a latent period during which the case may, and usually does, feel quite well and has no hesitation in taking a meal.

(3) The sudden onset after four to eight hours of acute symptoms. These commence with marked and increasing distress in breathing, coughing, and often severe pain in the chest. The cough is at first dry, and auscultation may reveal no moist sounds. This condition is speedily followed by the urgent signs of acute pulmonary edema which have been already detailed under chlorine poisoning (p. 13), and death may ensue in a few hours.

Treatment.—When once pulmonary edema has developed, the treatment should follow the lines already laid down for chlorine poisoning (p. 18). The experience of medical officers attached to mines on the Rand, where nitrous fumes are frequently met with during blasting operations, points to the value of inducing emesis as soon after exposure to the fumes as possible, followed by a dose of such a stimulant as spir. ammon. aromat. A case of nitrous-fumes poisoning should be under medical observation at the time when acute pulmonary edema is likely to develop and a venesection of from 15 to 20 ounces should be made as soon as there is the slightest sign of its onset. Venesection must not be delayed until the patient's condition is grave and the stage of lividity has been reached, or it will be useless.

LACHRYMATORS.

Hitherto, no serious cases of poisoning have occurred as the result of exposure to the effects of shells containing substances employed merely on account of their lachrymatory properties.

The immediate effect of a trace of the vapor of such a lachrymator as benzyl bromide in the air is to cause profuse watering of the eyes, accompanied by smarting. If the concentration is somewhat greater, the smarting and pain in the eyes may become intolerable, so that it is impossible to keep the eyes open. The smarting and watering of the eyes will be quite sufficient to put a man completely out of action, because he is incapable of seeing, but protection of the eyes is easily obtained by the use of goggles.

With increasing concentrations of the vapor, other effects show themselves. The vapor is irritant to the lungs and upper respiratory passages and this leads to a burning sensation in the throat and coughing. Nausea is often present and not infrequently leads to vomiting, accompanied, it may be, by pain in the epigastrium. If it is impossible to withdraw from exposure to the fumes, slight confusion of mind and torpor may show themselves.

Under ordinary conditions the symptoms do not develop further, and though the case may become somewhat collapsed as a result of the vomiting and general discomfort, this is only temporary, and within an hour or two after getting into air free from the lachrymator there may be very little amiss with the man. The nausea and irritation of the throat soon pass off, though the eyes may remain sore for some little time, and even after the lapse of 12 hours redness of the eyelids and slight injection of the conjunctiva may still be evident. There are no subsequent toxic effects and the case will be fit for duty as soon as the primary effects have passed off.

It must not be forgotten that some of the acute lung irritants are also extremely powerful lachrymators, and that such substances may be used with a view to securing a double effect, viz, immediate blinding and simultaneous intense toxic effect on the lungs. In order to secure such an effect it is essential that the substance used shall be gaseous or shall vaporize with sufficient rapidity to attain a high enough concentration in the air to produce these intense toxic effects. Lachrymators such as benzyl bromide, which are liquid at ordinary temperatures, vaporize too slowly to produce such a concentration, and the symptoms caused by a lachrymator of this type, therefore, very rarely attain a degree of severity greater than those described above in detail.

The smell of benzyl bromide when in great dilution suggests the flavor of mustard and cress. Lachrymators as a rule have aromatic, pungent odors.

HYDROCYANIC ACID.

When in sufficient concentration this gas acts as a very rapid and sometimes almost instantaneous poison, affecting directly the central nervous system.

Symptoms.—These follow one another in rapid sequence: Giddiness, confusion, headache, indistinct sight, palpitation and pain in the chest and over the heart. Labored respiration. Unconsciousness, convulsions, failure of the respiration and finally of the heart. In large doses, immediate unconsciousness, dilatation of the pupils, a few gasping respirations, and death with or without convulsions.

The gas paralyzes the respiratory center very quickly, and with small fatal percentages the heart may continue to beat for a brief time after the respiration has ceased. With larger concentrations the heart may be stopped almost at once by the direct action of the poison.

When death is caused by inhalation of hydrocyanic-acid gas, it is unlikely that the smell of the gas will be detected at autopsy, as may be the case when poisoning is due to the ingestion by the mouth of a large dose of prussic acid.

Treatment.—Immediate treatment is the only measure of any avail if a man falls unconscious from hydrocyanic-acid poisoning. The case must be at once dragged into fresh air, and if the respiration has stopped, or is very weak and gasping, artificial respiration must be instantly applied by Schafer's method. It is possible by this means to resuscitate a case, and if this is so recovery will be perfect. Cold water may be splashed on the face and chest and friction applied to the limbs, but time should not be wasted on these meas-

ures before commencing artificial respiration. The same immediate treatment holds good for any other gas that causes rapid unconsciousness from its effects on the central nervous system.

CARBON MONOXIDE.

The great danger of carbon monoxide arises from the fact that the gas is colorless, odorless, and nonirritant, and that the onset of symptoms is so insidious that very often the first warning that a man may receive is failure in the power of his limbs which will prevent him from retreating into safety. Neither the box respirator nor other masks give protection against carbon monoxide; protection can only be attained by the use of special oxygen breathing apparatus.

Pathological changes.—At the autopsy, the blood may be red in color instead of dark if there is a considerable degree of saturation of the hemoglobin with carbon monoxide. If the case has continued to breathe for some time after reaching an atmosphere free from carbon monoxide, this gas will have been partly or entirely displaced from the hemoglobin and the blood after death will have its normal color.

The simplest method of detecting the presence of carbon monoxide in blood is to compare the color of a dilute solution of the suspected blood with a similar solution of normal blood. Take a drop or two of blood from the finger of a normal person and dilute it in a test tube very considerably with water (a one-half of 1 per cent solution is a convenient strength), so that when examined by transmitted daylight the color of this solution is a reddish-yellow. Then take a drop or two of the suspected blood and dilute it similarly with water, so that the depth of color of the solution is the same as that of the solution of normal blood when both are viewed by transmitted light. On examining the quality of the color it will be found that the solution made with the suspected blood, if it contains carbon monoxide hemoglobin, is definitely pinker than that made with the normal blood, though it will not have the full pink tint of the same normal blood solution if the latter be shaken with coal gas so as to saturate it quite completely with carbon monoxide.

The lungs show no abnormal changes in cases of rapid death. Small punctate hemorrhages may be found in the white matter of the brain and sometimes ecchymoses in the meninges if the case has been exposed to a concentration of carbon monoxide sufficient to cause prolonged unconsciousness.

Symptoms.—Except with very massive doses, when loss of consciousness is very rapid, the symptoms develop very gradually, as the gas is only absorbed slowly. If a man is at rest in a concentration of the gas of 1 part in 1,000 it will take about two hours before definite giddiness appears and he will not be definitely disabled until the lapse of two and one-half hours. The rate of absorption of the gas is much quickened when the breathing is deepened during muscular exercise and the exercise also leads to great accentuation of the symptoms. With a concentration of 2 parts in 1,000 a man will be seriously affected in half an hour if he is performing a moderate amount of muscular work, and this concentration may prove fatal with prolonged exposure.

Small animals are far more quickly affected by carbon monoxide than man is, owing to the natural great ventilation of their lungs and the rapidity of their circulation. A mouse or a canary will show definite symptoms of carbon monoxide poisoning in a tenth of the time that a man will. If small animals are used to give an index of the presence of carbon monoxide in a suspected atmosphere, it must be remembered that though they show symptoms long before a man feels any effects, the man will in the end be reduced to the same condition as the animal, and he ought therefore to leave the dangerous atmosphere directly the animal shows signs of being affected, unless he is protected by special apparatus.

The first sign that tells a man that something is amiss is very frequently a feeling of loss of power in the limbs. Giddiness, slight confusion of mind, and breathlessness and palpitation on the least exertion also show themselves. The confusion of mind and loss of power in the legs frequently preclude a man from withdrawing from danger, even though he is dimly aware that safety is only a few yards distant. The failure of power in the limbs and mental confusion rapidly increase and the man may appear drunk, shouting incoherently, laughing, swearing, or praying. Apathy and complete helplessness supervene, and failure of the intellectual powers gradually passes into complete unconsciousness, which may finally terminate in a painless death.

The symptoms may remain stationary at any stage, since the degree of saturation of the hemoglobin with carbon monoxide reaches a final end point which is determined by the relative concentrations of the carbon monoxide and the oxygen which are simultaneously trying to combine with the hemoglobin.

Even in cases of mild gassing with carbon monoxide, a severe headache accompanied by nausea is very likely to develop.

Treatment.—The symptoms detailed above are due to the gradual diminution of the oxygen-carrying power of the blood and the exposure of all the organs of the body to increasing want of oxygen. It is clear that any increase in the oxygen demands of the body is to be avoided, and any man, therefore, who shows definite signs of gassing should be carried to a place of safety. If he attempts to walk himself he is quite likely to fall down unconscious. When a moderately gassed case reaches fresh air he sometimes falls unconscious, while other cases may commence to shout and struggle, in which case their movements need to be controlled.

Any case showing definite symptoms should be removed as soon as possible to some place of safety where he can remain at rest for an hour or two before evacuation. Rest is essential.

As carbon monoxide hemoglobin is a dissociable compound, the carbon monoxide is gradually driven out of its combination with hemoglobin by the oxygen of the air as soon as an atmosphere free from carbon monoxide is reached. In fresh air it will take an hour or two before the blood is entirely freed from carbon monoxide, but the process can be rendered five times as rapid by giving the patient pure oxygen to breathe. It is important therefore to begin the administration of oxygen by some efficient method as soon as possible after the case has been removed from the poisonous atmosphere. A suitable method has been described under chlorine poisoning (p. 19) and the oxygen apparatus that is kept at mine rescue stations is also most efficient. Administration of oxygen should be kept up as continuously as possible for half an hour to an hour, depending on the severity of the symptoms. It should be remembered that if a case can be kept at rest for half an hour and oxygen administered immediately after being removed from the poisonous atmosphere, he will be in far better condition to travel than if he has to be removed to a more distant point. If the breathing is very shallow, administration of oxygen may be combined with artificial respiration.

Collapse should be combated by external warmth and by friction of the limbs.

In chlorine poisoning the pulmonary edema and damage to the lungs and the consequent interference with the gaseous exchange taking place between the blood and the air in the lungs persist for some time and may necessitate the administration of oxygen for several days. In carbon monoxide poisoning the structure of the lungs is not interfered with and oxygen is administered with the deliberate intention of accelerating the discharge of carbon mon-

oxide from the blood. When once this has been accomplished, i. e., after half an hour's or an hour's administration, there is no need to continue the oxygen administration, as the oxygen-carrying power of the blood has now become normal again. Any symptoms that persist are due to effects that were produced while the blood was charged with carbon monoxide and are unlikely to be influenced by oxygen administration when once the carbon monoxide has been got rid of. Further oxygen administration is therefore required only if cyanosis begins to develop subsequently from secondary cardiac or respiratory failure.

Cases of carbon monoxide poisoning have been known to recover, even when they have remained unconscious for so long as 48 hours after removal from the poisonous atmosphere. In cases that have been severely gassed the possibility of subsequent cardiac dilatation must not be lost sight of, and cases of severe gassing should not be returned to duty until confidence is felt that the circulation has recovered from the strain. As a result of damage to the nervous system while the blood was charged with carbon monoxide, paralysis of single muscles or of groups of muscles, or different forms of mental disturbance are sometimes found as sequelæ.

NEED FOR FURTHER INVESTIGATION.

Knowledge on the various points discussed in this pamphlet is still far from being stable. Medical officers are therefore asked to assist by making further observations and at once writing down notes on any of the following particulars, about which chance may enable them to supply useful information. Such notes should be forwarded to the division surgeon.

1. Smells of the gas. This may be musty, pungent, aromatic like lilac, garlic, mustard and cress, bitter almonds, and so on.

2. The symptoms in gassed men, especially those of onset, when they are in any way remarkable or differ from those described for cloud gas in this pamphlet. All points not observed by the medical officer himself should be subjected to careful cross-examination.

3. The findings at autopsy of very early cases of gas poisoning, with an account of the manner of death.

4. The value of various methods of treatment of severely gassed cases, with control observations where possible.

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