CONTENTS

AIR RAID PRECAUTIONS**

Medical Treatment of Gas Casualities*

Issued by the Home Office Air Raid Precautions Department

GENERAL DESCRIPTION OF WAR GASES

CHAPTER III

LACHRYMATORS OR "TEAR GASES"

9. Characteristics and Description of Tear Gases

The lachrymators, and the nasal irritants which are described in the next chapter, possess certain important general features:—

(1) Their action is selective, i.e., they usually only attack exposed sensory nerve endings or mucous membranes, such as those of the eye, the naso-pharynx and the respiratory tract.

(2) They are effective in extremely low concentrations.

(3) Their effects are immediate, but temporary. Withdrawal from the gas-charged atmosphere is followed by rapid recovery. Permanent disabilities rarely occur, though such cases may be experienced after an unusually heavy dosage.

There are many compounds, both liquid and solid, which may be used as lachrymators in war-time. The following are typical examples:—

(*a*) *Chlor-aceto-phenone* (*C.A.P.*).—A colourless, crystalline solid melting at 54° to 59° C. (i.e., 129.2° to 138.2° F.) and boiling at 245° C. (473° F.). Though only sparingly soluble in water, it dissolves readily in all the organic solvents. Chlor-aceto-phenone is a very stable compound which does not decompose on heating or detonation; its lachrymatory effects, however, are soon lost by reason of the condensation of the substance to the solid, inert state soon after the initial dispersion, and it is therefore classed among the non-persistent gases. This gas is used in gas chambers or vans in the course of anti-gas training. It is also the gas used by the authorities in some foreign countries for dispersing mobs.

(*b*) *Ethyl-iodo-acetate* (*K.S. K.*).—A dark brown, oily liquid with a smell resembling that of "pear drops". Its high boiling point (180° C. or 356° F.) and comparatively low vapour pressure ensure for it a certain degree of persistence on the ground.

(*c*) *Bromo-benzyl-cyanide* (*B.B.C.*).—In the pure state this is a yellowish, crystalline solid, stable at ordinary temperatures and melting at 24.8° C. (76.6° F.). In the crude form, as employed in war, B.B.C. is a heavy, oily, yellow liquid with a penetrating, bitter-sweet smell. The liquid boils ar 242° C. (467.6° F.); it is more stable, and has a lower vapour pressure than ethyl-iodo-acetate, hence it persists longer than the latter as an effective lachrymator when spread on the ground.

---

*Published by permission of the Department of Health, Government of Nova Scotia

**With this issue THE BULLETIN completes publication, except for tables, of A. R. P. Handbook No. 3.

10. Action of Lachrymators on the Body

Exposure to any of these lachrymators gives rise to an immediate, acute and localised irritation of the sensory nerve-endings on the corneal and conjunctival surfaces, which may vary from a mild irritation to an intense stinging sensation according to the strength of the concentration. Through reflex action, this is followed by profuse watering of the eyes and spasm of the eyelids, and the latter may be so acute as to render it impossible to keep the eyes open.

With a rise in the concentration of the vapour, further effects may make their appearance. The irritant action of the gas on the respiratory passages and lungs produces a burning feeling in the throat and discomfort in the chest, and, if the exposure be continued nausea and vomiting may result.

C.A.P. possesses the characteristic attribute, absent in the case of the liquid lachrymators, of irritating the bare skin, especially if the skin be hot and moist. This irritant action is not sufficiently serious to necessitate special protective garments. Persons with dry skins, however, who do not sweat, are inclined to be hypersensitive to C.A.P. and may develop an obstinate dermatitis if often exposed to the vapour.

As a rule, persons exposed to lachrymators never exhibit more severe symptoms than those described above, as the very high concentrations necessary to produce lung lesions are not met with in the open. In confined spaces, however, where accidental splashes of the actual liquid on the skin may occur, or the inhalation of a high concentration may be experienced, more severe results may be expected: these vary from severe conjunctivitis, with tracheitis and bronchitis from the effects of the vapour, to blistering of the skin, keratitis and corneal opacities after contamination with the liquid.

The rare fatalities that occurred in the last war were characterised by a severe pulmonary oedema following the accidental inhalation of massive concentrations of the gas.

The respirator affords complete protection to both eyes and lungs against all concentrations of tear gases likely to be met in war. The use of goggles alone is not recommended, as, in addition to the liability of leakage and constant dimming, they offer no protection to the respiratory tract.

For practical reasons it is important to recognise the difference between the effects produced on the eyes by "tear gases" and by "blister gases". In the case of tear gases the person affected is for the moment blinded by his own tears and by his natural instinct to close his eyes to minimise the irritation: he may require assistance to get away from the poisonous air, but that is all, for in pure air the effects will quickly pass away. In the case of a "blister gas" like mustard gas the inflammation comes on slowly, but when once it has become established it lasts for some time, and perhaps for a day or two the sufferer will be quite unable to see owing to the swelling of the eyelids and the inflammation of the surface of the eye.

11. Treatment for Lachrymators

In the great majority of cases, adjustment of the respirator will suffice to alleviate the symptoms, and usually to clear up the condition completely. Experience has shown that even after severe exposures all symptoms disappear within 12 hours. No treatment, other than simple symptomatic, is usually necessary, and no after-effects follow exposure. In the rare cases where

acute conjunctivitis or respiratory affections develop, treatment should be symptomatic and follow general principles. If the eyes be contaminated by the actual liquid from gas sprays, bursting bombs, etc., lavage with normal saline will be necessary.

CHAPTER IV

NASAL IRRITANTS OR "NOSE IRRITANT GASES"

12. Characteristics and Description of Nose Irritant Gases

These sensory irritants are solid arsenical compounds which can be dispersed by heat or detonation in the form of a very fine, almost invisible, particulate cloud or smoke. They share with the lachrymators the general characteristics described at the beginning of Section 9 in the previous chapter.

The object underlying their original employment in the last war was to penetrate the respirator container in use at that time, in the belief that the distressing symptoms, following even a brief exposure, would induce men to discard their respirators as useless; this would expose them to the effects of lethal gases, such as phosgene, which were often released simultaneously. The nasal irritants did not meet with any striking success, because the methods in use at that time for the release of the gas were unsatisfactory.

The following are typical examples of the nasal irritants:—

(*a*) *Di-phenyl-chlor-arsine* (*D.A.*):—A colourless, crystalline solid melting at 38° to 40° C. (100.4° to 104° F.) and boiling at 333° C. (631.4° F.) at ordinary atmospheric pressure; insoluble in water.

(*b*) *Di-phenyl-amine-chlor-arsine* (*D.M.*):—A yellow, almost odourless, crystalline solid melting at 195° C. (383° F.) and boiling (with decomposition of the compound) at 410° C. (770° F.) at ordinary atmospheric pressure; insoluble in water, and difficult to dissolve in the ordinary organic solvents.

(*c*) *Di-phenyl-cyano-arsine* (*D.C.*):—A colourless, crystalline solid, almost entirely odourless, with a melting point of 33° C. (91.4° F.) and boiling at 346° C. (654.8° F.) at ordinary atmospheric pressure; almost insoluble in water, but dissolving readily in oils and in organic solvents.

These compounds are solids at ordinary temperatures, but when heated or dispersed by an explosive they are vaporised, without decomposition, in the form of an almost invisible cloud of minute particles which remain suspended in the air.

13. Action of Nose Irritant Gases on the Body

The main feature of these arsenical irritants is their power of causing violent sensory irritation in man even though present in extremely low concentrations.

The effectiveness of the arsenical gases depends on the amount inhaled before symptoms (which are delayed in onset for some minutes) make themselves felt. Even if the affected person withdraws from the poisonous atmosphere directly the irritant effects are felt, the symptoms continue to increase in severity for some time before they begin to subside. Unlike the tear gases,

therefore, whose sensory effects are immediate but disappear rapidly on adjustment of the respirator, the arsenical irritants are characterized by an increase in the severity of the initial symptoms after adjusting the respirator or leaving the gas-laden atmosphere. This relatively slowly developing action of the arsenicals and the subsequent aggravation of symptoms constitute their chief danger in the case of uninformed persons, as they may engender distrust in the respirator.

The symptoms are characteristic, and consist of acute pain in the nose and accessory sinuses with a sense of "fullness" in the head and repeated sneezing (hence the term "sternutators" often applied to these gases). A burning sensation in the throat, and one of tightness and pain in the chest, also a feeling of grittiness in the eyes with pain and lachrymation, and aching of the gums, are common, while salivation, with nausea or even vomiting, are important symptoms. Acute mental distress is very marked in severe cases, who feel and look utterly miserable. This condition of intense discomfort is very alarming to the inexperienced.

The irritant effects are transitory, and symptoms usually disappear within an hour. Even in severe cases no lasting organic lesion is likely to follow exposure to these gases in the open.

The respirator charcoal, which arrests the lethal gases, has little or no protective value against the particulate clouds of these arsenical compounds, but special filtering devices are added to trap these particles or smokes.

Symptoms of true arsenical poisoning may occur through persons using water drawn from bomb or shell craters contaminated with these arsenicals. The arsenic content in these craters may be very high, and men have been known to suffer from dermatitis after shaving with water drawn from them.

14. Treatment for Nose Irritant Gases

In the great majority of cases a brief period of rest is the most that is required. In a few exceptionally severe cases, however, pain may call for medical relief, when the inhalation of a little chloroform may be found of transitory assistance. A 5 per cent. solution of sodium bicarbonate for nasal irrigation or as a gargle will help to allay the irritationof the nose and throat.

If a respirator is being worn, care should be taken to remove the clothes previous to taking off the respirator, or the arsenical particles shaken out of the clothes may be inhaled.

CHAPTER V

ASPHYXIANTS OR "LUNG IRRITANT GASES"

15. General Description of Lung Irritant Gases

The chief members of this group are chlorine, phosgene, di-phosgene and chloropicrin. Chlorine and phosgene are true gases under normal conditions of temperature and pressure, while di-phosgene and chloropicrin are liquids of comparatively high boiling points—a quality which renders them somewhat persistent.

Chlorine was the gas first used by the Germans in the last war. On release from the cylinder, bomb, etc., it forms a greenish yellow cloud with a pronounced smell of bleaching powder. It is highly irritant to the mucous

membranes of the upper respiratory passages, and a marked feature of its action is the violent and paroxysmal cough which it induces, and which presists even after the cessation of exposure.

Phosgene is the most important member of the group, and the most toxic. This gas is a liquid which boils at 8.2° C. (46.8° F.) with the evolution of a dense, colourless vapour. Although the least immediately irritant of the group, phosgene is readily detectable, even in concentrations which are harmless to the lungs, by its characteristic smell of musty hay.

Di-phosgene is an oily liquid boiling at 128° C. (262.4° F.) and smelling like phosgene. Apart from its more pronounced lachrymatory power, the symptoms produced by this gas are practically identical with those caused by phosgene.

Chloropicrin is a yellow liquid boiling at 112° C. (233.6° F.); its smell resembles that of chlorine. Chloropicrin is the most irritant member of the group, causing more sensory irritation of the respiratory passages than chlorine. It is also cumulative in its action, and frequent exposure to small doses may gradually lead to a greatly increased susceptibility with a liability to attacks of nocturnal "asthma". It is also a strong lachrymator.

In view of the comparatively low toxicity of chlorine and chloropicrin when compared with phosgene, and also because of the ease with which their presence, even in low concentrations, is advertised by their immediate iritancy, it is less likely that either of these gases will be widely used in modern warfare in the field. There is no doubt, however, that they are dangerous gases if liberated in confined or built-up areas, and their moral effect if used against civilian centres might well be great.

16. Methods of Dispersion of Lung Irritant Gases

With the exception of chlorine, these asphyxiant gases may be dispersed by any type of projectile in ordinary use—shell, aircraft bomb, mortar bomb or Livens drum. In addition, both chlorine and phosgene may be dispersed from cylinders either mounted in fixed positions or carried on moving vehicles; in the case of phosgene the emission of the gas is hastened, in cold weather, by mixture with chlorine.

In the last war a combination of two or more gases of this group was almost invariably used in any cloud gas attack or gas shell bombardment. It appears doubtful, however, whether any asphyxiant gas other than phosgene would be employed in future wars, and, in view of the high concentration necessary to produce effective results, it is likely that phosgene will only be used in large calibre projectiles or in cylinders mounted on moving vehicles.

By whatever means these chemical agents are distributed, the resultant gas clouds are carried down wind. Action on unprotected persons in the path of the cloud is only limited by the period of their exposure in the cloud. In the case of those who have respirators available the effective exposure is only the brief time occupied in adjusting the respirator.

Nevertheless, it must be borne in mind that the sudden release of a heavy cloud of asphyxiant gas in enclosed areas or in close proximity to personnel is fraught with danger even to persons who have (but are not wearing) respirators, since the intense spasm induced by a single breath of a heavy concentration of the gas may interfere with the quick adjustment of the respirator.

17. Mode of Action of Lung Irritant Gases

All lung irritant gases cause essentially the same type of pathological effect, this being most pronounced on the alveoli of the lungs and on the smaller bronchial tubes, and the great danger to be feared is the onset of acute pulmonary oedema. The rate of onset and the degree of oedema depend on the particular gas and on its concentration, and also, to a lesser degree, on the duration of the exposure.

These gases are also quite effective lachrymators, especially di-phosgene and chloropicrin; but they are far less powerful in this respect than the true lachrymators.

Their relative toxicity varies, except in the case of phosgene and di-phosgene where it is approximately the same. In lethal concentrations it is found that chloropicrin is about four times, and phosgene (or di-phosgene) practically ten times, more toxic than chlorine; and, while chlorine and chloropicrin cause more damage to the lining membranes of the respiratory passages, phosgene is more effective in the production of pulmonary oedema.

There is, however, little essential difference between the actions of the various members; the treatment and the prognosis for all alike depend on the extent of the lesions produced in the lungs. The extent of these lesions varies as much with the concentration of the gas as with its particular nature, and they are to be determined by a consideration of the clinical features rather than by reference to the exact nature of the toxic substance.

As a type of the group, the morbid anatomy and the signs and symptoms of phosgene gas poisoning will now be described; differences from those of poisoning by other gases of the group will be considered as they arise.

18. Morbid Anatomy of Lung Irritant Cases

The essential lesions are pulmonary oedema, rupture of the pulmonary alveoli and concentration of blood, with increased viscosity and a tendency to thrombosis.

The earlier death ensues, the greater is the degree of pulmonary oedema found at *post-mortem* examination. Oedema may be fully established within two hours of gassing, when the lungs are found to be small or normal in size, heavy and completely water-logged and with no sign of emphysema.

When death occurs later on the first day the lungs are voluminous, heavily oedematous and congested with blood, while aerated patches of emphysema, especially at the edges of the lungs, alternate with patches of collapse. On section, frothy serous fluid mingled with blood drips from the lung tissue, and petechial haemorrhages may be visible on the surface of the lungs. The pleural cavity almost invariably contains a quantity of serous, perhaps blood-stained, effusion which may vary in amount from two to twenty ounces.

In the case of deaths occurring on the second or third day, evidence of aeration of the lungs is present, especially in the lower lobes, and serous fluid does not drip so freely from the cut surface. With later deaths this dripping of serous fluids has ceased, and commencing broncho-pneumonia and pleurisy may indicate that secondary bacterial infection has set in.

The greater aeration of the lungs of cases dying on the second and third days, taken in conjunction with the clinical history of the severe cases that survive, makes it evident that the oedema fluid is rapidly absorbed from the lungs from the second day onwards.

In severe cases the haemoglobin percentages may rise as high as 140 with a corresponding rise in the red cell count. Associated with this concentration is the occurrence of thrombosis in the pulmonary blood-vessels, and also, to a variable extent, in those of other organs of the body.

Phosgene has relatively little effect on the upper air passages, but in chlorine and chloropicrin poisoning the bronchial tubes, and even the trachea, may show serious damage. The epithelial lining may be severely affected and desquamation may take place; the liability to blockage of these channels is therefore great, and the paroxysms of violent coughing, so typical of chlorine and chloropicrin poisoning, tend to induce a disruptive emphysema which is much more marked in these cases than in phosgene poisoning. Subcutaneous emphysema was rarely seen in the last war except after gassing by chlorine.

19. General Symptoms and Signs of Lung Irritant Poisoning

Exposure to an atmosphere containing phosgene or other pulmonary irritants may lead to two types of acute casualties:

(*a*) *Acute, with Violent Onset.*

Typical cases of this type are best seen after exposure to chlorine or chloropicrin; but even phosgene, in an effective concentration, causes immediate sensory irritation of the respiratory passages and catching of the breath, accompanied by a cough and a sensation of constriction and pain in the chest; this is followed by gasping respirations which are interrupted by fits of violent coughing. Even after leaving the poisoned atmosphere the respiration remains rapid and shallow, and any attempt to draw a deep breath gives rise to painful discomfort and cough. Retching and vomiting are prominent features in the early stages of poisoning, and headache and a profound sense of fatigue often prostrate the patient.

In chlorine and chloropicrin poisoning a marked feature is the paroxysmal cough, accompanied by vomiting, which not only occurs during the exposure, but persists for a long time afterwards.

With the onset of oedema the breathing becomes rapid and panting, but of a characteristically shallow type; the ears and lips, and eventually the whole face, assume a cyanotic tint which, in cases of chlorine poisoning, may deepen to the intense violet of fullest cyanosis, with visible distension of the superficial veins of the face and neck.

In phosgene poisoning this full cyanosis is often omitted, and the patient passes rapidly into a state of circulatory collapse with a feeble, flickering pulse of over 120, a cold, clammy skin and a leaden hue of the face. In both these types of asphyxia the onset of cyanosis is marked by increasing restlessness and apprehension as the patient realises that he is being suffocated by his own secretions.

Apart from the fulminating cases in which death follows within two or three hours after exposure, lung irritant casualties can be divided into three types:—

(1) The milder case, with flushed face, somewhat rapid respiration and a painful cough.

(2) The severe case, with "blue" cyanosed face, distended neck veins and a full strong pulse of 100.

(3) The severely collapsed case, with a leaden "grey" cyanosis of the face and a rapid, thready pulse.

The mild case is often drowsy, and soon falls into a sleep from which he wakes refreshed. Coughing and a sense of debility may persist for a few days, and a temporary bradycardia may develop, but convalescence is assured.

Even cases of severe cyanosis tend to recover in two or three days if the colour is well maintained and the pulse rate does not exceed 100. At any time, however, and particularly if subjected to physical effort, these cases may rapidly pass into the dangerous condition of "grey" cyanosis and collapse, with thready and irregular pulse, from which they may not recover.

The mortality among the "grey" cases is high, death being due to failure of circulation, or to a fatal broncho-pneumonia which may interfere with recovery. When this infective complication develops, the sputum becomes purulent, the temperature rises and death usually follows rapidly.

(*b*) *Acute, with Insidious Onset.*

Cases occurred in the last war in which men who had been exposed to phosgene gas had been able to carry on their work for an hour or two with only trivial discomfort, and even to march from the trenches to their billets; thereafter they became rapidly worse and passed into a condition of collapse with a progressive pulmonary oedema which sometimes proved rapidly fatal.

In other cases men whose only complaint was a slight cough and tightness of chest had suddenly collapsed, and even died abruptly, some hours later on attempting to perform vigorous muscular work. Varying degrees of the same delayed effect of phosgene poisoning were met with, but in all cases the deficiency of oxygen (the result probably of pulmonary oedema already existing) had not been felt until muscular exertion increased the need for oxygen.

This delay in the onset of serious symptoms is not evident in chlorine gas poisoning, where the violent paroxysms of coughing, the painful dyspnoea, and the repeated attacks of vomiting convey the impression that the case is seriously ill from the start.

20. Physical Signs in Lung Irritant Poisoning

The percussion note may remain resonant over the chest, notwithstanding the existence of pulmonary oedema. The breath sounds are weakened, especially behind; they may also be harsh in character, but never tubular. Fine rales are heard, chiefly in the axillary region and at the back and sides of the chest, while rhonchi may be noted occasionally.

In the early acute stage the physical signs give little indication of the gravity of the case or the extent of the damage to the lungs. The colour, the pulse and the character of the respiration are the chief guides to prognosis. With the development of inflammatory complications and rising temperature the physical signs become those of pleurisy, bronchitis or broncho-pneumonia.

21. Prognosis of Lung Irritant Cases

Cases of the "blue" type which react favourably to the administration of oxygen usually do well, and if the circulation and the activity of the respiratory centre can be maintained the oedematous fluid in the lungs is absorbed within four or five days.

In the "grey" type, when cardiac weakness is increasing, the prognosis is bad; if recovery takes place it is often succeeded by a broncho-pneumonia which usually proves fatal. If, however, a case lasts into the third week after gassing, he may justly be expected to survive the acute infection. In

80 per cent. of the deaths due to phosgene or chlorine in the last war, the death occurred within 24 hours of gassing.

In the last war broncho-pneumonia was found to be more frequent and serious in men who had been suffering from bronchitis previous to gassing; similarly, men with pre-existing emphysema or lung disease were handicapped in their struggle against pulmonary oedema, since the margin available for respiration was correspondingly less.

22. Treatment of Lung Irritant Cases

Cases of all degrees of severity may be met with, and it may be difficult at times to decide whether or not a man has really been gassed. The benefit of the doubt should be given to the patient in all cases, and any man showing any feature of lung irritant poisoning should be rested for 24 hours for observation. It should be borne in mind that a delayed action may be exhibited by some pulmonary irritants, notably phosgene and the nitrous gases; but if no objective symptoms have arisen after the lapse of 48 hours the patient can be discharged.

(*a*) *Treatment in the Acute Stage.*

The essentials of treatment for acute poisoning by any pulmonary irritant gas are rest, wamth, venesection and oxygen. It is necessary to think of lungs choked by inflammatory oedema which none the less may be absorbed in three or four days if the circulation can be maintained for so long.

(1) *Rest.*—The importance of rest cannot be exaggerated; in the earlier stages undue muscular exertion may lead to an aggravation of the symptoms, while in the later stages, when the oxygen supply of the body is interfered with by pulmonary oedema, any attempt to perform muscular work may have disastrous consequences.

All cases should be treated as stretcher cases, and their clothing eased so as not to impede breathing. If this is not possible, walking cases should be given every assistance, so that they may avoid physical effort as much as possible. As with every type of gas casualty, the patient's contaminated clothing should be removed on reaching the first aid post or hospital. Those who show definite symptoms should not be allowed to leave their beds or stretchers for any purpose whatever.

(2) *Warmth.*—This helps to combat shock, and also to diminish the oxygen consumption that is entailed by the muscular movements of shivering; attention should therefore be directed to this point when the patient's clothing is removed.

(3) *Venesection.*—As soon as cyanosis begins to appear, 15 to 20 ounces (300 to 600 ccs.) of blood should be withdrawn from a vein by a large bore needle. This treatment is beneficial for all cases except those of the leaden grey hue with failing circulation and a rapid, thready pulse. For this latter group it is harmful. After venesection the headache often disappears, dyspnoea is somewhat diminished and sleep usually follows. If these results follow, nothing further may be needed. But if symptoms of cyanosis and pulmonary oedema seem to be increasing, oxygen treatment should be begun at once.

Experiments carried out on animals late in the last war indicated that when venesection was combined with the intravenous infusion of isotonic salt solution still better survival results might be expected. There was no

evidence that the infusion, when performed some time after venesection, led to an increase of lung oedema. Although this combined treatment was not applied to human casualties, it is possible that the method may prove a useful means of treating cases with pulmonary oedema when the haemoglobin estimation shows an unduly high concentration of the blood.

(4) *Oxygen.*—Oxygen should always be given to casualties with serious pulmonary oedema, that is, to those with intense blue cyanosis or grey pallor. The aim should be to tide the patients over the critical period of the first two or three days, and for this purpose oxygen should be administered continuously by means of some special apparatus, such as Haldane's mask or a nasal catheter, that will ensure a suitable mixture with air.

The oxygen need not be warmed, and a sufficient current of it should be used (from two to ten litres a minute) to ensure a change in the patient's colour from livid blue or grey to a pink tint. If the light is insufficient to allow cyanosis to be judged, guidance may be obtained from the behaviour of the pulse. This treatment must be maintained, day and night if necessary, with a progressive lessening of the oxygen supply, until the patient does not lapse into cyanosis when the oxygen is withdrawn. When oxygen is given continuously over long periods, intermission should be made for five minutes every half hour.

If the supply permits, oxygen should also be given to the milder cases of oedema in order to prevent their lapsing into a more serious state of asphyxia.

The experience of the last war showed that no patient in whom it was possible to restore a pink colour by the proper use of oxygen died from simple pulmonary oedema. In a hospital likely to receive any considerable number of cases of lung irritant poisoning (not of broncho-pneumonia from mustard gas), it is helpful to establish for their treatment some definite routine of oxygen administration on the lines of the suggestions contained in Appendix C.

(*b*) *General Treatment.*

Serious cases are best treated in a well lit and well ventilated ward, protected from chill. The diet should be fluid and sparingly given in the acute stage, but bland drinks should be allowed freely.

Expectoration should be encouraged by some postural device; vomiting is helpful in emptying the lungs, and often occurs spontaneously, but it is liable to produce exhaustion, and it should not be induced by powerful drugs such as apomorphine or ipecacuanha. Raising the foot of the bed or stretcher three or four feet for a few minutes at a time, with the idea of draining fluid from the chest, is sometimes effective in helping free expectoration.

Expectorants should not be given to severe cases during the first two or three days for fear of increasing the tendency to cough and so augmenting the damage in the lungs. In mild cases, or when the acute symptoms have abated in the severe cases, ordinary expectorant mixtures containing ammonium carbonate and vinum ipecacuanhae may be given with advantage and are helpful in checking the possible development of infective bronchitis.

No drugs were found to be of any special value in the last war. Atropine did not prove effective in checking oedema or in relieving bronchial spasm, while morphia is dangerous and should only be used in small doses (1/6th gr.) to control extreme restlessness. The relief of asphyxia is the best means of relieving the headache and the best cardiac stimulant is oxygen.

If pulmonary complications develop (such as infective bronchitis, broncho-pneumonia, etc.), the patient should, if possible, be treated in a separate ward; otherwise, he should be separated by at least six feet from his nearest neighbour.

(*c*) *Treatment in the Convalescent Stage.*

No case should be moved, for purpose of convalescence, until definite cyanosis or severe symptoms have disappeared; it is also very important that a note of the special symptoms attending the acute illness should be forwarded with each case in order that subsequent treatment may be rightly controlled.

The milder casualties are likely to recover after a short rest; those who have passed through a stage of severe cyanosis, however, or who have suffered from a complicating broncho-pneumonia require a prolonged period of convalescence.

All except the more severe cases should be got up from bed as soon as possible; slight bronchitis or gastric disturbances, which usually are only temporary, do not contraindicate this, but cases of abnormally rapid or slow pulse should be rested a little longer.

A system of carefully graduated exercises, with full opportunities for lying down and resting in the intervals, should be instituted; the response to exercise of each individual, however, must be carefully studied, and exhaustion must be guarded against as symptoms of disordered action of the heart may develop, and add weeks or months to the period of convalescence.

23. After-effects of Poisoning by Lung Irritant Gases

Apart from infective broncho-pneumonia, which usually appears towards the end of a week and which may, in severe cases, develop the usual septic complications, the sequelae of poisoning by the asphyxiant gases are, contrary to popular belief, much less grave than was anticipated in the last war. The great majority of cases were restored to good health. Some continued to show inability for severe muscular effort or even for moderate exercise, associated with tachycardia and a rapid, shallow type of breathing. Recurring frontal headache, generally worse after exercise, and epigastric pain of a temporary nature were frequent, and, while pain in the chest was variable, the presence of a mild bronchitis was found in an appreciable proportion of cases.

In the last war, the two important, though not common, sequelae which tended to prolong invalidism were "D.A.H." and nocturnal "asthma".

"D.A.H." or irritable heart, with precordial pain, a sense of exhaustion, dyspnoea and persistent tachycardia after exercise but no evidence of organic heart disease, was the commonest and most persistent after-effect. A small proportion proved intractable, and there was evidence to show that this invalidism was increased if the men were pressed to physical effort too early and too fast at the beginning of convalescence.

Nocturnal asthma, which differed from the ordinary asthma of civil life, took the form of spasmodic attacks, lasting from three to thirty minutes, with shallow and rapid, but not difficult, respirations and with no abnormal physical signs in the chest during the attack. The pulse might be slow and full, or rapid and almost impalpable, and both the haemoglobin percentage and the red cells were increased.

Both disabilities almost always yield in time to a slowly progressive routine of graduated exercise, coupled with careful supervision and feeding, and firm reassurance to dispel neurasthenic factors.

Patients who have passed through a phase of infective broncho-pneumonia should always be considered as a group apart.

24. Invalidism after Lung Irritant Poisoning

Until knowledge came through clinical experience, there was much apprehension in the last war of permanent damage to the lungs after the inflammation caused by these chemical irritants. It was thought that severe emphysema and fibrosis of the lungs, or perhaps pulmonary tuberculosis, might result among the survivors. Nothing so gloomy occurred. The after-histories of selected groups of cases were followed out in detail during the war, and the records of the Ministry of Pensions were analysed up to 1920 in order to produce the evidence related in the Official History of the War. Subsequent experience has revealed nothing to alter the conclusions then reached. It was evident that men who had suffered from the most severe cyanosis with acute pulmonary oedema could recover rapidly and completely. Many such went back to full military duty after a convalescent period of from three to four months. Others, as described above, suffered from neurasthenic features of exhaustion or from temporary loss of wind and endurance. But a small proportion did develop permanent disability, with progressive dyspnoea, recurrent bronchitis and a radiographic picture of scattered fibrosis and emphysema in the lungs. Since it was proved that a man could recover completely from the effects of the chemical irritant, it is probable that these rare examples of chronic invalidism were due to slow fibrosis caused by the secondary complications of broncho-pneumonia.

As regards tuberculosis, a general survey of the situation in the last war was made, and it soon became evident that, in spite of what was being written in certain countries, there was, as would seem logical, no ascertainable connection between gassing and tuberculosis. On the other hand, it is unquestionable that gas aggravated any pre-existing tuberculous condition, where such was present, as would be expected.

CHAPTER VII

LEWISITE

37. General Description of Lewisite

Lewisite, or chloro-vinyl-di-chlor-arsine, is an important chemical compound developed towards the end of the last war, so that knowledge of its action rests on laboratory experience rather than on the study of actual war casualties.

In the pure state lewisite is a powerfully toxic substance, embodying the aggressive qualities of the asphyxiant gases, the irritant characteristics of the tear and nasal irritant gases, and the universal action on all contaminated tissues of the blister gases. Under modern conditions of chemical warfare, however, its vesicant action would predominate.

Broadly speaking, the effects of lewisite on the human body are similar to those produced by mustard gas, but as it has an immediate irritant action

both on the respiratory tract and on the skin, it is more easily detected. In order to avoid repetition, therefore, the following remarks will be confined mainly to salient points of difference in the pathological effects of the two vesicants.

It is assumed that, as in the case of mustard gas, lewisite could be used either in bombs or as spray.

38. Physical and Chemical Properties of Lewisite

A study of the main physical and chemical characteristics of lewisite will be found of assistance when comparing its action of the body with that of mustard gas.

Appearance.—Lewisite is a heavy, oily liquid, colourless in the pure state, but darkening on standing. The impure form, which is more likely to be used in war, is dark in colour.

Odour.—Pure lewisite has no smell, but is extremely pungent and irritating to the nose, giving rise to symptoms of coryza, with sneezing and lachrymation. On contact with moisture or in the impure form, it possesses an odour very closely resembling that of geraniums. This characteristic smell, coupled with the extreme irritancy of the vapour, renders lewisite easy of recognition even in the presence of other gases.

Boiling and Freezing Points.—The boiling point of lewisite is high, viz. 190° C. (374° F.); hence, like mustard gas, it is classed among the persistent chemical warfare agents. Unlike mustard gas, however, lewisite has a low freezing point (about—13° C. or 8.6° F., as opposed to 14.4° C. or 57.9° F. respectively for pure mustard gas)—a difference of practical importance, inasmuch as lewisite will still be an effective liquid in very cold weather when mustard gas may be frozen solid and its aggressive action in abeyance.

Solubility.—Lewisite is insoluble in water; contact with water results in hydrolysis of the compound with the production of hydrochloric acid and organic oxides—a process which is greatly hastened by an elevated temperature. Lewisite is readily decomposed by alkalies. It is, however, freely soluble in the ordinary organic solvents, in the petroleum series of hydrocarbons, and in oils and fats. As in the case of mustard gas, the lipoid solubility of lewisite endows it with its characteristic property of rapid penetration of the skin.

Stability.—Lewisite is much less stable than mustard gas from a chemical point of view, although, in the absence of hydrolysis, it retains its vesicant properties for a considerable time. Hydrolysis in the presence of hot water or an alkali is rapid. As with mustard gas, strong oxidising agents or chlorine will neutralise lewisite, though not so readily.

Penetration.—Like mustard gas, lewisite possesses powerful penetrative properties which, added to the persistent quality of the gas, enable it to render clothing and other materials contaminated by it dangerous to wear or to handle.

39. Toxicity of Lewisite

Lewisite is both an asphyxiant and a vesicant, more rapid than mustard gas in its action, producing more discomfort on inhalation and more irritation when placed on the skin. It thus lacks the insidious character of mustard gas inasmuch as its extreme pungency and penetrative odour ensure its early detection when similar concentrations of mustard gas might not be noticed.

Like mustard gas, lewisite will attack any part of the body exposed to it. Its vapour is a powerful irritant of the whole of the respiratory tract, and, under suitable conditions, may produce vesication of the skin; in the liquid form it penetrates the tissues rapidly and, after a short latent period, gives rise to severe blistering. Liquid lewisite in the eye entails immediate incapacitation.

In contrast with mustard gas contamination, which is never followed by a general systemic poisoning through absorption of the chemical, a massive skin contamination by liquid lewisite may produce acute arsenical poisoning in which all the organs of the body may be affected and arsenic may be found in all the tissues.

As mentioned in Section 31 for mustard gas cases, the situation of persons contaminated by lewisite may be aggravated by wounds or other physical injury.

There is no evidence, as yet, of the acquisition of a hypersensitivity after repeated burns by lewisite—a condition which has been proved to occur occasionally after similar mustard gas exposures.

40. Comparative Dangers of Lewisite and Mustard Gas

Lewisite and mustard gas may be compared as follows:—

(*a*) The easy detection of lewisite as compared with mustard gas tends to reduce its effectiveness.

(*b*) Although lewisite with its higher vapour pressure tends to produce a higher concentration of the vapour than mustard gas under similar conditions, the effective duration of such a concentration is shorter.

(*c*) The rapid hydrolysis of lewisite in the presence of moisture renders it less suitable than mustard gas for purposes of ground contamination, or for use as a spray in moist climates.

41. Nature of Casualties from Lewisite Vapour

The irritant character of lewisite vapour, even in low concentrations, ensures its speedy detection, and compels everyone to seek the protection afforded by a respirator. This is in marked contrast with the insidious character of mustard gas vapour, in which the absence of immediate irritation may readily disguise its dangerous qualities.

The early detection of lewisite vapour should ensure, by immediate use of the respirator, immunity for the eyes and the respiratory tract; in the absence of suitable protective clothing, however, the skin of the body is liable to suffer through the action of the vapour absorbed by the garments, if the latter be dry. Wet clothing is a partial safeguard against the vapour, as the moisture in the material will tend to hydrolyse it.

A minor, though possibly alarming, peculiarity of lewisite vapour (which it shares with other arsenical "gases") is the temporary increase in the intensity of the nasal and respiratory irritation which appears *after* adjustment of the respirator, and which may lead to a lack of confidence in the respirator.

The types of injury to which lewisite vapour might give rise are summarised below.

(1) *Action on the Eyes.*

The irritancy of lewisite vapour and the consequent immediate adoption of protective measures tend to minimise effects on the eye, while the lachryma-

tion which it induces will form an additional factor of safety; it is not likely, therefore, that lewisite vapour will produce a large number of eye casualties.

If severe burning should occur it would be analogous to that produced by mustard gas vapour, but much more acute. Under these circumstances the eye lesion will undoubtedly be accompanied by pulmonary injury.

(2) *Action on the Respiratory Tract.*

The rapid adjustment of the respirator, enforced by the intolerable character of lewisite vapour, affords the greatest security against serious respiratory lesions. In the absence of such protection, the train of symptoms is much more acute than with mustard gas vapour, for even with low concentrations the nasopharynx is affected within a few minutes, and symptoms of coryza, with salivation and laryngeal irritation, quickly supervene. These are followed by a generalised bronchitis which is well established in 24 hours, and, which, in a severe case, may lead to broncho-pneumonia and death.

(3) *Action on the Skin.*

Wet clothing is somewhat of a safeguard against lewisite vapour, but its absorption by dry clothing renders the latter dangerous for continued wear.

Unless in high concentration on a hot, receptive skin, or on prolonged exposure (as when wearing contaminated clothing), the action of lewisite vapour does not go beyond an erythematous condition of the area affected. When vesication occurs, the skin presents an angry surface interspersed with small, shallow, turbid blisters which may coalesce to form one large vesicle; the irritation is more marked than with the corresponding mustard gas vapour burn.

42. Nature of Casualties from Liquid Lewisite

The immediate dangers incurred by moderate or severe contaminations with liquid lewisite are similar in nature to those following contamination by liquid mustard gas: the main difference lies in the greater rapidity of action of lewisite.

The types of injury which might result are summarised below.

(1) *Skin Burns due to Liquid Lewisite.*

(a) *On bare skin.*

As with mustard gas, liquid lewisite attacks the bare skin on contact, but penetration is much more rapid. Unlike mustard gas, however, which does not produce any sensory irritation of the skin on contact, the application of liquid lewisite is usually followed by a stinging sensation which may persist for some time, and which may well prove severe if an extensive area or a hypersensitive surface be affected.

The extent and severity of the resulting damage naturally varies, as in the case of mustard gas, with the degree of contamination and the part of the body affected; experimental evidence shows, however, that very small drops of lewisite are (except in the case of the eyes) less effective than similar sized drops of mustard gas, while the reverse is the case with large drops on the bare skin or on the outer surface of dry clothing.

Erythema of the skin following liquid lewisite contamination develops rapidly—usually in 15 to 30 minutes—and, apart from this rapid development, it may be indistinguishable from that caused by liquid mustard gas. Vesication is correspondingly early and is fully developed within 12 hours or less;

it is at this stage that a clear distinction can usually be made between lewisite and mustard gas burns. The *lewisite blister* is more sharply defined, overlies practically the whole of the erythematous area, and is filled with an opaque or opalescent fluid which, on examination, is found to be rich in leucocytes and contains traces of arsenic; the typical *mustard gas blister*, on the other hand, is surrounded by an angry zone of erythema, and contains a clear, limpid, lemon coloured serum with no trace of the vesicant.

In the absence of secondary infection the healing of small, localised lewisite blisters takes place more readily than in the case of similar mustard gas vesicles; if sepsis be present, however, and contamination be widespread, it is not likely that any appreciable difference in the period of invalidism will result.

The risk of serious arsenical poisoning following liquid contamination of the bare skin has not, naturally, been studied on the human subject; the danger, however, appears to be remote unless the contamination be massive, such as might follow an accident in factories or the bursting of a lewisite bomb in close proximity to some person.

(b) *On clothed skin.*

The effects of liquid lewisite on the skin through clothing, when compared with those produced by liquid mustard gas under the same conditions, are largely governed by the degree of contamination sustained and the amount of moisture present in the garments.

Unless the contamination be massive, or the clothing thin and scanty (as in the tropics), the action of liquid lewisite on the clothed skin may be ascribed entirely to that of the vapour evolved from the contaminated area.

(2) *Eye Burns due to Liquid Lewisite.*

The dangerous possibilities of aircraft spray, already referred to in connection with eye contamination by liquid mustard gas, are intensified in the case of liquid lewisite. However small the contamination, the impact of liquid lewisite on the conjunctiva elicits immediate pain, spasm and lachrymation. The vesicant acts, subjectively, like a powerful caustic, and the victim becomes an immediate casualty; this is in marked contrast with liquid mustard gas which does not usually incapacitate until about one hour after contamination.

Clinical signs following lewisite contamination of the eye develop rapidly —much more rapidly than with similar mustard gas contaminations. An acute inflammatory condition of the conjunctiva, with pronounced engorgement of the blood vessels, is well established in 10 to 15 minutes; oedema of the eyelids, intra-ocular pain and photophobia follow rapidly, and within three or four hours the clinical picture is alarming—an intense oedema, with eyelids closed and adhering by their margins, separation of which releases purulent fluid; chemosis, submucous haemorrhages and extensive ulceration of the conjunctiva; a hazy cornea, and generally, a condition even more distressing than that produced by liquid mustard gas in 24 hours.

The prognosis in liquid lewisite contamination of the eye is even more serious than with liquid mustard gas.

43. Preventive Treatment in case of Lewisite Contamination

The extreme rapidity with which liquid lewisite penetrates the skin limits the value of all preventive treatment unless it be immediate; with lewisite, even more than with mustard gas, speed is of the utmost importance.

Theoretically, lewisite may be neutralised locally by the application of water, especially if the latter be hot and contain an alkali; it may also be removed by solvents, or it may be destroyed by bleaching powder, as in the case of mustard gas. Practically, it has been found that none of these methods is successful in preventing a burn unless applied within one minute after liquid contamination of the skin.

The prompt application of aqueous bleach or of solvents, especially if the liquid contamination be still visible on the skin, may succeed in preventing vesication; penetration, however, is so rapid that the use of bleach, if belated, may only serve to irritate an area already damaged.

This does not necessarily imply that no action can be taken to lessen the eventual consequences of contamination. The speedy removal of all contaminated clothing, followed by a hot bath combined with the liberal use of soap, is just as helpful in reducing the severity of possible lewisite burns as in the case of mustard gas, and is just as essential if further contamination is to be avoided; indeed, the period during which contaminated clothing may safely be worn is even shorter with lewisite than with mustard gas, especially if the contamination consist of large drops or splashes of the liquid.

After *vapour* exposures, a hot bath will usually suffice to ward off serious effects; irritation of the eyes or of the naso-pharynx may be treated with irrigations of sodium bicarbonate.

44. Curative Treatment for Lewisite Casualties

The curative treatment of lewisite lesions is mainly symptomatic, and follows the same lines as the treatment of mustard gas casualties. The preliminary bath advocated in the treatment of mustard gas casualties must similarly be administered to all lewisite casualties; in addition to removing all traces of the vapour from the surface, the bath will also reduce the risk of secondary infection of any skin lesion that may subsequently develop.

If vesication occur, and especially if the area thus affected be extensive, it is essential that the fluid in the blister be evacuated early, the epithelium removed and the raw surface irrigated, in order to lessen the danger of absorption of arsenic. In the absence of sepsis such burns heal more rapidly than the corresponding mustard gas burns.

For reasons already stated, burns of the eye by lewisite vapour are not likely to be so numerous or so severe as with mustard gas; liquid lewisite contamination, on the other hand, will present a serious problem, and treatment will be difficult to carry out owing to immediate spasm and the early and extensive swelling.

Clinical evidence of arsenical absorption, as the result of gross liquid contamination of the skin, may be found in the presence of arsenic in the urine, with, possibly, signs of renal and hepatic congestion and gastric and intestinal disturbances.

CHAPTER VIII

PARALYSANT GASES

This chapter deals with two gases, hydrocyanic acid and hydrogen sulphide, which are not ordinarily regarded as war gases because, though they are extremely lethal, it is not easy to create lethal concentrations of them under

war conditions. It is, however, possible that means might be devised of using them. They are also used industrially, and a description of their characteristics and effects is therefore included in this book. All respirators provided by the Government will give adequate protection against any concentration likely to be met as a result of their use in war.

45. Hydrocyanic Acid (Prussic Acid)

Physical and chemical characteristics.

Hydrocyanic acid is a clear, colourless liquid of low boiling point (26° C. or 78.8° F), very volatile and smelling strongly of bitter almonds.* It is very soluble in water and in alcohol, but such solutions decompose rapidly. Watery solutions do not redden litmus paper.

The vapour of hydrocyanic acid is somewhat lighter than air and diffuses rapidly when released. In closed spaces it is extremely toxic; in the open, however, the dispersion of the gas is so rapid that relatively low concentrations result which are not lethal. This fact explains the failure of hydrocyanic acid gas shells in the last war in the open field, where they caused but few casualties.

Mode of action.

The gas is a protoplasmic poison which arrests the activity of all forms of living matter by inhibiting oxidation. In high concentration, such as may be found in a confined space, this gas may well be considered a fulminant poison, as it may cause death with dramatic rapidity through paralysis of the respiratory centre in the brain.

In the open, however, the gas loses much of its potential activity through diffusion; moreover, in low concentrations, the gas may be detoxicated in the body, as quickly as it is absorbed, to products which are relatively harmless. The failure of this gas in the open is therefore not surprising.

A valuable test for confined spaces is to employ susceptible animals (such as canaries, pigeons or dogs) to indicate the presence of the gas; canaries are particularly susceptible, as they succumb in about two minutes when exposed to a concentration which is not rapidly harmful to man.

Hydrocyanic acid gas may in certain circumstances be absorbed by the skin. Owing to the ease with which the gas dissolves in water, the skin absorption danger is greatly increased if the weather be hot, the cutaneous blood vessels dilated, and the skin bathed in sweat.

Symptoms.

With high concentrations the effects are rapid. The symptoms are ushered in by uneasiness and vertigo, palpitation and hurried breathing; unconsciousness and convulsions follow quickly, and death occurs through paralysis of the respiratory centre and failure of the circulation.

Concentrations that are not lethal may yet produce headache or giddiness, and sometimes nausea or inability to concentrate; recovery, however, is usually rapid and complete.

Treatment.

Treatment must be immediate, and the primary, urgent necessity, in order to restore respiration, is to reduce the concentration of the gas in the

*Some persons, as an idiosyncracy, are unable to distinguish the smell of hydrocyanic acid.

circulation. At once remove the victim from the poisonous atmosphere, and use artificial respiration. The administration of oxygen with an admixture of 5 to 7 per cent. carbon dioxide is useful. The carbon dioxide, through central stimulation, will help to secure thorough ventilation of the lungs.

Various prophylactic and antidotal methods have been suggested based on the laboratory neutralisation of hydrocyanic acid gas by certain chemicals, such as sodium thiosulphate, methylene blue, glucose, etc., but only partial success has, so far, followed their adoption. The main essential in successful treatment is immediate artificial respiration.

46. Hydrogen Sulphide (Sulphuretted Hydrogen)

Physical and chemical characteristics.

Hydrogen sulphide is a colourless gas possessing a foetid odour resembling that of rotten eggs, more offensive in weak than in strong concentrations. Although this characteristic odour can be detected in concentrations low enough to be harmless, fatigue of the sense of smell occurs early, and the odour may cease to serve as a warning. Again, very high concentrations, though highly irritating to the eyes and throat, may be unrecognisable by the sense of smell and may, like hydrocyanic acid gas, be rapidly fatal. The gas, which is inflammable, is heavier than air. Hydrogen sulphide was tried in the last war as an offensive gas, but its use was abandoned.

Its presence may be readily detected, apart from the smell, by exposing lead acetate paper, sheet copper or slightly moistened silver articles, which are all blackened in the presence of hydrogen sulphide.

Mode of action.

Hydrogen sulphide acts both as a local irritant and as a systemic poison. Local irritation is confined to the tissues and exposed mucous membranes of the eyes, throat and respiratory tract, while systemic poisoning follows the invasion of the lungs by moderate or high concentrations of the gas.

High concentrations of the gas produce death with the same dramatic suddenness as with hydrocyanic acid, due in both cases to paralysis of the respiratory centre in the brain. Moderate concentrations give rise to bronchitis and symptoms of pulmonary oedema. There is no evidence that abnormal combinations with haemoglobin are formed. The respirator affords protection against this gas.

Symptoms.

Symptoms of acute poisoning are usually ushered in by panting respiration, pallor and rapid unconsciousness; this is quickly followed by cessation of breathing, often accompanied by convulsive movements. The heart continues to beat for some minutes, but, unless the victim is removed from the gassed area and artificial respiration employed immediately, cardiac failure and death will result. In less acute cases of poisoning violent irritation of the eyes and severe inflammation of the respiratory tract, which may prove fatal, are the most prominent symptoms.

Sub-acute cases of poisoning, as met with occasionally in industrial life, are not likely to be seen frequently in war time; they give rise to symptoms of general ill-health, with chronic conjunctivitis and affections of the respiratory and digestive tracts.

Treatment.

As in the case of poisoning by hydrocyanic acid gas, treatment must be prompt. It consists essentially in removal of the victim from the poisonous atmosphere and the administration of artificial respiration, preferably with inhalation of oxygen mixed with 5 to 7 per cent. of carbon dioxide. The latter stimulates the respiratory centre in the brain, and the rapid oxidation of the residual hydrogen sulphide in the blood ensures that no lasting after-effects follow the exposure.

Artificial respiration should be persisted in for a long period, even though there may be no signs of life. This is proved by industrial practice.

The treatment of the sub-acute type is symptomatic; recovery is usually complete if permanent freedom from further exposure can be secured.

CHAPTER IX

OTHER DANGEROUS GASES AND FUMES

The gases dealt with in this chapter are not likely to be used as war gases, but may be met with under certain conditions in war, as well as in time of peace. Some of them are toxic gases, such as carbon monoxide and the nitrous fumes which are generated from burning explosives, while others are substances used for the production of screening smokes, which may be dangerous to handle. In addition, reference is made to the atmospheric dangers encountered in fighting fires.

47. Carbon Monoxide

Carbon monoxide is a colourless, odourless gas, extremely insidious because of its entirely non-irritating character, and the consequent impossibility of recognising its presence in the atmosphere by the senses. It always accompanies any process of incomplete combustion of carbonaceous material, and is therefore commonly met under normal conditions of everyday life. The gas burns with the characteristic blue flame so often seen flickering over a coke or smouldering coal fire, and it forms an explosive mixture with air.

Carbon monoxide is always present in dangerous amounts in the exhaust gases of internal combustion engines, coke stoves or smouldering fires, while varying quantities of it are present in all types of illuminating gas; it also forms the deadly constituent in the so-called "after-damp" in mines.

Carbon monoxide may be met with in dangerous quantities as a result of bursts of high explosive bombs, in blasting operations, and in burning buildings, if the conditions confine the fumes in a narrow space, as well as in the event of fracture of gas mains and pipes.

The respirator does not afford any protection against carbon monoxide, but complete protection is of course given by a self-contained oxygen breathing apparatus, such as is used by firemen.

Small animals, such as mice or canaries, can serve as valuable indicators of the presence of carbon monoxide as, owing to their rapid metabolism, they show signs of carbon monoxide poisoning before man is affected.

Mode of action.

Carbon monoxide owes its poisonous properties to the fact that it combines with haemoglobin to form a dissociable red compound just as oxygen

does, and that its affinity for haemoglobin is about three hundred times that of oxygen. But for this property of combining with haemoglobin carbon monoxide would be a physiologically inert gas like nitrogen or hydrogen.

When a man breathes air containing carbon monoxide, the relative amounts of oxygen and carbon monoxide present in the atmosphere determine the proportion in which carboxyhaemoglobin is found in the blood. When the amount of oxygen is three hundred times that of carbon monoxide half of the haemoglobin can combine with the carbon monoxide and half with the oxygen. This is about the blood-saturation at which unconsciousness occurs.

As the concentration of the gas in the air rises, the saturation of the haemoglobin with carbon monoxide increases, and the oxygen-carrying capacity of the blood progressively diminishes until symptoms of anoxaemia (oxygen want, in this instance without cyanosis) make their appearance. Carbon monoxide is therefore cumulative in its action.

Moreover, the rate of absorption of carbon monoxide is very much accelerated by muscular exertion or by mental excitement, which causes an increase in the breathing and circulation rates. This results in a more rapid diminution in the available oxygen content of the blood, with a corresponding increase in the severity of the symptoms of oxygen want.

The resulting effects are due to anoxaemia alone, and their severity is determined by the degree of oxygen want. There are no pathological changes in the lungs such as follow the inhalation of asphyxiant gases, nor are the red blood corpuscles injured; when freed from their combination with carbon monoxide, the corpuscles are as capable of resuming their normal function as oxygen carriers as they were before exposure to the gas.

Death occurs when saturation of the haemoglobin reaches about 75 per cent., but lower degrees of saturation of the blood may prove fatal if exposure to the poisonous atmosphere is prolonged. The colour of the blood and tissues, *post mortem*, may be bright red.

Symptoms.

The great danger in carbon monoxide poisoning is that, owing to the non-irritant properties and the cumulative action of the gas, the victim may not realise, until too late, that there is any danger present in the atmosphere. The first symptom may be a loss of power in the limbs so that, although he may then appreciate his danger, escape may be difficult or impossible.

Where the proportion of carbon monoxide to oxygen is high, loss of consciousness may be very rapid, with practically no warning. More commonly, however, the onset of symptoms is gradual and insidious, and may be ushered in by a feeling of weakness, giddiness, vomiting, and indistinct vision; this is followed by breathlessness, palpitation and a loss of power in the limbs, and the least exertion at this stage may cause collapse.

The loss of muscular power and the confused cerebration often preclude a man from withdrawing from danger even though he is dimly aware that safety is only a few yards distant. Not infrequently there is a stage of acute mental excitement, which may simulate alcoholic intoxication or even mania. This is more common in the milder cases. Apathy and a sense of complete helplessness supervene, followed by unconsciousness, with or without convulsions; the victim becomes comatose, with stertorous breathing, a low-tension pulse and subnormal temperature, and death results if he is left in the poisoned atmosphere.

The colour of the face in cases of carbon monoxide poisoning may vary with the rapidity of the onset or the degree of anoxaemia. A leaden tint is often seen after profound coma, while in other cases the face may be pale and moist with perspiration; often, however, the cheeks are pink and the lips of a vivid carmine tint.

At the autopsy, the blood may be red in colour instead of dark if there is a considerable degree of saturation of the haemoglobin 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 haemoglobin, and the blood after death will have its normal colour.

The simplest method of detecting the presence of carbon monoxide in blood is to compare the colour 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 $\frac{1}{2}$ per cent. solution is a convenient strength) so that when examined by transmitted daylight the colour 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 colour 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 colour it will be found that the solution made with the suspected blood, if it contains carbon monoxide haemoglobin, 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.

Recovery from the initial symptoms may be followed by some degree of mental confusion and slow cerebration which may persist for varying periods, while headaches, often of a severe or migrainous type, are characteristic. Among the after-effects of severe poisoning may be mentioned cardio-vascular disorders, especially tachycardia and dyspnoea, which may continue for months. There is a predisposition to pneumonia, usually as a sequel to a long exposure to the gas, while disturbances of the central nervous system may occur ranging from a simple neuritis to paresis and even mental derangement, usually of temporary duration, though sometimes lasting as the result of cellular damage to the brain caused by protracted anoxaemia.

Treatment.

The majority of cases of carbon monoxide poisoning recover with prompt treatment, although a relapse, or even sudden death, may occur where exposure to the gas has been prolonged.

The time required for recovery depends however on how long the victim has been exposed to the poisonous atmosphere. Unconsciousness may last for as long as 48 hours after regaining pure air, and yet the person may recover; but the longer unconsciousness lasts the less is the chance of recovery.

Treatment consists in the prompt administration of oxygen, or *preferably* oxygen, or even air, *combined* with 5 *to* 7 *per cent. of carbon dioxide*, aided, if necessary, by artificial respiration.

Pure oxygen can displace the carbon monoxide far more rapidly than ordinary air, while the carbon dioxide stimulates the respiratory centre and induces deeper breathing, thus facilitating the elimination of carbon monoxide from the circulation. It is hardly necessary to add that the expired air must not be re-breathed by the patient, and with this in view, a suitable apparatus

should be used such as the Haldane oxygen apparatus, which may be set to deliver eight to ten litres a minute, or one of the types of apparatus which allow the administration of a mixture of carbon dioxide with either oxygen or with air.

A characteristic effect of carbon monoxide poisoning is a lowering of the body temperature, due to a disturbance of the heat regulating centre and to a reduction in the normal oxidative processes. Even in mild cases patients may complain bitterly of cold, and it is necessary that this symptom be combated by means of hot coffee, blankets, hot water bottles and other familiar measures. Rest, too, is imperative in order to avoid any increase in the oxygen requirements of the body and to reduce the demands on the ill-nourished heart.

In serious cases a slow intravenous injection of 5 ccs. of 25 per cent. coramine solution, or similar respiratory stimulant, is valuable. Blood transfusion has been found of great value in desperate cases. Venesection is quite valueless.

During convalescence, especially after severe or prolonged anoxaemia, particular care should be taken that no great strain be thrown on the heart owing to the risk of acute dilatation.

48. Nitrous Fumes

When nitro-explosives are incompletely detonated or subjected to slow combustion, especially in confined spaces, considerable quantities of nitrous fumes are given off consisting chiefly of nitric oxide (NO) and nitrogen peroxide (NO^2 and N^2O^4).

These fumes, which have an orange-yellow or reddish-brown colour, are very soluble in water, and react readily with moisture and oxygen to form nitric and nitrous acids. In damp surroundings, therefore, the concentration of these gases in the atmosphere will be lowered.

Nitrous fumes may be met with when detonation of a blasting charge is incomplete. In industry, dangerous concentrations may be evolved when nitric acid is heated, or when it comes in contact with organic material, such as wooden floors, after accidental spilling.

The respirator affords limited protection against nitrous fumes, but as it gives no protection against carbon monoxide, which is often generated simultaneously, reliance should not be placed on a respirator save in an emergency.

Nitrous fumes may be readily demonstrated by means of test papers which have been previously dipped in a solution of starch and potassium iodide and slightly acidified; a blue coloration develops on them when they are exposed to the gases.

Though even small amounts of nitrous fumes in the air are mildly irritating to the eyes and the respiratory tract, they are not sufficiently so to serve as a warning of a highly dangerous atmosphere.

Mode of action.

The action of nitrous fumes on the lungs closely resembles that of phosgene. They are particularly dangerous because they do not produce any marked sensory irritation, and men may therefore fail to realise the serious danger which may follow their inhalation.

When inhaled, the nitrous fumes come into contact with the moisture ever present in the respiratory tract, and form nitric and nitrous acids; this

produces a local caustic effect, to which is superadded a general systemic action due to absorption of the alkaline nitrites formed by the interaction of the acids with the alkaline secretions in the presence of oxygen.

As with phosgene, the local action gives rise to an intense congestion of the lungs and the production of an acute inflammatory condition and pulmonary oedema. This always overshadows the general systemic effect of the alkaline nitrites, which, however, contribute to the clinical picture through their enfeebling action on the circulation and the possible diminution, through the formation of methaemoglobin, of the oxygen carrying capacity of the blood.

With nitrous fumes, as with phosgene, the initial symptoms of coughing and irritation are generally transitory, and a period of quiescence precedes the onset of the acute symptoms. This apparently delayed action may vary in duration from two to 24 hours or more, according to the conditions of exposure; the usual duration of the period is between 10 and 20 hours after exposure. Once this period is over, the clinical signs develop rapidly, and the whole course of a possibly fatal illness may be run in a few hours.

Symptoms.

The initial symptoms, on exposure, are slight irritation of the eyes, nose and throat, accompanied perhaps by a little cough—symptoms which are seldom marked and which quickly subside during the latent period which follows. The termination of this latent period, which may be precipitated by physical exertion, is marked by the onset of acute clinical signs and symptoms such as a dry hacking and painful cough, a sense of constriction in the chest, and distressing breathlessness.

In mild cases this may be a prelude to a bronchitis which is limited to the upper bronchi and is accompanied by a profuse muco-purulent expectoration. In more severe cases, however, a condition of acute bronchial spasm may set in, with pulmonary congestion and cyanosis, rapidly followed by a pulmonary oedema which may be haemorrhagic in character. Restlessness is extreme, and in fatal cases consciousness is retained almost to the end, the patient struggling vainly for breath while, with blood stained fluid trickling from his mouth and nostrils, he drowns slowly in the fluid exuded in his lungs

Treatment.

The general principles of treatment in cases of poisoning by nitrous fumes follow the same lines as those already outlined for cases of phosgene poisoning, stress again being laid on the importance of enforcing complete physical rest from the time of exposure and on the early administration of oxygen as soon as cyanosis develops.

Venesection gives better results when practised early, even before the onset of pulmonary oedema, in subjects with a full bounding pulse, but it is generally contra-indicated where the pulse is soft and thready.

It should be remembered that a severe and often fatal broncho-pneumonia is a complication in some cases of lung irritant poisoning.

Convalescence is apt to be prolonged, and the experience of the last war showed that the combined action of carbon monoxide and nitrous fumes had a harmful effect on the heart, necessitating careful surveillance and graduated exercises.

49. Screening Smokes in General

The five substances described in the next five sections can be used as screening smokes to conceal important places. These smokes are irritating when inhaled in close proximity to their source, but are not toxic in the concentrations that render them effective as screens.

The chief danger associated with their use arises through accidental contact with the chemicals used in their production. These chemicals are all corrosive or dangerous to handle, and accidental contamination of the eye or splashes on the skin with the liquids will result in severe ulceration or burns.

Should contamination with the liquid chemicals occur, first aid treatment must be undertaken immediately. If the eye be affected, prompt and copious lavage with water, or with warm sodium bicarbonate solution, may mitigate the resulting effects; splashes on the skin should be treated with excess of water in order to dilute rapidly and wash away the corrosive liquid, while any article of clothing contaminated by the chemical should be discarded at once.

The respirator gives efficient protection against all the screening smokes, and clothing is not affected by exposure to them in the concentrations met in the open.

50. Phosphorus

At ordinary temperatures phosphorus is a solid which can be handled safely in water, but when dried in air it burns fiercely, producing a dense white smoke.

Phosphorus may also be used by an enemy as an incendiary filling in bombs or in shell, and flying fragments or melted particles of the burning chemical may be embedded in the skin of persons close to the bursting missile. These fragments continue to burn on the skin unless smothered; first aid treatment should therefore consist in the immersion of the affected part in water, or, in the absence of enough water, in the application of a thick pad soaked in water.

As the melting point of phosphorus is 44.4° C. (112° F.), the particles embedded in the skin can be removed, under water at or above this temperature, by means of forceps or a gauze sponge, care being taken that none of the fragments be overlooked. The resulting burns should be treated as thermal burns, but they are apt to be slow in healing. Owing to the ready solubility of phosphorus in oils and fats, no oily or greasy dressings should be used until it is certain that all the fragments have been removed.

A form of first aid advocated by American writers is the immersion of the affected part in, or the covering of the area with, thick pads soaked in a 1 or 2 per cent. solution of copper sulphate. The action is to coat the particles of phosphorus with an inert compound by chemical action, and so to arrest the burning and enable removal to be carried out.

51. Chlorosulphonic Acid (C.S.A.)

This is a fuming, highly corrosive liquid which, on contact with quicklime, gives off a thick white cloud closely resembling a dense mist. At close quarters this is sufficiently irritating to the eyes and throat to necessitate the wearing of a respirator, but at a distance of 200 yards or more from the source of emission this can easily be dispensed with.

Owing to its highly corrosive nature, C.S.A. requires great care in handling; moreover, in contact with water it generates intense heat and acid may be scattered in all directions.

Contamination of the eye with the liquid should be treated immediately with large quantities of water, followed by lavage with a 3 per cent. sodium bicarbonate solution; a few drops of castor oil and a light pad over the eye will assist in allaying the irritation.

Splashes on the skin should be flooded with water to remove the contaminant, and sodium bicarbonate solution should be applied locally thereafter. Drying should be effected by mopping up excess moisture gently with swabs or absorbent wool, and not by rubbing.

52. Oleum

Oleum is a brownish-yellow, corrosive liquid consisting of sulphuric acid with a percentage of sulphur trioxide; when exposed to air it gives off dense white fumes with a somewhat sulphurous smell.

As with C.S.A., accidental splashes should be treated immediately with an excess of water to wash off the acid, followed by the local application of sodium bicarbonate solution.

53. Titanium Tetrachloride

This is a yellow, non-inflammable and corrosive fluid which, on contact with damp air, gives off a heavy dense white cloud. Advantage is taken of this property for the production, by aircraft, of vertical smoke curtains extending down to the ground or sea level. The smoke consists mainly of fine particles of free hydrochloric acid and titanium oxychloride, and its efficiency depends largely on the moisture present in the air.

The smoke is unpleasant to breathe, but it is not toxic; the wearing of goggles or a respirator, however, may be necessary when entering a smoke curtain if the spray is still falling, owing to the danger of drops entering the eyes. The usual precautions must be taken when handling the liquid, and accidental contamination of the eyes or of the skin should be treated immediately by free lavage with water, followed by local application of sodium bicarbonate solution.

54. Stannic Chloride

Stannic chloride is a fuming, straw-coloured, corrosive liquid which produces a heavy white cloud on contact with air, and is therefore sometimes utilised by aircraft for the production of vertical smoke curtains. The treatment necessitated by accidental splashes is similar to that associated with titanium tetrachloride.

55. Noxious Products of Combustion

It is common knowledge that fire fighting, especially when practised inside buildings or in confined spaces, often entails risk, either because of noxious gases or through a deficiency of oxygen; hence the various protective devices used by firemen such as respirators and oxygen breathing apparatus.

In all fires in confined spaces the nature and concentration of the toxic gases produced vary with the rate of combustion and with the character of the burning material. Thus, a slow rate of combustion results in a heavy

concentration of carbon monoxide and carbon dioxide, in addition to an oxygen deficiency, while burning cordite (as in a magazine) gives rise, in addition, to the evolution of nitrous fumes.

When chemical extinguishers are used to quell fires in confined spaces, additional toxic gases may be produced causing further danger to unprotected men.

The unfailing presence of carbon dioxide hastens the onset and increases the severity of any toxic symptoms that may result. Carbon dioxide is more than a simple asphyxiant in that, in comparatively low concentrations, it causes increased breathing and thereby increases the quantity or dose inhaled of any noxious gas that may be present. In concentrations above 20 per cent. it produces unconsciousness and death.

The utility of the ordinary anti-gas respirator in fire fighting is strictly limited, and is confined to the arrest of particulate products of combustion and of such gases as can be dealt with by the charcoal in the container. These do not include either carbon monoxide or carbon dioxide. Further, as the respirator cannot compensate for any oxygen deficiency that may arise, it is essential that, when fighting fires inside confined spaces where a free dilution of the atmosphere or a free escape for the noxious gases generated is not possible, an oxygen or air breathing apparatus should be worn.

The toxic properties and mode of action of carbon monoxide and of the nitrous fumes have been described. A short description is appended below of the toxic effects of two types of fire extinguishers in common use, followed by a brief study of the conditions under which a deficiency of oxygen may be met.

56. Dangers from Use of Chemical Fire Extinguishers

The following are two types of filling for chemical fire extinguishers in common use, sold under various trade names:—

(1) *Carbon Tetrachloride.*—This is a volatile liquid, boiling at 76.7° C. (170.1° F.), which is extensively employed as a dry-cleaning agent and as a popular and effective fire extinguisher.

When carbon tetrachloride is sprayed on a fire or on a heated surface the chief decomposition products, in addition to the unchanged chemical, are phosgene, hydrochloric acid and chlorine. The production of phosgene is more marked when the liquid comes in contact with heated rusty iron and when large quantities of the extinguisher are used in the presence of moisture.

Although the thermal decomposition products are more or less irritant to breathe, this irritancy may not be such as to compel men, faced wih a dangerous emergency, to leave a burning room. Under these circumstances a very real danger arises from the continued inhalation of vaporised carbon tetrachloride or its products of decomposition.

Recent experience has shown that exposure to thefumes of carbon tetrachloride itself in a confined space such as a garage or between decks may give rise to serious illness, often delayed in its onset, of renal and hepatic origin. The illness may be ushered in by pyrexia, general malaise and abdominal pain—a commonplace clinical picture which may lead to errors in diagnosis.

Personal idiosyncracy plays a part in the character, as well as in the severity, of the resulting symptoms; but as a rule, signs of impaired kidney function are always present, and may vary from a trivial rise in blood pressure

to an acute uraemia. Evidence of liver damage may also be seen in the jaundice, the slow pulse, the abdominal pain and haemorrhage from stomach and bowel so characteristic of the toxic jaundice caused by the organic halogens.

(2) *Methyl Bromide.*—Another type of fire extinguisher contains methyl bromide as its chief constituent. This is a gas at ordinary temperatures, but it is readily liquefied at 0° C. to a clear, colourless and extremely volatile liquid which boils at 4.5° C. (40.1° F.). The gas is almost odourless.

Methyl bromide is toxic, and its thermal decomposition products are practically irrespirable. The liability of this extinguisher to produce poisoning, however, is chiefly determined by the rapid rate of volatility of the undecomposed chemical, which is much higher than that of carbon tetrachloride. The rapid vaporisation of methyl bromide in confined spaces may easily result in such a high concentration that a toxic dose may be inhaled before the danger is appreciated.

In high concentration methyl bromide has a profound effect on the central nervous system, producing unconsciousness and giving rise to epileptiform seizures and paralysis, both motor and sensory.

In less severe cases vertigo, visual troubles and general weakness are the usual symptoms, and it appears that the dose need not be large to produce such symptoms.

57. Dangers of Oxygen Deficiency

Oxygen deficiency occurs as the result of fires in confined spaces, and the presence of carbon monoxide and carbon dioxide adds to the danger.

There are many situations, however, other than actual fires in closed compartments, where a reduction of the oxygen percentage in the atmosphere may occur. Apart from such occasions as high altitude flying or climbing, oxygen deficiency may be met with, both in peace time and under war conditions, under the following circumstances:—

(*a*) In the air of wells, disused mine galleries, underground shafts and tunnels, etc. Through the oxidation of organic and mineral matter in the soil, the composition of the air in such spaces may be seriously, and sometimes totally, deficient in oxygen on first entry. Even after thorough ventilation a constant watch on the purity of the atmosphere in the space must be maintained, as a fall in the barometric pressure tends to fill the confined area with residual nitrogen welling out of the surrounding strata.

(*b*) In air-tight compartments such as double-bottoms of ships. When these compartments are sealed for any length of time, the whole of the oxygen in the enclosed air may be used up by the ordinary process of the rusting of iron or steel bulkheads or through oxidation of the linseed oil in the paint commonly used in these spaces.

(*c*) In badly ventilated compartments, such as ships' holds or coal bunkers, in which oxidisable or oxygen-absorbing substances, such as grain, fruit, potatoes or coal, are stored.

Symptoms of anoxaemia.

In healthy adults, the percentage of oxygen in the air breathed must be reduced by about a third before any symptoms become obvious, unless heavy muscular work is being undertaken. If exposure to such an atmosphere be maintained, or if the concentration of oxygen be reduced still further, a chain of symptoms follows which is insidious in its onset and which is typical of

anoxaemia from whatever cause it may arise, namely:—headache, visual disturbance and mental dullness, loss of muscular power and of co-ordination, dyspnoea and weakened cardiac action; the power of judgment may be seriously impaired, while loss of memory is of common occurrence. There may be progressive cyanosis of the grey type with circulatory failure. With an extreme reduction in the oxygen percentage in the atmosphere, a condition of acute anoxaemia results in immediate loss of consciousness.

Treatment.

Immediate removal to fresh air is imperative, and, if the breathing has stopped, artificial respiration should be resorted to at once. This should be supplemented, if possible, by the administration of oxygen, by means of the Haldane oxygen apparatus at the rate of 8 to 10 litres per minute; the addition of 5 to 7 per cent. of carbon dioxide to the oxygen will greatly enhance the value of the latter by stimulating the respiration.

Precautions.

The precautions which may be taken may be summarised as follows:—

(*a*) Test the suspected atmosphere by means of a safety lamp, such as is commonly used in coal mines, for fear of the possible presence of explosive or combustible gases. A flame is very sensitive to any variation in the oxygen percentage of the air, and will be extinguished with a fall of 3 to 4 per cent. in the oxygen content at ordinary barometric pressure.

In this connection it may be useful to remember that a state of negative pressure may exist in an air-tight compartment owing to the reduction of its oxygen content, and that on opening the space the initial rush of pure air may dilute the atmosphere of the space in the immediate neighbourhood of the opening. This dilution may be sufficient to allow a candle to burn at the entrance, but may not affect the dangerous character of the air in remote parts of the same compartment.

(*b*) Thorough ventilation of all suspected spaces or compartments, including all remote corners.

(*c*) The invariable use of a life-line attached to the body of the first person to enter the space and of any unprotected rescuing personnel.

(*d*) The employment of the only sure safeguard both against the presence of noxious gases and the absence of a sufficiency of oxygen, namely, any type of self-contained oxygen breathing apparatus.

CHAPTER X

THE RECOGNITION AND DIFFERENTIAL DIAGNOSIS OF GAS CASES

A suitable introduction to this Chapter is the following quotation from the Official History of the War, Medical Services, "Diseases of the War", Vol. II (1923), page 317. It refers to the period of the battle of Loos (September, 1915), when phosgene was being used and before mustard gas was introduced.

"A report from the West Riding Casualty Clearing Station, which admitted 248 gas casualties, stated—'The majority of cases of gas poison-

ing received at this hospital showed no sign of that condition.' Other Medical Officers frankly admitted that they were so handicapped by their lack of experience of cases of gas poisoning that they were often in doubt whether they were dealing with men suffering from gas poisoning or not."

This report emphasises the difficulties of diagnosis that will always arise when persons are exposed to the dangers of gas poisoning and when neither they nor their medical attendants have had practical experience of the results of such poisoning.

58. The Importance of Accurate Diagnosis

In this chapter an attempt is made to indicate and deal with the difficulties of medical men to whom gas casualties are sent for treatment. .The proper treatment of such cases demands a correct diagnosis of the condition from which they are suffering. In order to avoid men being sent back to duty when they are in the quiescent period which may follow even severe gassing with a lethal gas such as phosgene, and also to prevent the evacuation as casualties of those suffering merely temporary discomfort from lachrymatory or sensory irritant gases, it is essential that all medical men should be familiar with the symptomatology of gas poisoning.

The doctor confronted with a supposed "gas" case is presented simultaneously with three problems. First: is the patient really suffering from the effects of gas at all, and, if so, to what extent? Second: if so suffering, then from the effects of what particular gas, or more properly from the effects of which of the recognised groups of poison gases? Third: does the case present such novel features as to suggest the use of some unknown gas?

All three questions are equally important. On the answer to the first will depend the main decisions as to the general line of management required. On the answer to the second will depend the decision as to the definite line of treatment to be adopted. If the case falls in the third category, it is essential that this vital fact should be made known immediately to those responsible for research upon the problems of treatment, of prevention, and of protection.

59. General Rules for Diagnosis

When there is doubt as to whether a person has been gassed or not, it is an important rule to withhold decision for 24 hours.

When dealing with early gas cases always give the patient the benefit of any doubt. Force is lent to this injunction by a study of the many reported cases of delayed action, and the severe and frequently fatal results of erring on the wrong side, particularly in cases due to lung irritant gas.

It is here that the value of enforced rest becomes so obvious. This period of rest, under fairly close expert supervision, can very well be taken in many cases in the patient's own home.

If no further symptoms develop within the 24 hours, close supervision may be discontinued.

It should be clearly recognised that the known and classified groups of war gases divide themselves broadly but importantly into two main classes —(*a*) those which possess the power of acting only upon the end organs of some specific sensory nerve or nerves, or upon some special tissue, and (*b*) those which can attack any living tissue with which they come in contact.

Recognition of this fact is one of the main points of intelligent differential diagnosis. Added importance attaches to this fact because the likelihood of meeting poisons which do not fall into one or other of the recognised groups is relatively very remote.

The development of obvious features of poisoning will generally make it possible in less than 24 hours to classify the case as having resulted from either skin vesicant or pulmonary irritant gas, while further differentiation may often be indicated by information as to the type of gas that had been employed over the area in which the casualty occurred.

For the diagnosis of cases due to the recognised war gases the outline chart given in Appendix D will, it is hoped, prove of value. By the method of elimination it should enable the doctor to fit the case into one or other of the four recognised groups of Tear Gas, Nose Irritant Gas, Lung Irritant Gas, and Blister Gas, or, failing that, to label it either as not due to gas at all, or else as due to a new factor.

The term "new factor", as used above, does not necessarily imply a new chemical substance. What it does mean is that effects are being produced which do not correspond with the recognised possibilities of the materials included under the standard four-group classification.

60. Recording of Case Histories

The problem is, therefore, how existing knowledge of chemical warfare agents can be used to the best advantage?

To obtain a history either from the patient, or from others in a position to supply the details, is of the utmost importance. History, in these cases, is of far greater value and importance than in the ordinary medical or surgical case. Because of its importance it should be fully detailed, and every effort commensurate with the exigencies of the situation should be made to verify the details.

The history of the manner of development of symptoms in the first 24 hours gives the clue to the diagnosis. The progress of the case in the next few days determines the time subsequently needed for convalescence. Both must be recorded, since the casualty may later pass to other centres for treatment, and the doctors there will require accurate information as to the early stages of the cases for proper guidance in their work.

Questioning, whether of patient or of others possessing or likely to possess the required knowledge, should follow some such lines as:—

(i) When did the alleged gassing take place, e.g. minutes, hours, or days previous?

(ii) Under what conditions did the alleged exposure to the gas take place, e.g. in the open or under cover, during action or at rest, etc.?

(iii) What did the gas smell or look like?

(iv) Was any special local condition or circumstance noted, e.g. type of bombs or shells falling (gas projectiles burst with much less violence than high explosive and do not flame like incendiary); peculiar smell and, if so, how described; unusual appearance of immediate area such as might be caused by persistent gas, etc.?

(v) For how long (seconds, minutes or hours) did the exposure last?

(vi) Was a respirator available, and of what type; if so, was it put on and for how long?

(vii) Were the effects produced immediately or after some delay, and, if the latter, after how long?

(viii) What effects, if any, were produced at the time?

(ix) What effects, if any, appeared later?

(x) Did the symptoms persist after escape from the polluted area or adjustment of the respirator?

When considering the replies (and in cases of serious doubt it is as well to make a written, tabulated statement, if the conditions of the moment make this at all possible), it must be remembered that quite as much value should be given to *negative* evidence as to positive.

As examples of negative evidence might be instanced the fact that the absence of any statement relating to the air passages in a case complaining of acute and immediate eye effects would be strong presumptive evidence against a lung irritant as the probable cause, whilst the absence of any acute effect at the time of exposure in a case with obvious eye trouble would be strong presumptive evidence in favour of mustard gas.

Particular attention is directed to the answers obtained to question (v) —"how long did the exposure last?"—and it must be very clearly understood, and quite definitely conveyed to the patient, that this question links up very directly with question (vi)—"was a respirator available and, if so, was it put on and for how long?"

Furthermore, the question relates both to the period of exposure without, *and* to the period of exposure after adjustment of the respirator.

These answers may have a very direct bearing upon prognosis and treatment. For instance, if a patient alleges exposure to mustard gas vapour without a respirator for some hours and yet exhibits only slight eye and/or skin effects 24 hours later there need be no great fear of any serious lung damage.

On the other hand if the alleged exposure had taken place at night and the fact could be elicited that the patient had slept most of the time, then the effects on the closed eye might be quite mild but the damage to the skin and lungs extensive and serious.

The significance of the replies to the questions will now be considered, and the various types of gases dealt with in turn.

61. Lachrymatory Gases: Diagnosis

These gases are easily recognisable by the *immediate* and severe lachrymation and the spasm of the eyelids that they produce. As met with under war conditions they are otherwise relatively harmless, and affected persons do not usually require any treatment whatsoever. Their effects are transitory except in the unusual event of a drop of actual liquid reaching the eye, as might happen to those close to a bursting bomb or shell. The condition is not serious and clears up quickly, in sharp contrast with that resulting from eye contamination by drops of liquid vesicant.

62. Nose Irritant Gases: Diagnosis

With these gases, the severity of the symptoms which may follow within a few minutes after inhalation may mislead an inexperienced person. The subject may look very distressed, and usually complains of intense pain in the naso-pharynx, throat and chest, with aching of the gums and teeth; cough-

ing, sneezing, profuse salivation and expectoration are generally marked, while retching, or even vomiting, may be present.

Such a patient is only too well aware that he has been acutely poisoned by gas, and may even think that he is in danger of losing his life. Paradoxically, despite the misery associated with such intense symptoms there is little serious danger associated with this type of gassing. The treatment consists of a brief period of rest coupled with the assurance that recovery will be rapid and complete.

63. Asphyxiants: Diagnosis

The asphyxiant gases—chlorine, phosgene, di-phosgene and chloropicrin —have each their own distinctive smell, and the history of the patient's subjective symptoms should be of value in determining to which particular gas gas he was exposed. Chlorine and chloropicrin vapours are intolerable to breathe even in concentrations which are relatively harmless; their action is in no way insidious, and, to persons provided with a respirator, this quality of extreme irritation serves as a safeguard against pulmonary injury. With the vapours of phosgene and of di-phosgene, which resemble each other fairly closely, the same does not hold good. Concentrations which may be detected readily enough by their smell of mouldy hay, and by the cough and slight lachrymation that they cause, may not prove in any way intolerable to breathe, and serious or even fatal results have followed the prolonged inhalation of these gases in low and relatively non-irritant concentrations.

If there is any reason to suppose that people have been exposed to these gases without respirators they should be examined with the greatest care. A history of slight lachrymation accompanying the inhalation of an asphyxiating gas which induced coughing will usually be given. The history may also describe the smell and the choking character of the gas, the cough it produced and the tightness of the chest felt at the time; symptoms which may have been accompanied by vomiting.

The greatest difficulty may be experienced in assessing whether a person is likely to become ill as the result of such inhalation. There may be no trace of cyanosis, but rather a mild pallor and coldness of the extremities due to shock. Attacks of coughing may recur which may be accompanied by a profuse frothy expectoration, while pain in the chest, particularly on deep inspiration, may be present and respiration may be rather shallow and rapid; headache and severe general fatigue may be pronounced.

Owing to the nervous state of such persons following an attack of gas, the character of the pulse is no sure guide unless it is unusually disturbed. Examination of the chest in the early stage after the inhalation of an asphyxiant gas usually reveals little abnormality. Before the development of extensive oedema a few moist rales may be heard in the axillae or over the base of the lungs, but their absence does not justify the supposition that the lungs have escaped injury.

An examination of the blood may also prove negative, as pulmonary oedema in the early stages does not necessarily give rise to any observable change in the concentration of the peripheral blood. When oedema is fully developed, the haemoglobin may reach 120, while the red cell count of the peripheral blood may even attain the figure of 8 million per cubic millimetre. But by this time the severe clinical features of acute pulmonary oedema will suffice to give the needed information.

The conclusion which we must therefore accept is that, following exposure to a gas of the asphyxiant group which has caused definite discomfort at the time, all should remain at rest under medical care for 24 hours. The only persons who may safely be discharged are those who are known to have been exposed to low concentrations for a short period (a few minutes) only, and who show no observable residual symptoms as described above.

64. Mustard Gas Vapour: Diagnosis

The faint garlic-like odour of mustard gas vapour may readily pass unnoticed; but if it is appreciated and the respirator is adjusted immediately, there will be no ill effects to the eyes and lungs. Some two to 24 hours later, however, skin burning may develop, if the person concerned remained in the mustard gas atmosphere, in ordinary clothing, while wearing his respirator. If, therefore, conjunctivitis, laryngitis and reddening of the skin are absent some 24 hours after exposure the person concerned may be assumed to have escaped injury from the gas.

If, however, about 24 hours after exposure, and without acute onset at the time of exposure, the eyes show conjunctivitis, with some loss of voice and a general reddening of the skin, usually most marked in the genital region, it may be concluded that the patient is suffering from the effects of mustard gas vapour.

A further period of 24 hours at the most will show whether the eye condition is of sufficient severity to require hospital treatment. As has been mentioned, although the eyes are the parts of the body most vulnerable to the effects of mustard gas vapour, the pulmonary and skin damage may exceed that caused to the eyes if the patient was exposed to the vapour during sleep.

65. Arsenical Vesicant Vapour (Lewisite): Diagnosis

Exposure to lewisite vapour has not the same insidious quality as a similar exposure to mustard gas vapour. Lewisite vapour has a very strong geranium-like odour, and in a minute or two causes sharp stinging in the nose and a burning pain in the chest. The degree of lachrymation produced is mild, but the markedly hostile character of the atmosphere is so quickly apparent to the senses that it compels those exposed to it to move out of it or to put on their respirators.

People who wear respirators in a high concentration of lewisite vapour, but are otherwise unprotected, may suffer from skin burning similar to that caused by mustard gas. These burns, however, differ from mustard gas burns in their more rapid development and the more severe irritation of the skin. As a rule there should be no difficulty in discriminating whether the vapour of mustard gas or lewisite was the causative agent in skin burns on persons who wore respirators, as, apart from any other distinctive sign, their clothing will have the strong pungent reek which is the particular characteristic of these vesicant arsenical vapours, and which cannot possibly be confused with the faint garlic-like odour of mustard gas.

66. Liquid Mustard Gas and Other Vesicant Liquids: Diagnosis

Liquid vesicant contamination of the skin or clothing of persons may be visible and easily recognised; on the other hand the presence of a few stray drops on the clothing can easily escape detection. To help in the immediate

recognition of liquid vesicants when sprayed from aircraft, detectors have been devised of which the simplest form is a stiff paper disc previously painted with a special yellow paint; a drop of the liquid vesicant falling on it immediately shows up as a bright red spot. Although this device is of the greatest value, it must be remembered that if the spray is sparse in its distribution—as may occur downwind of the spraying aircraft—it may miss the small area presented by a single detector disc.

The very faint smell of a few drops of mustard gas on the clothing and the lack of any skin irritation at the time give an insidious character to this form of attack, and contamination may only become evident when skin burning follows in a few hours. The treatment required is the removal of the clothing with the least possible delay, and thorough bathing of the body.

With lewisite or other arsenical vesicants it may not be so difficult to recognise a slight liquid contamination of the clothing, as, even if the strong smell has passed unnoticed, the skin beneath the contaminated spot usually begins to sting within a few minutes. These two features should render the detection of lewisite on clothing less difficult than is the case with mustard gas.

Under certain circumstances, however, people may be sprayed so heavily that there can be no doubt about their contamination; they may be visibly covered with drops of liquid, and their skin and clothing may reek of the contaminant. The immediate cleansing treatment of such persons is not generally the responsibility of the doctor; but if the eyes have received liquid contamination, immediate and continuous medical treatment will be necessary. The eye treatment required consists of copious lavage with warm saline repeated not less frequently than at hourly intervals. The rapidity with which such treatment can be carried out after eye contamination is of great importance; serious injury will in no case be averted, but permanent blindness may be prevented by prompt and thorough but gentle treatment.

67. Paralysant Gases: Diagnosis

Hydrocyanic acid gas and hydrogen sulphide gas are commonly known as paralysant gases. This description refers to their action in very high concentrations, when, by a paralysis of the respiratory centre in the brain, they quickly produce sudden unconsciousness and failure of respiration. In weak concentrations they are not harmless, but in comparison with other gases their action is of a mild character.

The recognition of casualties caused by these gases rests on a history of exposure to an atmosphere with a smell either of bitter almonds or rotten eggs; this being followed by rapid loss of consciousness, with or without convulsions and by failure of respiration. Subsequent recovery is usually rapid and without complications.

68. Carbon Monoxide: Diagnosis

The recognition by a doctor of cases of carbon monoxide poisoning in war time should not be difficult if he is familiar with the possible sources of such poisoning (as detailed in Section 47) and if he can obtain a history of the circumstances under which the condition occurred.

If unconsciousness has resulted, or even if breathing is failing, the treatment is immediate removal from the poisonous atmosphere and recourse to artificial respiration in pure air. After this the differential diagnosis should be considered.

The distinction of severe carbon monoxide poisoning from other comatose states rests chiefly on the history of the case, the pinkness of the lips in the early stages, and the examination of the blood by the test given in Section 47. Less severe degrees of poisoning occur with symptoms of headache, giddiness, breathlessness on exertion, weakness in the legs, and cerebral states varying widely from irritability to mental confusion. Such cases do not call for artificial respiration, but they need careful control lest over-exertion harm a heart which is already strained by anoxaemia.

69. Nitrous Fumes: Diagnosis

The recognition of cases of poisoning by these gases presents many difficulties in the early stages owing to the insidious nature of the onset. The incomplete detonation of a charge in a mining gallery, or the breathing of the vapour arising from the spilling of nitric acid on a wooden floor, may evoke a little coughing which soon subsides: hours later, however, an oedema of the lungs may follow and prove fatal.

The recognition of such potential casualties must to a large extent depend on the history of exposure; when such a history is established, the treatment must, as in cases of phosgene gassing, be largely that of watchful anticipation for a period of at least 24 hours. In the meantime, complete rest is imperative, and if pulmonary oedema should develop, its recognition and treatment follow closely the lines already laid down for cases of phosgene gassing.

70. Screening Smokes: Diagnosis

These smokes are harmless in the concentrations which form effective screens, and casualties do not therefore occur except in the case of men who may come in contact with the compounds before they have been dissipated in the form of smoke.

The Nova Scotia Medical Bulletin

Official Organ of The Medical Society of Nova Scotia.

Published on the 20th of each month and mailed to all physicians and hospitals in Nova Scotia. Advertising forms close on the last day of the preceding month. Manuscripts, preferably typed and double-spaced, should be in the hands of the editors on or before the 1st of the month. Subscription Price:—$3.00 per year.

VOL. XVIII. OCTOBER, 1939 No. 10

Evolution

THIS issue of the BULLETIN, with further information on the medical treatment of gas casualties, taken from *Air Raid Precautions Handbook, No.* 3, is presented without apologies. The enthusiastic attendance at the series of lectures given to the Halifax Medical Society by Major G. R. Burns, M.D., on this perhaps vital subject, and the comments made on the BULLETIN for September seem reason enough to complete the presentation begun last month.

It is interesting to note that *Handbook No.* 3, the text of our issue, is one of more than fifteen booklets prepared and issued by the Air Raid Precautions Department of the British Home Office, for the instruction of civilian populations.

If the physician-student cares to interest himself in the scientific fundamentals of the subject he would do well to approach it through comparative anatomy. Here, in the field to which modern obstetrics, embryology, physiology, eugenics and many of our most advanced sciences owe so much, is found the origin of gas warfare. With the skunk and his defenses most of us have had unhappy familiarity at some time or other. As with mustard gas, decontamination of person and clothing is an essential sequence of treatment.

In the sea, certain members of the octopus family eject an inky pigment, analagous to gas, which diffuses into the water producing a sort of smoke screen to protect them from attack. It is to be noted that the octopus, a dangerous enemy with great jaws and powerful tentacles, uses his "gas" not in attack, but as a defensive mechanism.

Most highly developed technique in gas warfare may be credited to a creature of the very air from which we expect attack, the Bombardier Beetle (you will observe his military title), who carries in his nether regions a sac full of acrid, brownish fluid. On being attacked from behind the bombardier hurls an explosive charge into the face of his enemy, with a loud pop!—burning, blinding and terrifying. Here is the modern trend, to combine gas with high

explosive. But the bombardier, in the evolutionary scheme, antedates our bombs by a million years.

In common with his lesser animal brothers, the human has found that a disabling, demoralizing gas is more effective than any known lethal ones. Rational, creative, immortal, he has been able to add one triumph. Skunk, octopus and the bombardier beetle, in their instinctive ignorance, have used gas only in self-defence. Man has made it an offensive agent of destruction. It has been said, he is just a little below the angels. There is, then, yet room for fresh glory, when he has attained their skill; when he can make carnage as did the cohorts of Heaven in aid of Israel. You remember, Byron tells of it (or if you prefer, the Book of Kings)—

> For the Angel of Death spread his wings on the blast,
> And breathed on the face of the foe as he pass'd;
> And the eyes of the sleepers wax'd deadly and chill,
> And their hearts but once heaved, and for ever grew still!

A.L.M.

Department of the Public Health

PROVINCE OF NOVA SCOTIA

Office—Hollis Street, Halifax, N. S.

MINISTER OF HEALTH - - - - HON. F. R. DAVIS, M.D.,F.A.C.S., Halifax

---

Chief Health Officer - - - - - - - DR. P. S. CAMPBELL, Halifax.
Divisional Medical Health Officer - - - DR. C. J. W. BECKWITH, D.P.H., Sydney.
Divisional Medical Health Officer - - - DR. J. J. MACRITCHIE, Halifax.
Divisional Medical Health Officer - - - Dr. J. S. Robertson, D P. H., Yarmouth.
Statistician and Epidemiologist - - - Dr. Harold Robertson, C. P. H., Halifax.
Director of Public Health Laboratory - - DR. D. J. MACKENZIE, Halifax.
Pathologist - - - - - - - - - DR. R. P. SMITH, Halifax.
Psychiatrist - - - - - - - - - DR. ELIZA P. BRISON, Halifax.
Sanitary Engineer - - - - - - - R. DONALD MCKAY, B.Sc., A.M.E.I.C.
Superintendent Nursing Service - - - MISS M. E. MACKENZIE, Reg. N., Halifax.

OFFICERS OF THE PROVINCIAL HEALTH OFFICERS' ASSOCIATION

President - - - - - - DR. H. E. KELLEY - - - - - - - Middleton
1st Vice-President - - - DR. R. C. ZINCK - - - - - - - Lunenburg
2nd Vice-President - - - DR. C. I. MACMILLAN - - - - - - Baddeck
Secretary-Treasurer - - - DR. P. S. CAMPBELL - - - - - - Halifax

COUNCIL

DR. W. D. FORREST - - - - - - - - - - - - - - Halifax
DR. J. E. LEBLANC - - - - - - - - - - - - - West Pubnico
DR. T. R. JOHNSON - - - - - - - - - - - - - Great Village

MEDICAL HEALTH OFFICERS FOR CITIES, TOWNS AND COUNTIES

ANNAPOLIS COUNTY

Hall, E. B., Bridgetown.
Braine, L. B. W., Annapolis Royal.
Kelley, H. E., Middleton (Mcpy. & Town).

ANTIGONISH COUNTY

Cameron, J. J., Antigonish (Mcpy).
MacKinnon, W. F., Antigonish.

CAPE BRETON COUNTY

Densmore, F. T., Dominion.
Fraser, R. H., New Waterford.
Francis, Bernard, Sydney Mines.
Sutherland, Harvey, Glace Bay.
McLeod, J. K., Sydney.
O'Neil, F., Sydney (County, South Side).
Murray, R. L., North Sydney.
Townsend, H. J., Louisbourg.
Gouthro, A. C., Little Bras d'Or Bridge, (Co. North Side).

COLCHESTER COUNTY

Eaton, F. F., Truro.
Havey, H. B., Stewiacke.
Johnston, T. R., Great Village (Mcpy).

CUMBERLAND COUNTY

Bliss, G. C. W., Amherst.
Gilroy, J. R., Oxford.
Hill, F. L., Parrsboro, (Mcpy).
Cochrane, D. M., River Hebert (Joggins).
Withrow, R. R., Springhill.
Stuart, C. E., Parrsboro.

DIGBY COUNTY

Belliveau, P. E., Meteghan, (Clare Mcpy).
DuVernet, Edward, Digby.
Rice, F. E., Sandy Cove, (Mcpy).

GUYSBORO COUNTY

Chisholm, D. N., Port Hawkesbury (Mulgrave).
Sodero, T. C. C., Guysboro (Mcpy).
Moore, E. F., Canso.
Monaghan, T. T., Sherbrooke (St. Mary's Mcpy).

HALIFAX COUNTY

Morton, A. R., Halifax.
Forrest, W. D., Halifax (Mcpy).
Payzant, H. A., Dartmouth.

HANTS COUNTY

Bissett, E. E., Windsor.
MacLellan, R. A., Rawdon Gold Mines (East Hants Mcpy).
Reid, A. R., Windsor, (West Hants Mcpy).
Shankel, F. R., Windsor, (Hantsport).

INVERNESS COUNTY

Chisholm, D. N., Port Hawkesbury.
Grant, T. E., Port Hood.
Proudfoot, J. A., Inverness.
McNeil, A. J., Mabou, (Mcpy).

KINGS COUNTY

Bishop, B. S., Kentville.
Bethune, R. O., Berwick, (Mcpy).
de Witt, C. E. A., Wolfville.
Moreash, R. A., Berwick.

LUNENBURG COUNTY

Marcus, S., Bridgewater (Mcpy).
Donkin, C. A., Bridgewater.
Donaldson, G. D., Mahone Bay.
Zinck, R. C., Lunenburg.
Zwicker, D. W. N., Chester, (Chester Mcpy).

PICTOU COUNTY

Blackett, A. E., New Glasgow.
Chisholm, H. D., Springville, (Mcpy).
Bagnall, P. O., Westville.
Crummey, C. B., Trenton.
Dunn, G. A., Pictou.
Parker, V. H. T., Stellarton.

QUEENS COUNTY

Ford, T. R., Liverpool.
Smith, J. W., Liverpool, (Mcpy).

RICHMOND COUNTY

Deveau, G. R., Arichat, (Mcpy).

SHELBURNE COUNTY

Corbett, J. R., Clark's Harbour.
Fuller, L. O., Shelburne, (Mcpy).
Dinsmore, J. D., Port Clyde, (Barrington Mcpy).
Lockwood, T. C., Lockeport.
Churchill, L. P., Shelburne, (Mcpy).

VICTORIA COUNTY

MacMillan, C. L., Baddeck, (Mcpy).

YARMOUTH COUNTY

Hawkins, Z., South Ohio, (Yarmouth Mcpy).
Caldwell, R. M., Yarmouth.
Lebbetter, T. A., Yarmouth, (Wedgeport).
LeBlanc, J. E., West Pubnico, (Argyle Mcpy).

Those physicians wishing to make use of the free diagnostic services offered by the Public Health Laboratory, will please address material to Dr. D. J. MacKenzie, Public Health Laboratory, Pathological Institute, Morris Street, Halifax. This free service has reference to the examination of such specimens as will assist in the diagnosis and control of communicable diseases: including Kahn test, Widal test, blood culture, cerebro spinal fluid, gonococci and sputa smears, bacteriological examination of pleural fluid, urine and faeces for tubercle or typhoid, water and milk analysis.

In connection with Cancer Control, tumor tissues are examined free. These should be addressed to Dr. R. P. Smith, Pathological Institute, Morris Street, Halifax.

All orders for Vaccines and sera are to be sent to the Department of the Public Health Metropole Building, Halifax.

Report on Tissues sectioned and examined at the Provincial Pathological Laboratory, from September 1st., to October 1st., 1939.

During the month, 219 tissues were sectioned and examined, which with 29 tissues from 6 autopsies, makes a total of 248 tissues for the month.

Tumours, simple	29	
Tumours, malignant	29	
Tumours, suspicious of malignancy	..	
Other conditions	161	
Tissues from 6 autopsies	29	
		— 248

Province of Nova Scotia Division of Vital Statistics

Provisional Monthly Report—August 1939

	August, 1939				July, 1939
	Total	Male	Female	Rate	Rate
No. of live births	942	492	450	20.2	21.7
No. of stillbirths	37	22	15	37.8**	32.5**
No. of deaths	358	195	163	7.7	10.7
No. of deaths under 1 year of age	42	22	20	44.6*	54.4*
No. of deaths from puerperal causes	4	...	4	4.2*	6.9*

Causes of Death	Int. List No.	August, 1939 Total	Male	Female	Rate	July, 1939 Rate
Typhoid Fever	1	1	1	..	.....	.....
Measles	....	..	..	..	.....	.....
Scarlet Fever	....	..	..	..	.....	.....
Whooping Cough	9	10	4	6	21.5	.....
Diphtheria	....	..	..	..	.....	.....
Influenza	11	3	2	1	6.4	21.5
Pulmonary Tuberculosis	23	28	15	13	60.2	79.5
Other forms of Tuberculosis	24-32	3	1	2	6.4	8.6
Cancer and other Malignant tumors	45-53	52	23	29	111.7	120.3
Cerebral homorrhage, thrombosis and ombolism	82a, 82b	12	7	5	25.8	36.5
Diseases of the Heart	90-95	57	35	22	122.5	204.1
Diseases of the Arteries	96, 97, 99, 102	39	18	21	83.8	83.8
Pneumonia (all forms)	107-109	12	7	5	25.8	45.1
Diarrhea and Enteritis under 2 yrs. of age	199	6	5	1	6.4*	2.0*
Nephritis	130-132	19	9	10	40.8	73.1
Diseases of Early Infancy	158-161	13	5	8	13.8*	31.7*
Accident	175-195	26	20	6	55.9	73.1

* Rate expressed as number of deaths per 1000 live births.
**Rate expressed as number of stillbirths per 1000 total births.

Provisional Monthly Report of Births and Deaths August, 1939.

	BIRTHS									DEATHS															
	Total Births	Live Births Total	Legitimate M.	Legitimate F.	Illegitimate M.	Illegitimate F.	Still Births Total	Still Births M.	Still Births F.	Total	All Causes M.	All Causes F.	Maternal	Under 1 year of Age	Whooping Cough	Influenza	Pulmonary Tbc.	Other forms of Tbc.	Cancer	Cere. hem. Embolism Thrombosis	Heart Disease	Disease of the Arteries	Pneumonia All Forms	Diarrhea under 2 years	Nephritis	Diseases of Infancy	Accident
Nova Scotia	979	942	458	431	34	19	37	22	15	358	195	163	4	42	10	3	28	3	52	12	57	39	12	6	19	13	26
Annapolis	35	33	17	15	..	1	2	1	1	13	7	6	..	..	..	..	..	..	4	1	2	1	..	..	1	..	3
Antigonish	28	24	18	5	..	1	4	2	2	11	4	7	..	1	..	..	1	1	3	2	1	1	..	..	..	1	..
Cape Breton	198	193	95	86	7	5	5	4	1	64	39	25	..	16	4	1	3	..	6	..	13	9	4	3	4	3	6
Colchester	44	44	21	21	1	1	..	..	..	17	8	9	..	4	2	..	..	..	1	1	4	1	..	..	..	2	2
Cumberland	68	65	32	30	2	1	3	1	2	30	16	14	2	3	1	..	1	..	4	1	3	5	1	..	3	1	1
Digby	31	29	14	14	1	..	2	2	..	3	1	2	..	1	..	..	..	..	1	..	..	1	..	..	..	1	..
Guysboro	22	20	7	13	..	..	2	2	..	4	3	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Halifax	191	185	96	71	13	5	6	4	2	3	44	39	1	8	3	..	11	1	13	3	9	11	5	1	5	1	3
Hants	35	33	10	19	3	1	2	1	1	14	7	7	..	..	..	..	2	..	3	..	3	..	1	..	1	..	..
Inverness	36	35	18	17	..	..	1	..	1	17	8	9	1	2	..	..	1	1	2	..	3	3	..	1	1	..	1
Kings	59	57	26	29	2	..	2	..	2	20	11	9	..	..	..	2	4	..	2	3	1	2	..	..	2	..	..
Lunenburg	61	57	23	31	2	1	4	1	3	14	7	7	..	1	..	..	..	..	3	..	2	1	1	..	..	1	1
Pictou	53	53	24	25	3	1	..	..	..	23	13	10	..	..	..	..	1	..	4	..	6	2	..	..	..	..	5
Queens	23	23	11	12	..	..	..	..	..	5	3	2	..	1	..	..	1	..	1	..	..	..	..	1	..	..	1
Richmond	11	11	8	3	..	..	..	..	..	9	6	6	..	3	..	..	..	..	1	..	1	..	..	..	1	1	..
Shelburne	34	32	17	14	..	1	2	2	..	11	5	3	..	1	..	..	1	..	2	..	3	2	..	..	1	1	..
Victoria	16	15	6	8	..	1	1	1	..	4	2	2	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..
Yarmouth	34	33	15	18	..	..	1	1	..	16	11	5	..	1	..	..	1	..	2	1	6	..	..	..	..	1	2

Note: These figures are based on the Birth and Death certificates received by the Division of Vital Statistics, Halifax, N. S., up to and including September 10, 1939 and represent the number *registered* with the Division Registrars during the month of August, 1939.

Society Meetings

Antigonish-Guysboro Medical Society

The Antigonish-Guysboro Medical Society was organized October 11th, 1939, at St. Martha's Hospital, Antigonish.

Officers elected for the ensuing year are as follows:

Hon. President: Dr. J. J. Cameron, Antigonish.
Hon. Vice-President: Dr. E. F. Moore, Canso.
Hon. Secretary: Dr. P. S. Campbell, Halifax.
President: Dr. J. J. Carroll, Antigonish.
1st Vice-President: Dr. J. S. Brean, Mulgrave.
2nd Vice-President: Dr. T. T. Monaghan, Sherbrooke.
Secretary-Treasurer: Dr. C. B. Smith, Goldboro.
Executive: Dr. W. F. MacKinnon, Antigonish; Dr. Clare Sodero, Guysboro; Dr. D. J. MacMaster, Antigonish; Dr. J. L. MacIsaac, Antigonish.
Representatives on the Executive of the Medical Society of Nova Scotia: Dr. J. L. MacIsaac, Antigonish; Dr. T. B. Murphy, Antigonish.

The next meeting of the Society will be held at St. Martha's Hospital on Wednesday, November 1st, 1939.

OBITUARY

THE BULLETIN extends sympathy to Dr. A. M. Marshall of Halifax on the death of his father, Mr. G. R. Marshall, which took place in Halifax on September 23rd, after a lengthy period of illness. Mr. Marshall was born in Springfield, Annapolis County, seventy-six years ago. He came to Halifax early in life, and was first principal of Richmond School and later the Chebucto Road School. He retired from teaching in 1924. Surviving besides his widow, the former Elizabeth Cameron, are a daughter, Mary, a teacher at Quinpool Road School, and two sons, Dr. Arthur M. Marshall, Halifax, and Dr. Clyde Marshall, former Psychiatrist of Nova Scotia, now a professor at Yale University.

The BULLETIN also extends sympathy to Dr. C. S. Morton of Halifax, on the death of his mother, Mrs. Elizabeth Pallem Morton, which occurred at Owen Sound, Ontario, on October 16th, at the home of her grand-daughter, Mrs. Don Whyte. Mrs. Morton was the last surviving member of the family of Dr. James Pallem, London physician, who settled at Chatham, N. B. early in the last century and for fifty years practised throughout New Brunswick. Survivors include two sons, Arthur C. Morton of Montreal and Dr. C. S. Morton of Halifax, and two daughters, Mrs. W. H. Talbor-Crosbie, Guelph, Ontario, and Mrs. Arthur J. Dove, Toronto.

Personal Interest Notes

A MARRIAGE took place in September at the home of Mr. and Mrs. M. A. Miller, North Sydney, when their only daughter, Reta Virginia, became the bride of Dr. A. R. Gaum, son of Mr. and Mrs. Louis Gaum, Sydney. Immediately following the ceremony Dr. and Mrs. Gaum left on a motor trip to New York City and the Southern States.

Dr. and Mrs. S. W. Williamson of Yarmouth spent a few days recently in Providence, R. I., visiting relatives.

Dr. A. C. Fales of Wolfville, was the winner of first prize in the Grey Trout class of the fishing contest recently concluded by the Halifax *Chronicle* during the past season. Dr. Fales' prize winning fish was 28½" long and weighed 9¼ lbs.

At one of the Kiwanis luncheons in September at Yarmouth the guest speaker, Dr. J. S. Robertson, District Medical Officer, was introduced by the chairman, Dr. C. K. Fuller. Dr. Robertson spoke on "Milk", and gave his listeners a clear description of the work being done to educate the farmers to produce and market clean and wholesome milk. He invited the members to co-operate in helping the officials to attain this goal.

The BULLETIN regrets to learn that Dr. E. T. Granville of Bedford is ill with pneumonia at the Halifax Infirmary. His condition is progressing favourably. Dr. Kenneth Lemon is taking charge of Dr. Granville's practice during his illness.

The BULLETIN extends congratulations to Dr. and Mrs. H. L. Mellish of Havelock, N. B., on the birth of a son on September 29th, and to Dr. and Mrs. C. J. W. Beckwith of Sydney on the birth of a son.

Dr. and Mrs. W. W. Bennett, who have been in England since last January, have returned to their home in New Germany.

Dr. and Mrs. A. C. Gouthro of Little Bras d'Or Bridge, who have been on a visit to New York have returned home.

PHYSICIAN WANTED

A letter has been received by the secretary from a prominent member of the community of Prince William, N. B. asking help in securing a physician for that district. Any physician interested may secure further information from this office.

H. G. S.

Science Must Go Forward

Advance will continue to be made, stated Dr. Penfield, after reference to the advent of anaesthesia, asepsis, the X-ray, vaccines and vitamines, "with libraries full of ill-digested knowledge of the nerves, glands and allergical reactions. We cannot turn back, any more than you would give up your auto for a horse and carriage. The leadership must lie in the universities. It has become too complicated for any other agency."

If that be true of medicine, then, said the speaker, it was true of political economy. Today, faced by complex economic and political problems at home and abroad, who can chart a true course unless he can see the truth without bias? To see the truth, to see it in perspective and to teach it, that was the eventual purpose of universities, and their growth must be provided for on a more generous and more comprehensive scale.

Dr. Penfield traced the history and the purposes of the old universities in Europe,—Rome, Salerno, Bologna, Paris, Berlin and Oxford, some of which acted as trade guids, others for research. Touching on Berlin, here marked: "In the post-war period the German universities have carried on largely by virtue of their inertia, and unless the attitude of the present totalitariangovernment alters, their prestige as centres of independent investigation may well vanish."

In stating that the fate of universities had to some extent depended upon native wit but more largely upon gifts of money from church and state, Dr. Penfield said no nation seemed to be able to pay for both war and education, pointing out that the annual expenditure for higher education in England was only about £5,000,000. Billions were borrowed for war, but no country ever borrowed for education. He referred to the munificence of gifts of Lord Nuffield, who in donating an extra two million over what he had given already, stressed the importance of providing permanently for scientific advance and direct application of these advances to medical practice. Nuffield had emphasized the analogy between medicine and political science, and had further shown the necessity of bringing the gap between medical research and political practice.

What Every Woman Doesn't Know—How to Give Cod Liver Oil

Some authorities recommend that cod liver oil be given in the morning and at bedtime when the stomach is empty, while others prefer to give it after meals in order not to retard gastric secretion. If the mother will place the very young baby on her lap and hold the child's mouth open by gently pressing the cheeks together between her thumb and fingers while she administers the oil, all of it will be taken. The infant soon becomes accustomed to taking the oil without having its mouth held open. It is most important that the mother administer the oil in a matter-of-fact manner, without apology or expression of sympathy.

If given cold, cod liver oil has little taste, for the cold tends to paralyze momentarily the gustatory nerves. As any "taste" is largely a metallic one from the silver or silverplated spoon (particularly if the plating is worn), a glass spoon has an advantage.

On account of its higher potency in Vitamins A and D, Mead's Cod Liver Oil Fortified With Percomorph Liver Oil may be given in one-third the ordinary cod liver oil dosage, and is particularly desirable in cases of fat intolerance.

Coughs, Colds and Other Respiratory Diseases

With the onset of winter weather, with its wide fluctuations of temperature, coughs and colds become very prevalent and a source of much invalidism.

One of the best methods of treating an irritating cough, and an aid in preventing its progress is the prompt application of Antiphlogistine to the chest. Heated to a comfortable degree of heat and adequately applied—to the depth of ½ inch—it will not need renewing for 24 hours. In any case of bronchitis, cough, influenza, pleurisy, etc., its early application is an aid in ameliorating the symptoms and of promoting early resolution. In addition, patients should be instructed concerning the necessity of maintaining proper ventilation of their rooms at all times. Stuffy, overheated rooms are not only detrimental to a respiratory patient, but are a fruitful cause of respiratory disorders.

For Immediate Release

Following a request to the Canadian Custodian, the Montreal Office of Merck & Co., Limited, a subsidiary of Merck & Co., Inc., manufacturing chemists of Rahway, N. J., has received official notification from the Department of the Secretary of State, Ottawa, through the office of the Custodian, that neither Merck & Co., Inc., nor Merck & Co., Limited, are connected with any foreign concern on the British "blacklist".

The recent press despatch from New York included the name of "the Merck chemical company" among several hundred foreign concerns on the "blacklist" of the British Government. Subsequent publication of the official list in full disclosed that the company referred to was "Casa Chimica Merck" of Brazil and "Merck Quimica Argentina" of the Argentina, South America.

Commenting on this situation, George W. Merck, president of Merck & Co., Inc., stated that the Rahway concern is an American company, incorporated under the laws of New Jersey; that its officers and directors are American citizens; that more than 99½% of the stock is owned by approximately a thousand people in all walks of life, residing in the United States and Canada; that the company has no corporate connection with any concern outside of the United States, with the exception of its wholly-owned Canadian subsidiary, Merck & Co., Limited of Montreal; and that no foreign concern has any interest in Merck & Co., Inc.

Merck & Co., Inc. manufactures and distributes nearly 3,000 drugs and chemicals for the medical, dental, pharmaceutical and allied professions; for various branches of industry, and the general public.