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MEDICAL MANUAL OF CHEMICAL WARFARE

1943

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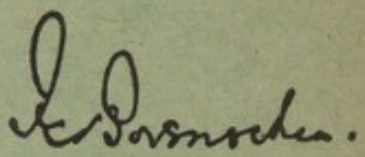

THE WAR OFFICE,

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CHAPTER I

GENERAL DESCRIPTION OF WAR GASES

1. Definition of " gas " in chemical warfare.

In chemical warfare the term " gas " is applied to any substance, whether solid, liquid, or vapour, which is used for its poisonous, irritant, blistering, burning or corrosive effects.

Gases may be liberated in the air as vapour or smoke ; or may be brought into contact, in liquid or solid form, with personnel, material, or ground.

2. The use of gas in the Great War.

Chemical warfare in the modern sense was first introduced by the Germans in April, 1915, on the Western Front, chlorine gas being used by them in successive attacks until May of the same year, when tear gases also were used. These attacks found the Allies not only unprepared but also inexperienced in the effects of chlorine. Casualties were very heavy, and means of protection had to be improvised at once.

The first official respirator (a cotton pad soaked in hyposulphite of soda, glycerine and sodium carbonate) was issued in May, 1915, and after that date defence, on the whole, kept ahead of attack—so much so that the use of phosgene gas by the Germans in December, 1915, found the Allies relatively well protected against its effects.

With a view to overcoming this protection the Germans introduced the arsenicals (or " nasal irritants ") and mustard gas. The former were intended to penetrate the box respirator which was then in use by the Allies, while the latter, having a very faint smell and causing no immediate irritation, might be expected to take effect before the need for putting on the respirator was realized. Comparative failure attended the use of the arsenicals ; mustard gas, on the other hand, was highly successful, for, in addition to its insidiousness, it could attack all parts of the body not protected by the gas mask and it had the great offensive value of persistency.

3. Probable changes in the use of gas.

In view of the efficiency of the modern respirator, the ease with which it can be adjusted, and the knowledge of chemical warfare instilled into troops in their anti-gas training, it is probable that the old wartime lethal agents, such as chlorine or phosgene, will be less extensively used in future against armies in the field.

Mustard gas, however, lends itself to novel methods of distribution. In the Great War most of the mustard casualties were caused by vapour rising from ground contaminated by shells ; but aeroplanes can now spray the gas directly over troops in the open or drop it in bombs over areas that are to be denied the enemy. By means of bombs on aerodromes, dockyards and dumps, it is possible to contaminate aircraft, stores, food and ammunition so that they become dangerous to handle.

Other powerful vesicants, including lewisite, may also be employed.

4. Objects to be achieved by the use of gas.

Gas may be used with one or more of the following objects in view :—

(*a*) To produce casualties by using it directly against personnel.
(*b*) To produce casualties by contaminating stores and material.
(*c*) To harass troops by compelling them to wear respirators for prolonged periods, and thus to reduce their mobility, efficiency and endurance, and, possibly, lower their morale.
(*d*) To deny ground by rendering it dangerous to occupy.
(*e*) To contaminate food and similar stores.
(*f*) To lower the morale of the civil population and induce a will to compromise or surrender.

The type of gas used will vary with the object in view. Lethal gases such as phosgene will cause serious casualties among troops, even though they be equipped with efficient respirators, unless their training against gas is of a high order. Similarly, mustard gas used in shell or from aircraft will cause numerous casualties, in spite of respirators and other protective equipment, if training in their use is defective. Even against troops fully equipped and trained, mustard gas will act as a powerful harassing agent, although casualties in large numbers may be avoided.

The arsenical irritants and lacrimators, when directed against personnel equipped with respirators and trained in their use, should not produce casualties, but they may impair efficiency.

5. Concentration, and period of exposure thereto.

It is evident that the higher the concentration of the gas, the shorter will be the period of exposure required to produce casualties among personnel exposed to it ; and the converse holds good.

High concentrations can be obtained by liberating the gas in confined or sheltered areas ; or, under conditions of open warfare, by the expenditure of an enormous quantity of gas. In the latter case, however, the element of surprise is essential if adequate effects are to be obtained on trained personnel, especially when highly irritant gases are used.

It should also be borne in mind that, with persistent liquids, a high temperature will tend to produce a high concentration of vapour at the cost of persistence, whereas a low temperature will act in the reverse direction.

6. Factors governing the use of gas.

It is obvious that a strong wind will rapidly dilute and disperse all concentrations of non-persistent gases, while in the case of a persistent gas the rate of evaporation of the liquid will be increased, thus tending to clear the area more rapidly. With a low wind velocity, on the other hand, a high local concentration may be obtained with both types of gases.

Temperature plays an important part, both by influencing the diffusion of the gas by convection currents and by affecting the persistence of such liquids as mustard gas ; thus warm, sunny weather is inimical to the most effective use of gas. Very cold weather, however, also has its disadvantages, as it reduces very markedly the immediate value of persistent gases : in the case of mustard gas, for example, evaporation will be very greatly reduced as this liquid freezes at comparatively high winter temperatures (about 44° F. for the crude product). On the other hand, mustard gas in the frozen state is not by any means inert, as contact with it will still produce a burn. If the frozen mustard gas is carried (*e.g.* on boots) to warmer surroundings it will soon liberate effective vapour concentrations.

Non-persistent gases are best used when air turbulence is minimal (*e.g.* dawn, dusk, or a clear still night). For persistent gases the desiderata depend on whether long persistence or high vapour concentration is the aim : rain tends to destroy them, while humidity retards vaporization.

The character of the terrain is also important. All gases hang about built-up areas, enclosed spaces, woods, valleys, and hollows, where they are less exposed to sun and wind. Persistent gases penetrate, and persist in, sand or soft ground, but evaporate from a hard surface.

7. Classification of gases.

Chemical warfare agents have been subjected to various classifications according to our knowledge of their physical or their physiological properties. In the first year of their use in the Great War, when both the deadly chlorine and the comparatively innocuous " tear " gases were employed, a broad classification into " *lethal* " and " *harassing* " types was adopted. Later, when mustard gas was introduced, a new and tactically important distinction between " *persistent* " and " *non-persistent* " gases was recognized.

From the medical standpoint, however, these physical characteristics of gases were less helpful, and accordingly a new " physiological " grouping was introduced which tabulated the various gases according to their main action on the body. This action may, to a large extent, be regarded as specific, as chemical warfare agents usually produce their most marked effects on some particular structure of the body, especially when they are present in low concentrations. Thus we have " pulmonary " and " sensory " irritants, whose main effects are produced on the lungs and sensory nerve endings of the upper respiratory tract respectively ; the " lacrimators," which affect chiefly the eyes ; the " vesicants " or blistering gases, by which any part of the body surface may be attacked ; the " paralysants," which act directly on the nervous system, etc.

Such a classification is by no means rigid, for some of the gases really possess the characters of more than one of the groups ; but it has the merit of convenience. With a view to combining both tactical object and physiological effect, the following classification may be adopted :—

I.—VESICANTS

Vesicants or " blister gases," such as *mustard gas* (H.) and *lewisite*. These are substances which, whether in the liquid, solid or vapour state, will damage any part of the body with which they come in contact. Typical effects of the vapour are acute conjunctivitis, inflammation of the mucous membranes lining the respiratory tract, and burning of the skin varying from erythema to vesication. The effect of the liquid on the skin is severe vesication.

When death occurs after the inhalation of mustard gas, it usually supervenes on a septic bronchitis with broncho-pneumonia.

II.—LETHAL

(*a*) LUNG IRRITANTS or " choking gases." These gases, which include *chlorine*, *chloropicrin* (P.S.), *phosgene* (C.G.) and *diphosgene*, are essentially lung irritants exerting their main action on the deeper, as well as the upper, respiratory passages. They are used primarily as lethal agents, and, in the absence of an efficient respirator, they usually cause pulmonary oedema.

(*b*) PARALYSANTS, such as *hydrocyanic acid* and *hydrogen sulphide*. Although these highly toxic gases did not prove a success in the late war, they are included here because of their potential danger if used in different circumstances. In high concentration, both these gases can produce death rapidly through paralysis of the respiratory centre.

(*c*) ARSENIURETTED HYDROGEN (ARSINE).—This gas exerts its poisonous action only after absorption through the lungs into the body. It causes destruction of the red corpuscles in the blood, hæmoglobinuria, jaundice and anæmia, associated with damage to the kidneys and liver.

III.—HARASSING

(*a*) LACRIMATORS or "tear gases," such as *ethyliodoacetate* (K.S.K.), *bromobenzyl cyanide* (B.B.C.), and *chloracetophenone* (C.A.P.). Even low concentrations of the gases given off by these compounds will immediately irritate the eyes, causing profuse lacrimation and intense spasm of the eyelids —symptoms, however, which disappear on leaving the contaminated area. In very high concentrations they may act as lung irritants.

(*b*) NASAL IRRITANTS or "nose gases." These are organic arsenical compounds such as *diphenylamine-chlorarsine* (D.M.) and *diphenylcyanarsine* (D.C.). These solid arsenicals, when suitably dispersed, produce clouds of minute particles which, if inhaled even in exceedingly low concentration, will give rise to acute physical distress with intense pain in the nose and chest, lacrimation, salivation and even vomiting. The symptoms, however, although alarming at the time, usually subside within an hour after removal from the gassed area.

IV.—CORROSIVES AND INCENDIARIES

CORROSIVES.—Such substances as *chlorosulphonic acid* (C.S.A.), *titanium tetrachloride* (F.M.) and a number of the *chlorinated hydrocarbon* series, are generally used to produce smoke screens. They are capable of causing corrosive burns and, like some of the inorganic acids, might possibly be employed offensively.

INCENDIARIES.—*Phosphorus* (W.P.), which likewise provides smoke screens, is also a dangerous incendiary substance distributed in bomb, shell or grenade. *Thermite* and *fuel oils* are now familiar in the same rôle.

V.—ACCIDENTAL

(*a*) CARBON MONOXIDE.—This dangerous gas is frequently met with in the course of mining and tunnelling operations, in the interior of burning buildings, in tanks, pill-boxes and gun emplacements which are badly ventilated, and, generally, wherever combustion occurs in the absence of an adequate supply of oxygen. Thus it is found in confined spaces following the burst of a high-explosive shell, or when slow-combustion stoves, charcoal braziers or internal combustion engines are used in such spaces. It is present in ordinary illuminating gas, leakage of which may cause serious poisoning. The gas produces its insidious effects through its well-known interference with the respiratory functions of the blood. *Ordinary respirators give no protection.*

(*b*) NITROUS FUMES.—These gases are given off by burning cordite, or when detonation of nitro-explosives is incomplete. They act as powerful and very insidious lung irritants, with delayed symptoms resembling those of phosgene poisoning, and it is important to remember that they are often accompanied by carbon monoxide. Respirators generally afford partial protection against nitrous fumes, but none against carbon monoxide.

(*c*) FUMES which may be encountered in FIRE FIGHTING.—In addition to the risk of encountering carbon monoxide, and possibly nitrous fumes, when fighting fires in confined spaces in wartime, danger may also arise from the toxic gases evolved by fire-extinguishing chemicals. Apart from its possible toxicity, such an atmosphere may be seriously deficient in oxygen.

CHAPTER II

VESICANT OR "BLISTER" GASES

Mustard gas (H.) (the "Lost" or "Yellow Cross" of the Germans and the "Yperite" of the French) was by far the most effective chemical agent used in the Great War for the production of casualties.

Lewisite, an arsenical preparation developed towards the end of the Great War, is another powerful blistering agent like mustard gas; but, unlike the latter, it has an immediate irritant action on the eyes, nose, and respiratory tract and (in the liquid form) on the skin, and is therefore more easily detected.

Ethyldichlorarsine, used by the Germans in 1918, is not so strongly vesicant as lewisite or mustard gas, but like lewisite is irritating to the respiratory tract.

It is important to remember that the phenomenal development of aircraft since the Great War has been the means of introducing new methods of gas dispersion. Not only has the limited range of artillery been surpassed by the use of air gas bombs, but a more widespread, and almost instantaneous, dissemination of liquid agents has been assured by the adoption of aircraft gas spray—factors which have greatly enhanced the possibilities of vesicants as chemical weapons and correspondingly increased the difficulties of defence against them.

MUSTARD GAS

8. Physical and chemical properties of mustard gas.

Appearance.—In the pure state mustard gas (dichlorethyl sulphide) is a clear, heavy and somewhat oily fluid, straw-coloured, but in the crude form it is a heavy, dark-coloured, oily liquid.

Odour.—In the absence of chemical methods for the ready detection of mustard gas, the sense of smell is the most reliable guide to its presence. The mustard-like or garlicky odour, though faint in low concentrations, is characteristic and must be memorized in anti-gas training. But mustard gas may produce casualties in concentrations the smell of which may readily escape notice.

Boiling point and vapour pressure.—The boiling point of mustard gas (217° C. or 423° F.) is high, and its vapour pressure is correspondingly low (0·05 mm. Hg at 10° C., and 0·45 mm. Hg at 40° C.)—hence its slow vaporization at ordinary temperatures and its consequent quality of persistence.

Freezing point.—The freezing point of pure mustard gas is 14·4° C. (58° F.), while that of the crude variety is considerably lower, viz. 7° to 8° C. (44° to 45·4° F.)—somewhat high freezing points which limit the usefulness of the gas in cold weather, although contact with the frozen material is still a source of danger. In these circumstances the odour may not be detectable.

Density.—Mustard gas has a high specific gravity (1·28 at 15° C. or 59° F.) and, as it is not miscible with water, it readily sinks to the bottom when added to it.

Solubility.—Although mustard gas is only very slightly soluble in water (under 1 per cent.), both the liquid and the vapour are freely soluble in animal oils and fats, and it is because of this lipoid solubility that mustard gas finds an easy entry into the skin. Other substances that readily dissolve mustard

gas are alcohol, ether, petrol and kerosene, carbon tetrachloride, acetone, carbon disulphide, and many other organic solvents.

Stability.—Both physically and chemically mustard gas is a stable substance ; it is unaffected by normal ranges of atmospheric temperature, though simple heat disperses it by hastening evaporation. It is only very slowly hydrolysed by water ; hot water, however, hastens this decomposition, the products of which (hydrochloric acid and thiodiglycol) in ordinary circumstances are practically harmless. For its chemical neutralization strong reagents are usually required, such as chlorine (as in bleaching powder), potassium permanganate, or other strong oxidizing agents.

Powers of penetration.—Liquid mustard gas has great powers of penetration, and will soak into all but the most impervious surfaces such as smooth metals, glass and glazed tiles. Like oil it is readily absorbed by clothing, but when small drops of liquid mustard gas fall on clothing any injury which results is as a rule caused by the passage of vapour rather than by the penetration of actual liquid.

Persistence.—Mustard gas is very persistent. Depending on weather conditions it may remain in a liquid and dangerous state for days or even weeks. It may persist under the surface of ground which appears free of the liquid. Frozen mustard gas may continue to give off vapour slowly for months. As the temperature rises the quantity of vapour given off will increase. The frozen liquid may therefore be carried by boots, etc., to warmer surroundings where it will melt and vaporize.

9. Toxic properties of mustard gas.

Toxicity.—Mustard gas is a poison which, besides causing local damage to the tissues with which it comes in contact, has harmful effects on the body as a whole. Both liquid and vapour produce local burns. Any part of the body exposed to the gas will suffer, though sites naturally warm and sweaty are most liable to be attacked.

Vapour concentrations.—Atmospheres which contain low concentrations of mustard gas are particularly dangerous, since the presence of the poison may escape detection and exposure may thus be prolonged.

Insidious characteristics.—The fact that there is no immediate irritation of the skin on contact with the liquid, nor of the eyes and respiratory tract on entering moderate concentrations of the vapour, constitutes one of the more serious dangers of this gas, as contamination may be unsuspected. Even when the gas has been detected by its characteristic odour, the sense of smell is soon dulled, or even lost, and the odour will cease to be appreciated. If, however, the respirator is speedily adjusted the odour will be detected whenever the respirator facepiece is raised to " test for gas." It is important to remember, also, that harmful concentrations of the gas can easily be masked by innocuous smokes or by fumes from high explosive.

Delayed action.—After exposure to mustard gas vapour or contact with the liquid itself no effects are noticed for some time. Signs and symptoms may not begin to appear until after the lapse of some hours, or even days, depending on the concentration of the vapour in the atmosphere and the length of exposure thereto. By this time it is too late to ward off the effects of the gas, and casualties result.

Delayed healing.—The affected tissues are devitalized, they are easily injured by rubbing or pressure, and they are very prone to infection. Where the gas has penetrated deeply, the healing process, even though sepsis be excluded, is

very slow owing to damage to capillaries, veins and lymphatics. It is only when the action of the gas is superficial and localized that the condition clears up rapidly.

Sensitivity.—All persons are sensitive to the action of mustard gas, and so far as is known all who have not previously been exposed to its effects possess approximately the same degree of sensitivity irrespective of race or colour.

Acquired hypersensitivity.—In contrast to normal sensitivity it has been found that persons who have suffered injury as a result of exposure to mustard gas may in some cases become hypersensitive to its effects. The condition may be induced by either the liquid or the vapour of mustard gas. It is not possible to say with certainty whether a similar condition may be induced by other types of blister gas.

10. Methods of dispersion of mustard gas.

Of the known chemical warfare agents, mustard gas is one of the most versatile in that it will fulfil three objects of gas attacks, namely : (*a*) to inflict casualties, (*b*) to harass troops, and (*c*) to render ground dangerous to occupy. Owing to its delayed action, however, it will not immediately arrest the advance of an attacking force.

The method of dispersion will vary with the tactical object the enemy has in view, but his choice will be extensive. Against columns of troops on the march, or against large concentrations of troops or transport, air spray will be an effective means of inflicting casualties ; whereas in position warfare, against personnel manning trenches or other fixed positions, mustard gas shell or other projectiles will probably be chosen, especially if surprise can be coupled with intensity of fire.

In the case of buildings, encampments and industrial centres generally, mustard gas air bombs will be more effective, especially if supplemented by high explosive or incendiary bombs.

For harassing purposes almost any method of mustard gas contamination may be utilized ; similarly, a heavy contamination of an area by means of air bombs, gas containers or shell fire may be successful in rendering such a position untenable or dangerous to occupy. With this object, also, gas " booby traps " may be set by a retreating enemy, while living quarters, dug-outs, trenches, etc., may be heavily contaminated by him before withdrawal by the use of hand contamination bombs, contact or delay-action mines, containers fired by time fuses, etc.

Of all methods, however, dispersion by aircraft spray or bombing is the one that is most likely to be favoured for general use, as by its means an enemy can command a rapid choice of objective, speedy action and intensity of contamination. Moreover, the utilization of aircraft for this purpose will lessen the difficulty of transport to front areas, as supplies of mustard gas in bulk could be retained in back areas.

11. Dangers to be anticipated from liquid mustard gas.

An air spray attack on troops in the open is one of the most likely sources of danger, since the range and speed of aeroplanes enable them to produce that surprise which is so essential for the effective use of gas. Moreover, a spray attack may be launched by an enemy at such a distance from the target that the attacker can be neither seen nor heard, and the first intimation of attack may well be the arrival of the liquid spray on the troops forming the target, or even the early stages of vesication.

Under these conditions adequate defence is obviously difficult, and casualties

are certain to occur even with well-trained troops. If the attack is totally unexpected a potential danger will be liquid contamination of the eyes—an occurrence that will put a man out of action for the rest of the campaign. Even if the possibility of such an attack be foreseen and personnel have their eyes protected by goggles or respirators, it will be difficult to avoid some degree of contamination of the person, and in the absence of special protective clothing mustard gas burns are inevitable unless the normal clothing be removed within a matter of minutes. The appearance of these burns will be delayed, and they will be slight or severe, localized or extensive, according to the number and size of the drops of the liquid on the clothing.

Gross contamination of the body, with correspondingly severe results, may also occur from splashes of liquid mustard gas due to proximity to a bursting mustard gas shell or bomb.

More insidious dangers from the liquid arise from the handling of material whose contamination is not suspected or from mustard gas in the frozen state.

12. Lesions produced by liquid mustard gas.

(1) *Eye burns due to the liquid.*

A splash of liquid mustard gas in the eye is painless, but severe symptoms develop in an hour or two. The dangerous effects of liquid are the same as those of vapour in high concentration and will be further considered in Section 15.

(2) *Skin burns due to the liquid.*

(*a*) *On bare skin.*—It must again be emphasized that although liquid mustard gas is a direct irritant to the skin, the reaction is not immediate. Its high lipoid solubility enables it to penetrate tissues rapidly, especially when the skin is hot ; but hours may elapse before effects appear.

The initial sign in a typical mustard gas burn is erythema at the site of contact ; the capillaries are engorged, and œdema supervenes. The erythema deepens, and in severe cases may even assume a livid hue : a pale, parchment-like area makes its appearance in the centre of this erythematous zone, and a vesicle, tensely filled with serum, gradually forms (Appendix IV, Plate I). This vesicle is the result of an inflammatory exudation of fluid which may continue for several days, according to the depth of penetration of the liquid gas ; the exudate, however, contains no actual mustard gas.

If the liquid contamination of the skin be widespread, as in a smear or splash, the erythema is followed by the appearance of numerous small vesicles which gradually coalesce to form large blebs, the underlying area being raw and œdematous ; such blisters may continue to develop in crops for several days.

Apart from itching, especially of warm moist regions such as the scrotum, there is little or no irritation during the period of vesicle development ; the vesicles themselves are painless. Owing to the devitalization and impaired blood supply of the affected tissues, secondary sepsis is very prone to develop. Healing is slow ; the resulting scar is soft and pliable and the surrounding skin often assumes a coppery pigmentation.

Primary (neurogenic) shock is absent. If the area of blistering is considerable, some degree of hæmoconcentration may occur, but the effects of mustard gas are less pronounced in this respect than those of lewisite (p. 27).

(*b*) *On clothed skin.*—Drops of liquid mustard gas on clothed areas of the body act by virtue of the high concentration of vapour evolved, the warmth of the underlying skin naturally assisting the process. A gross contamination of the clothing, on the other hand, such as may be produced by splashes or by accidental spilling, may result in actual contact of the liquid with the skin, when the action of the vapour would be superadded to that of the liquid.

All ordinary clothing is pervious to liquid mustard gas ; but it is obvious that penetration will be much more rapid in the case of the single thin cotton garment of tropical and sub-tropical countries than with the multiple layers of woollen clothing worn in temperate climates. If the garments be damp or wet the rate of penetration of small drops of liquid mustard gas is somewhat hastened.

13. Protection against liquid mustard gas.

In view of the penetrative properties of liquid mustard gas it is necessary, for bodily protection, to adopt materials which are, as far as possible, impervious to the liquid. The choice of such materials is narrow, nor do they conduce to bodily comfort ; hence they must take the form of additional equipment for use when necessity arises.

Thus the eyes may be safeguarded by the use of special eye shields or of a suitable respirator, while the skin can be protected by special garments, gloves and footwear.

It may be necessary to remark here that no wearing material as yet discovered is completely impervious to mustard gas ; penetration by the liquid, or by the vapour evolved therefrom, is only a question of time if no steps be taken to neutralize or remove the contaminant. *Hence the term " mustard proof " in connection with protective garments is only a relative one, and merely means that protective clothing is more resistant than ordinary garments.*

Once they become contaminated these protective materials are a potential danger, both to the wearer and to his neighbours. Special care, therefore, should be taken not to spread contamination by wearing them in enclosed spaces such as dug-outs, living-rooms, or public conveyances, nor to continue wearing them after the necessity for doing so has disappeared.

The cape, anti-gas, made of light oilskin fabric, gives useful protection against liquid blister gas, especially if the liquid is swabbed off at the first opportunity. Capes for stretcher-bearers have press-studs enabling them to clip the point of the cape between their legs.

Suits, anti-gas, light and heavy, are issued to special personnel and give protection for shorter and longer periods respectively.

14. Dangers to be anticipated from mustard gas vapour.

It has already been pointed out that this vapour can be harmful in concentrations not readily noticed by the sense of smell ; that the sense of smell tends to become dulled ; and that the odour may be masked by smokes or high explosives.

The cumulative effect of repeated small doses of the vapour is another insidious danger. A lack of gas discipline, a natural impatience engendered by the prolonged wearing of a respirator, or dire necessity may influence men, exposed to mustard gas vapour, to remove their masks occasionally.

To produce systemic poisoning the vapour concentration must be high. But when it is realized that half an hour's exposure to a concentration of one part of mustard gas vapour in one million parts of air is sufficient to incapacitate an unprotected man for about two weeks through conjunctivitis, it will be seen that a very minute quantity of liquid mustard gas in a closed room may easily produce casualties.

The degree of severity of mustard gas vapour casualties varies with the concentration and the period of exposure. The least severe case may show little conjunctivitis, with almost no erythema of the skin and only a slight hoarseness of the voice ; the most serious, on the other hand, may present a

picture of the most profound illness, usually with widespread skin burns, shock, severe eye effects and damage to the respiratory tract and to the bone-marrow.

A moderately severe case of exposure to the vapour when quite unprotected will present a typical appearance in 24 hours, with eye symptoms predominating; general reddening of the skin occurs, most marked in the genital region where the excoriation of the skin may cause distressing irritation, while, at about the same time, the respiratory system begins to show signs of involvement by a partial loss of voice and by a troublesome cough.

In the Great War fatal cases were almost unknown within the first 24 hours. Death occurred at any date from the second or third day to the third or fourth week, with a maximum about the third or fourth day, from secondary broncho-pneumonia and systemic poisoning.

The main features of mustard gas vapour casualties may be summarized as follows:—

(*a*) An insidious onset, probably within 2–24 hours. But symptoms have developed several days after the clothes were exposed to vapour.

(*b*) Injury to the eyes, varying from simple conjunctivitis of a temporary nature to a severe keratitis and grave complications.

(*c*) Laryngitis, involvement of trachea and bronchi, and possibly necrosis of the mucous membrane leading to severe bronchitis or broncho-pneumonia.

(*d*) Early nausea or persistent vomiting, accompanied by epigastric pain.

(*e*) Erythema of the skin—early in the case of exposed areas or of hot, moist surfaces—which may proceed to vesication or excoriation, and may be followed by secondary septic infection.

(*f*) Slow healing of the blistered, devitalized areas and pigmentation of the ensuing scar.

(*g*) Systemic effects, particularly upon the bone-marrow and lymphoid tissues, in the most severe cases.

15. Lesions produced by mustard gas vapour.

(1) *Action on the eyes.* (*See* Appendix IV, Plates VI and VII.)

Mild Lesions.—No discomfort is felt till some hours after exposure, when the lid margins become red and swollen, and the conjunctivæ hyperæmic, especially in the interpalpebral aperture (Plate VIA). The cornea shows only slight epithelial œdema. There is lacrimation and a little photophobia, but the condition clears up in 7–10 days.

Moderate Lesions.—The onset is similar, but pain, photophobia, and lacrimation are greater. Swelling of the lids prevents voluntary opening of the eyes for several days. The discharge may be clear and watery, but if neglected it becomes purulent and thick crusts on the lashes further prevent separation of the lids. Retention of discharges causes acute pain and favours complications. Cornea which has been directly exposed is grey and lustreless, but in uncomplicated cases the corneal epithelium heals quickly and the stage of fluorescine staining is not always seen. The eyes can be opened in 4–5 days; there is slight œdema of the cornea for 10 days or more; lacrimation and photophobia gradually diminish, and recovery takes about 3–4 weeks.

Severe Lesions.—Severe symptoms appear in about 5 hours, with intense burning pain, blepharospasm and rapid swelling of the lids, so that within 8 hours the eyes are closed. The secretion is watery unless secondarily infected, when it is purulent and very profuse. When the swelling subsides sufficiently for the eyes to open there is intense photophobia and the sight is misty. The cornea is hazy and œdematous and there is chemosis of the ocular conjunctiva which may protrude between the lids. Conjunctiva which has been directly

exposed in the palpebral aperture may be pale and bloodless, while the surrounding portions are hyperæmic (Plate VIB). There is often mild iritis and in very severe cases posterior synechiæ may form unless mydriatics are used. The œdema of the cornea extends through all the layers and may take 3 months to subside. Convalescence is slow and hindered by persistence of photophobia, lacrimation and blepharospasm for several months.

In these very severe cases vascularization of the cornea and secondary œdema set in after subsidence of the primary œdema or are continuous with it. Such changes, occurring where there has been prolonged exposure to vapour or direct contamination by liquid mustard, are serious, for they may continue to recur for months or even years, accompanied by some degree of impairment of sight, which increases with each attack. Corneal ulceration and vascularization may return more than 15 years after apparent cure (Plate VII) in cases where the scar is unstable because of deposition of cholesterin.

Complications.—Any stage may be complicated by bacterial infection, and the consequences tend to be severe because the tissues are devitalized by the chemical. Purulent conjunctivitis, corneal ulceration, sometimes with hypopyon, blepharitis, styes, Meibomian abscesses, and cysts may all supervene. Perforation of the globe and loss of the eye is a rare and late complication and symblepharon does not occur.

(2) *Action on the respiratory tract.* (*See* Appendix IV, Plates IV and V.)

The toxic effects of mustard gas vapour on the respiratory tract are shown by an early rhinitis (almost simultaneous with the onset of the conjunctivitis), accompanied by sneezing and the discharge of a profuse watery secretion soon to become mucopurulent.

The larynx is usually affected early, and hoarseness and aphonia are frequent. The laryngitis may be mild if exposure has been limited to a low concentration, but œdema and even sloughing of the vocal cords may follow exposure to a high concentration.

In a severe case, the laryngeal inflammation tends to be reproduced in the trachea and bronchi, when the dry irritating cough, originally couplained of at the onset of the laryngitis, is replaced by a loose cough accompanied by profuse mucopurulent expectoration and pain behind the sternum. A rising temperature and pulse indicate the onset of a severe bronchitis which may be complicated by sloughing of the inflamed tracheal mucous membrane; secondary infection of the latter soon leads to the development of a bronchopneumonia with cyanosis. Rarely, abscess of the lung, bronchiectasis, or even gangrene of the lung may occur—not caused directly by mustard gas vapour, but by the secondary bacterial invasion which follows.

In the great majority of cases, however, the lesion is confined to a bronchitis which clears up in a month or six weeks, leaving no after-effects.

(3) *Action on the skin.* (*See* Appendix IV, Plates II and III.)

As in the case of liquid mustard gas, the vapour owes its penetrative powers to its ready solubility in the lipoid tissues of the skin. The degree of skin burning is accentuated if the exposed skin area be a highly sensitive and tender region such as the scrotum, or if it be a surface which is subjected to constant friction, as is the case in the neck, the wrist and the ankles.

If the exposed skin surface be bare, the attack of the vapour will be direct, and the result more rapid than if the skin be clothed. This temporary protection of clothed areas is due to the fact that ordinary porous clothing material absorbs the vapour and retards its access to the skin; but if such clothing be worn beyond the period of actual exposure, or if the exposure be prolonged, the vapour retained by the clothing will increase the severity of the burns.

This temporary protection varies in duration according to the nature, texture, thickness and degree of humidity of the clothing. Thus, a thin open-work cotton garment in close apposition with the body surface will not greatly retard the access of the vapour to the skin, whereas thick close-woven material, such as serge and woollen clothing generally, will definitely do so, and may even save the area from burns provided that it be discarded on leaving the contaminated area. Again, damp and sweaty clothing will absorb more mustard gas vapour than the same clothing when clean and dry.

After a latent period, usually between 2 and 24 hours, an erythematous blush appears, and this may gradually deepen in intensity until the skin looks scorched.

This redness is not unlike the eruption of scarlet fever, and is usually accompanied by only a slight degree of irritation. The erythema is most marked on the skin areas which are hot and moist ; dense tissues like the scalp, the palm of the hand or the skin of the heel usually escape unless the concentration of the vapour be high and localized to that area, as, for example, from drops of liquid mustard gas on a cloth cap.

The affected area soon begins to show superficial blistering in the form of small vesicles which rapidly coalesce to produce large blisters full of a clear yellow serum ; on evacuating this fluid and removing the overlying epithelium, a raw, red, weeping surface is exposed. As a rule vesication is complete by the second day, but blisters may appear in crops for several days following exposure, even though all contaminated clothing be discarded at an early stage. Systemic disturbance is not pronounced, unless the burns are extensive and severe ; interference with sleep, however, may be caused by the distressing itching which may accompany the developing burns.

Very mild cases may show simply erythema, followed later by pigmentation with scurfy desquamation, the " blister " stage being absent.

In severe cases the erythema may deepen to a dusky, almost violet tint, with œdema of the skin, and blisters appear over the dark background overlying a deep red or hæmorrhagic base. Such blisters progress slowly, and are very prone to sepsis owing to the serious devitalization of the tissues ; ulceration is liable to spread beyond the limits of the blister, and healing is very slow. If sepsis occurs it adds to the severity and duration of all lesions ; the necrosed tissues form an excellent medium for pathogenic organisms, and death may result if extensive or deep burns are thus affected.

The healing of an uncomplicated vapour burn is more rapid than one due to liquid mustard gas, but all mustard gas burns take a long time to heal. They leave a brownish or coppery pigmentation of the epithelial layers in the areas affected by the erythema ; but this usually disappears with the normal desquamation of the superficial layers of the skin.

As a rule, serious after-effects are absent, and the scars resulting from vapour burns are shallow ; but a chronic eczematous condition or a generalized furunculosis may, rarely, follow such burns and prove obstinate to treatment.

(4) *Other effects of mustard gas vapour.*

(*a*) *Alimentary tract.*—It has already been mentioned that an early nausea, or even vomiting, accompanied by epigastric pain, often occurs in poisoning by mustard gas vapour. Although it may prove obstinate during the first day, it rarely persists for more than 48 hours ; similarly, the accompanying epigastric pain is of short duration, and the intestines are not usually affected. There are no lasting after-effects, but a functional condition of persistent nausea or vomiting has been observed occasionally.

(*b*) *Urinary tract.*—Traces of albumin have been found in the urine of early

fatal cases, most probably due to congestion from circulatory weakness and not to the action of the gas on the kidneys. It is only in the late stages of fatal cases, and particularly those with widespread septic burns, that renal complications have been noticed, such as an acute hæmorrhagic nephritis. Pain on micturition, however, and even retention of urine may result from a local œdema and vesication of the penis.

(*c*) *Blood.*—Application of mustard gas (liquid or vapour) to the skin of man causes a transient leucocytosis, followed by leucopenia and even agranulocytosis; the bone marrow may be severely damaged. An increase in the plasma sedimentation rate is often observed.

16. Protection against mustard gas vapour.

The general remarks on protection against liquid mustard gas apply equally well in the case of mustard gas vapour, except that most goggles afford no protection against the latter, and a respirator must be worn if the eyes are to be safeguarded.

A well-fitting respirator will effectively protect the eyes, the respiratory tract and the skin of the face, but, in the absence of special clothing or protective equipment, the rest of the body is open to attack.

It has already been mentioned that ordinary porous clothing, especially thick, dry woollen garments, affords some measure of safety during a short period of exposure to the vapour. The objection to an oilskin cape or suit is that, owing to its stiff and impermeable character, the wearer's movements give it a bellows-like action when bending or doing manual work. Contaminated air is thus sucked inside the garment through any available channel, and with vigorous work it is only a matter of time before the atmosphere inside the suit becomes as dangerous as that outside. Where circumstances permit the garments should be freely ventilated at frequent intervals in an atmosphere uncontaminated by the gas.

Protection against mustard gas vapour is, therefore, even more difficult to achieve than protection against the liquid.

17. Preventive treatment after contamination by mustard gas.

(1) *Preventive treatment for the eyes.*

If protective eye-shields are properly worn droplets of mustard gas should not gain access to the eyes. If, however, they do so, the resultant injury can be lessened if the eye is immediately flushed out thoroughly with water—e.g. from a water-bottle. This treatment must take precedence over other cleansing measures, such as the use of anti-gas ointment. If the eye cannot be flushed out with water within five minutes no benefit can be expected. The essential point of this treatment is that it must be *immediate.*

In view of the very serious results that may ensue from the penetration of liquid mustard gas into the eye, and the obvious difficulty of flushing out the eye immediately in the field, too much stress cannot be laid on the importance of wearing eyeshields whenever there is any risk of encountering aircraft spray.

If inflammation of the eyes is caused by prolonged exposure to the vapour of mustard gas emanating from the ground or other contaminated objects, such irrigation will not remove the mustard gas, since the poison has already been slowly absorbed and the damage done. Moreover, irrigation of the eyes takes a great deal of time and delays such essential measures as the removal of decontaminated clothing and cleansing of the skin. *The eyes should therefore not be irrigated as a routine, but only when there is a suspicion that a drop of liquid mustard gas has actually fallen into the eye within five minutes.*

(2) *Preventive treatment for the skin.*

To save the skin from damage it is necessary (i) to remove any clothing contaminated by liquid or vapour * ; and (ii) to free the skin from contaminating liquid.

Prompt local cleansing will suffice for a small localized liquid contamination of the bare hand, and a change of clothing is all that is necessary after exposure to a low concentration of the vapour. Each case will have to be considered on its merits. But, whatever the type or extent of the contamination, **speed is the essence of all preventive treatment.** When the skin is hot as a result of exercise, and in hot or tropical countries, the need for prompt action is especially great.

After exposure to vapour in the atmosphere, or after the outer clothing has been sprayed or come into contact with fluid, the most important measure is removal of clothes ; and in deciding how much clothing must be removed it should be borne in mind that a single drop of mustard gas on a garment can cause widespread vapour burns. As an additional precaution immediate washing of the skin with soap and water is recommended, though not essential.

For contamination of the skin by liquid there is a choice of methods. *The best to adopt is the one most readily available.*

(*a*) If the liquid is visible it should be removed by *swabbing*, e.g. with cotton-waste.

(*b*) *Ointment, anti-gas*, should then be applied in small quantities to the affected areas and rubbed in vigorously for not less than half a minute. The ointment chemically neutralizes the mustard gas, and if it is applied within five minutes of contamination blisters should be prevented. It may also be useful later if there is still fluid on the skin ; but where erythema has actually developed it is useless and indeed harmful. Nor must it get into the eyes.

The original ointment No. 1, made from " supertropical " bleaching powder and white vaseline (equal parts by weight), was irritant and is now obsolete. Ointment No. 2, composed of Chloramine-T in a vanishing-cream base, is also slightly irritant ; so it should not be used repeatedly on tender skin or under clothing, and any excess is best removed. Ointments No. 3 and 3A are non-irritant. Both No. 2 and No. 3 may be used protectively on the hands in lieu of gloves : they are rubbed in for half a minute, leaving a visible film, and swabbed off after work. Heat and dryness may cause deterioration of ointments, and they should be inspected frequently to make sure that they smell of chlorine when rubbed on the hand.

(*c*) Aqueous *bleach paste* may be used instead of ointment, and is preferable when large areas have to be covered. It can be painted on the skin with a brush, but must be washed off one minute later. Like the ointments, it should not be used after erythema has developed, and it is dangerous to the eyes.

The aqueous bleach paste consists of " supertropical " bleaching powder mixed to a creamy consistency with water—roughly, one part of the powder to one or two parts of water by volume, and it retains its effectiveness in temperate climates for several weeks if it be stored in enamelled containers with well-fitting lids. Bleaching powder is ordinary chloride of lime, while supertropical bleach is ordinary bleaching powder stabilized by the addition of quick-lime and fulfilling certain conditions of stability and chlorine content.

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* Should a Thomas's splint have to be applied special care will have to be taken not to restrict the circulation through the foot if it is necessary to remove the boot owing to its contamination by liquid mustard gas.

Ordinary bleaching powder is more irritating to the skin than the super-tropical variety, but in the absence of the latter it is quite suitable for preventive treatment when made into a paste with water, provided prolonged storage is not contemplated.

(*d*) *Repeated swabbing of the contaminated area with petrol, methylated spirit, kerosene, carbon tetrachloride* (*Pyrene liquid*) *or other solvent of liquid mustard gas.* It is important to remember that these solvents do not destroy the gas, but merely dissolve it ; hence the swabbing must be confined strictly to the contaminated area. Oilskin or rubber gloves must be used if they are available ; otherwise, the swab should be only partly moistened with the solvent, and it should be held between finger and thumb by the dry portion or preferably in forceps. The wet portion is applied to the contaminated skin so as to soak up the liquid contamination, care being taken that none of the solvent runs over the skin of either the subject or operator ; the contaminated swab is then discarded and the process is repeated with fresh swabs for several minutes, or as long as the characteristic odour of the gas persists on the skin. Thorough washing with soap and water, if available, will complete the treatment ; the contaminated swabs must, of course, be destroyed by burning or burying and the gloves and forceps decontaminated.

One disadvantage of this method in the hands of unskilled persons is that the solvent is apt to " run " on the skin and cause burns on areas comparatively far removed from the original site of contamination ; a further disadvantage is the liability of the operating fingers to become contaminated in the absence of gloves. Employed with care and intelligence, however, the method should be valuable in emergencies at aerodromes, among mechanized transport, and in other situations where mustard gas solvents are at hand. It is also a good means of removing liquid mustard gas from burns already showing erythema.

(*e*) *Washing with soap and water*, using hard soap and frequent changes of water. This process does not destroy the mustard gas, but merely removes it in the lather ; the washing must, therefore, be confined to the contaminated area, and the operator's hands should be safeguarded, if possible, by suitable gloves.

If the liquid contamination be small, localized and of known situation, this is an effective method of removing it, if carried out promptly. Vesication of the skin is usually prevented if the treatment is not delayed beyond five minutes, though an erythema may follow.

With a gross contamination, or when the drops of liquid mustard gas are numerous, the results of washing with soap and water are unfavourable, as it is difficult to avoid spreading the contaminant in the soapy lather to surrounding areas. In these circumstances bleach treatment is best.

Where erythema has already begun, the use of a solvent is recommended ; but if cleansing is delayed, thorough washing should still be carried out at the first available opportunity in the hope of mitigating the burning.

18. Curative treatment of mustard gas casualties.

The first essential is to prevent further injury from contaminated clothing : it may be necessary, therefore, to strip and wash the patient.

The second essential is to relieve immediate symptoms and to prevent the septic infection of possible lesions.

(1) *Treatment of the eyes.*

The value of initial flushing out of the eyes with water or saline has already been considered. Once conjunctivitis is established, irrigation will have no direct effect on the development of the mustard gas injury, and therefore no

time should be spent on it. The profuse flow of tears will do all that irrigation could do.

By the time the casualty is seen there is sure to be some degree of conjunctivitis, accompanied by swelling of the eyelids, and photophobia. The lids should if possible be opened to let the patient see for himself that his sight is not lost: it should be explained that he must go through a period of increasing discomfort before improvement begins and that he will recover.

At the earliest opportunity a drop of mydriatic should be put into the eye: the preparation of mixed hyoscyamus alkaloids known as "atropine alkaloids" is suitable for this purpose; or 1 per cent. atropine solution where supplies are plentiful. After the mydriatic has acted it may be useful to instil a few drops of a mild antiseptic. Strong antiseptics must on no account be used, and Albucid Soluble (2·5 per cent. solution) is usually recommended. It is one of the sulphonamide derivatives which forms a neutral solution with water and is supplied in 5 c.cm. ampoules of 30 per cent. solution. The contents of one ampoule diluted to 60 c.cm. with normal saline or water give a 2·5 per cent. solution. It should be stored in the dark.

Never irrigate unless the discharge is very copious. Experiment has shown that repeated (e.g. two-hourly) irrigation increases the severity of the lesion. If the discharge is excessive, wash it out with normal saline night and morning. A mild antiseptic may be instilled occasionally; but the essentials of treatment are to keep the eyelids very clean and the pupils dilated.

If blepharospasm is sufficient to close the eyelids completely, the case should be seen by an ophthalmic surgeon as soon as possible for two reasons:—

(i) So that mild cases may be diagnosed as such and not be transferred as severe casualties. This will lessen the development of "functional" cases and therefore reduce the drain on the fighting troops.

(ii) So that severe cases may be diagnosed as such and appropriate action taken.

Cocaine should *not* be used to relieve pain since it has a deleterious effect upon the corneal epithelium which is already damaged by mustard gas.

The eyes should not be bandaged nor closely covered, since free drainage of the discharge is essential, but a forehead shade is permissible. Such a shade can be easily improvised from brown paper or other material.

Never instil liquid paraffin (or other oily drops) during the first few hours, but after that it may be used to prevent the lids from becoming adherent.

Many of the casualties caused by exposure to vapour are likely to be mild. None the less such casualties may at first be unable to see owing to the swelling of the eyelids and pain, and the early photophobia, which has a genuine cause, may be succeeded by a functional photophobia which unduly protracts convalescence. If this is to be avoided and the mild cases returned to full duty without needless delay it is essential to remove the eye-shade as soon as possible and to make the casualty confident that he need fear no permanent injury to the eye nor impairment of vision. General tonic treatment, and suitable exercise that will keep the casualty from brooding on his condition, will hasten his recovery.

Mustard gas has, as the experience of the last war showed, a very high casualty-producing value, and undue wastage of the fighting troops can only be prevented by taking trouble over the treatment of the milder casualties so as to minimize the time spent either in the hospital or the convalescent camp.

It is, of course, the casualties who have been exposed for a considerable time to a heavy concentration of vapour, and in particular those cases in which

a drop of liquid mustard gas has entered the eye, that offer the most difficult problem, and it is those cases which demand the most careful attention of the ophthalmic specialist from the start.

(2) *Treatment of the respiratory tract.*

The early *rhinitis* is usually overshadowed by the condition of the eyes: Should there be pain and distressing discharge, it may be treated with copious warm douches of sodium bicarbonate in 5 per cent. solution several times daily. In the rare cases where a persistent mucopurulent discharge, associated with ulceration and occasionally with epistaxis, is long continued, an astringent lotion containing zinc sulphate gr. 1 with boric lotion oz. 1 will be found helpful.

Laryngitis and tracheitis.—The laryngeal irritation is best dealt with by topical treatment such as laryngeal spraying with medicinal liquid paraffin, or by the inhalation of steam from a pint of boiling water containing a teaspoonful of a mixture of menthol gr. 10 in tinct. benzoini co. oz 1. The harsh, dry cough may also be eased by breathing through a perforated metal mask (Burney Yeo) containing a pad of gauze moistened hourly with drops of some volatile antiseptic mixture such as :—

Menthol	gr. 20
Chloroform	min. 60
Creosote	„ 60
Ol. eucalypti	„ 20
Liq. iodi. mitis	„ 30
Sp. vini rect.	to one ounce.

The menthol in such a solution will help to alleviate the paroxysmal coughing which may otherwise result if such an anæsthetic as simple ether has to be used to allow operative treatment of an accompanying wound.

Broncho-pneumonia.—As the majority of deaths from mustard gas in the Great War were due to secondary infections of the respiratory tract, treatment should be directed, from the outset, towards combating bacterial invasion of the bronchi. All cases of mustard gas poisoning in which the respiratory tract is involved must be kept apart from other patients suffering from infective pulmonary disorders ; they should, if possible, be segregated in special wards, and the onset of broncho-pneumonia in one of them should entail his isolation.

In the various stages of the broncho-pneumonia, treatment follows the recognized procedure, including the use of sulphonamides. The prophylactic venesection advocated for phosgene cases, which is of value in the treatment of venous congestion, seldom has any place in the treatment of mustard gas cases, though occasionally it may be indicated at a later stage to relieve the right heart of embarrassment and cyanosis induced by a diffuse broncho-pneumonia. The same may be said of oxygen therapy, which, although essential in the pulmonary œdema caused by phosgene, is indicated only occasionally and at a late stage in mustard gas poisoning when cyanosis is established as the result of grave and widespread pulmonary damage.

As is evident from Plates V and VIII, the pulmonary lesions of mustard gas differ widely from those of choking gases such as chlorine and phosgene which rapidly produce œdema. When death from mustard gas poisoning takes place early, the lungs, instead of being saturated with fluid, are dry ; and the œdema seen later between patches of consolidated lung is secondary to the broncho-pneumonia.

(3) *Treatment of the skin.*

Sepsis is the most potent factor in delaying the healing of burns. When it is remembered that troops in the field are unavoidably dirty, and that mustard

gas devitalizes the skin, it is obvious that early preventive treatment is of paramount importance.

As a preliminary to all local treatment it is essential to cleanse the skin as thoroughly as its damaged condition permits, and to clip short all hair, if any, on the affected area. *It may be useful to repeat here that the application of bleach in any form to a skin which is already showing signs of damage will aggravate the ensuing burn.* It must also be noted that skin surfaces damaged by mustard gas are exceedingly susceptible to trauma, and that even the continued pressure of an ill-fitting bandage may extend the damage.

As treatment will vary according to the nature and degree of the burns, it will be best to consider these in detail :—

(i) *Erythema* : Mild cases which do not proceed beyond an erythema heal spontaneously, with possibly some desquamation and pigmentation. They may be compared with sunburns in severity and discomfort, and clear up just as readily. After washing with soap and water or with Dettol (e.g. 4 per cent. solution) a mildly antiseptic dusting powder may be applied (e.g. zinc oxide, starch, boric acid and chalk in equal parts), or calamine, or Limzol (equal parts of lime water, zinc oxide and olive oil). Where sepsis is anticipated the area may be treated with bacteriostatic agents, such as one of the sulphonamides. In the scrotal region irritation can be relieved with 1 per cent. acriflavine or with calamine lotion containing 1 per cent. carbolic acid. A warm bath is another useful means of relieving irritation.

(ii) *Vesication* : Small blisters can be treated in the same way as erythema and may disappear without breaking. Isolated large blisters should be evacuated under aseptic conditions with a syringe or opened with a scalpel or needle, gentle pressure being applied, if necessary, upon the walls of the blister with a sterile swab to ensure complete evacuation ; the intact epithelium should then be allowed to collapse and protect the raw, sensitive surface underneath. This evacuation of fluid from blisters may have to be repeated owing to the continued oozing of serum from the raw area. The further treatment of these isolated vesicles consists in the application of dry sterile dressings or saline packs.

When a considerable area is blistered, not only the lesion but also all the surrounding skin which may ultimately show damage should be cleansed with a non-irritating antiseptic such as Dettol solution, hairy parts being shaved or clipped. The blisters, unless already broken, should be punctured when they become tense. Since the exudation in such cases is very copious it is no good using a tanning treatment at the start, as any coagulum formed will be floated off by the continued exudation, and it will be impossible to secure a firm protective scab. The long duration of the exudation depends in part on the fact that the action of the poison continues for a considerable time after it has been absorbed by the skin, and the damage is therefore progressive. Measures which result in a reduction of the local œdema are therefore desirable.

Treatment at this stage with amyl salicylate helps to dry up the exudation, reduces the surrounding œdema and erythema, and diminishes the pain, irritation and discomfort. A pad of gauze soaked in amyl salicylate, and wrung out, should be applied and covered with Cellophane and a thin layer of cotton wool which should be lightly bandaged. The pad should be changed either once or twice a day.

When the exudation has subsided and the wound is dry, at the end of several days, if progress is unsatisfactory, amyl salicylate may be discontinued, and a dressing of crude cod liver oil (of *neutral* reaction) may be substituted after removing any *loose* fragments of blister skin.

A minor objection to amyl salicylate is its clinging sweet smell. But it is also somewhat irritant to the eyes and mucous membranes and should not be applied to the face or genitalia.

In the absence of amyl salicylate, a pad of sterile gauze wrung out in sterile normal saline should be applied to the burn in the early stages, being changed as often as it becomes soaked with exudate. As soon as the exudation has diminished sufficiently, the blister skin should be removed and the area treated with one of the remedies employed for thermal burns. Coagulants must not be used on the face or hands lest they should leave contracted scars. Crusts on the face may be removed with copper sulphate solution (copper sulphate gr. 4, zinc sulphate gr. 6, camphor water 1 oz. : dilute 1 part in 8 parts of water).

The healing of mustard gas burns is a slow process. In the case of the deeper burns a granulating area may be left, and treatment with a stimulating ointment, e.g. scarlet red ointment, may be helpful, care being taken not to damage the new granulation tissue. Preparations useful for lingering dermatitis and irritation are blue paint (brilliant green 1/1,000 and crystal violet 1/1,000 dissolved in equal parts of rectified spirit and water), sulphonamide ointments, or limzol (p. 21).

Severe burns in the neighbourhood of a joint may demand immobilization until healing is well advanced.

If the burn becomes septic, or there is deep necrosis of the skin, *mildly* antiseptic baths may be useful and soothing, warm hip baths of isotonic salt solution allaying the intense irritation of mustard gas burns of the genitalia. Hot fomentations are unsatisfactory. There is nothing to contraindicate the use of powdered sulphonamides on the wound.

The slow progress of mustard burns under treatment, compared with thermal burns, is attributable not only to lowering of the vitality of the tissues by the poison but also to persistence of an irritative process in the tissues even when necrosis has ceased. These factors explain the greater intensity of the inflammatory reaction, the longer delay in repair, and the special tendency to septic infection.

(4) *General treatment.*

Where nausea, vomiting or epigastric discomfort is present, the diet should be light and fluids may be given freely, especially milk and beef tea ; should these not be retained, the administration of gr. 10 to 20 of sodium bicarbonate may be of assistance, and the patient should be encouraged to drink water freely. As convalescence proceeds, and in all cases of uncomplicated body burns, a full diet is required, and this should be as varied as possible. Cases showing evidence of commencing fever, which may be a prelude to bronchopneumonia, should be suitably dieted.

Ascorbic acid has been recommended to combat systemic effects, but rapid saturation is essential (e.g. three doses of 1·0 gramme by mouth twelve-hourly).

Quoting from the " Official History of the War " :—" The management of the convalescent period provides the great test for the Medical Officer's ability, because he is required not only to treat the disease, but to restore morale, to cut short hospitalization, and to lift men out of the slough of self-analysis which so often follows gassing. The best results will therefore be obtained by placing gassed cases in selected hospitals under the care of Medical Officers with special aptitude for this.

" As soon as a man is convalescent and free from the danger of septic complications, he should be discharged from hospital to a convalescent centre, where a well-ordered routine of exercise, employment, amusement and rest will quickly restore him to a state of physical and mental fitness."

19. Functional after-effects.

In the Great War functional disorders led to wastage and invalidism which, to some extent, could have been avoided if personnel generally, and medical

officers in particular, had been more conversant with the limitations as well a the potentialities of chemical warfare substances.

Functional disorders fall in the main into two classes. In the first, exposure to gas—often to a minimal and barely a toxic concentration—may prove the final factor in upsetting a nervous system already breaking down as the result of physical or mental strain. In such circumstances, and especially when combined with ignorance, it may produce an " anxiety state."

In the second class a local, but real, organic lesion from mustard gas causes irritant reflexes, such as coughing or photophobia, and these are perpetuated by introspection, almost in a form of conversion hysteria, long after their organic cause has been cured. Lack of appreciation of this possibility by medical officers will cause much delay in returning men to duty.

Functional photophobia and aphonia are responsible for the great majority of cases. This is not surprising when it is realized that the initial trauma affects a highly organized special sense, and that fear of blindness or dumbness resulting from the injury may very well prolong the symptoms. Ill-advised and unnecessary treatment, however, is also a probable factor in many cases, as, for example, the continued retention of eye-shades long after the necessity for them has passed and the actual lesions have totally disappeared.

Persistent aphonia, often accompanied by a useless, harsh cough, is another striking evidence of auto-suggestion arising from the initial laryngeal irritation. The characteristic cough is either dry, or accompanied by watery sputum mainly of salivary origin. It is usually much worse at night, and is of a ringing harsh quality. If the medical officer realizes the nature of the condition and gives the patient confidence in his early recovery, this functional aphonia yields very rapidly to treatment by suggestion and breathing exercises.

Of all after-effects, functional or organic, those which seem to affect the heart present the greatest difficulty in assessment. Effort syndrome (formerly known as disordered action of the heart) with its shortness of breath and tachycardia following exercise, arises from so many diverse causes that gassing, in the Great War, was naturally regarded as one of them. It is clear, however, that under competent medical treatment, the incidence of effort syndrome in mustard gas casualties should be very low when serious complications, such as broncho-pneumonia, have been absent.

20. Invalidism after mustard gas poisoning.

Experience from the Great War showed that the chemical damage to the skin, to the respiratory passages, and to the outside of the eyes might cause prolonged devitalization of these tissues and a poor resistance to secondary bacterial infection, to say nothing of bone-marrow damage. There has been no subsequent evidence of the irritation leading to later malignant changes in any tissue. The trachea and bronchi in some cases showed a tendency to relapses of bronchitis, but there was no special proneness to pulmonary tuberculosis. In certain cases of severe injury to the eye, however, recovery was very slow, owing to repeated fresh ingrowths of blood vessels, followed by deposition of cholesterin, which in 15 years or less, led to corneal ulceration (delayed keratitis) after a varying symptomless period.

Invalidism in general was not prolonged, but it should be remembered that the casualties were very largely due to mustard gas vapour, and only rarely to direct splashes of the liquid. In all cases admitted to hospitals there was some degree of conjunctivitis and laryngitis as well as skin burns. The skin lesions from vapour healed quickly, usually in less than a month. Prolongation of invalidism was due rather to trouble in the respiratory passages and eyes, and to general debility. By following up the times of recovery in a large number of cases at convalescent depots in France, it was proved that at least 75 per

cent. of mustard gas casualties admitted to hospitals in the lines of communication, these being the severer cases evacuated from the army zone, could be returned to full duty in less than eight weeks. This involved an average stay in hospital of two to three weeks, during the last half of which time the casualties did not require to be in ward beds or to be specially attended by nursing orderlies.

The worst cases might remain in hospital for two months or even longer. Photophobia, either functional or associated with a tendency to recurrent keratitis, often lingered. Next as causes of invalidism came bronchitis and laryngitis, and lastly effort syndrome and neurasthenia or some general debility. But out of a group of nearly 800 severe cases detained in hospital beyond the ninth week, none died and ultimately only 0·5 per cent. were discharged as permanently unfit for service. The ultimate invalidism from mustard gas vapour was therefore very small. As in phosgene poisoning, it is probable that any persistent chest trouble was due to secondary bacterial infections rather than direct chemical action.

21. Contamination of equipment, water and food.

Although the medical officer is responsible only for the personal decontamination of individual contaminated *casualties* (i.e. persons who are *both* contaminated *and* sick or injured) who come under his care, he should be familiar with the general principles of decontamination of equipment and materials. He may be called upon to detect contamination of water and advise on the use of contaminated food (*see* Appendix III).

Satisfactory decontamination is essentially a skilled process, hence the personnel employed in carrying it out must be trained for the purpose.

Decontamination of stretchers.—If the ordinary pattern of stretcher is splashed with liquid mustard gas it is difficult to obtain complete decontamination, since the canvas is fixed to the woodwork by a large number of nails, which renders the removal and replacement of the canvas impracticable under field conditions. It is correspondingly important to prevent contamination.

Stretchers, when not actually being used, should, therefore, be kept under cover (e.g. indoors or under any improvised roof, tarpaulin, etc.) to prevent their being splashed with liquid blister gas. As an additional precaution the canvas should be covered with a stretcher cover. The standard article is made of special laminated paper and is expendable. This will prevent or delay the canvas of the stretcher being contaminated when carrying contaminated casualties. Alternatively, if a standard stretcher cover is not available the stretcher canvas may be protected with an improvised cover of resistant material such as a ground sheet or anti-gas cape. Such improvised covers can be decontaminated after use, if necessary, by one of the standard methods.

If, in spite of precautions, contamination does occur, the complete stretcher should be scrubbed with bleach paste and later washed thoroughly with water, after which it should be left to weather as long as possible. The handles may not be safe for contact with the bare hands for several days, but this difficulty can be overcome by wearing anti-gas gloves. Any danger from residual contamination on the canvas can be counteracted by the use of the covers described above.

If the canvas can be readily removed it can be decontaminated by immersing it in boiling water for 30 minutes.

Stretchers which have been exposed to mustard gas vapour only should be allowed to weather in the open air.

LEWISITE

22. Lewisite, or chlorovinyl-dichlorarsine, is an important chemical compound developed towards the end of the Great War. Knowledge of its action rests on laboratory experience rather than on a study of military casualties.

In the pure state, lewisite is a powerfully toxic substance, embodying the aggressive qualities of the lung irritant gases, the irritant characteristics of the tear and nasal irritant gases, and the vesicant properties of the blister gases. Under modern conditions of chemical warfare, however, its vesicant action would predominate ; hence its inclusion in this group.

The following remarks will deal mainly with salient points of difference between the effects of lewisite and mustard gas.

23. Physical and chemical properties of lewisite.

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 lacrimation. 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° or 57·9°, 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 with its aggressive action in abeyance.

Solubility.—Lewisite is sparingly soluble in water, though readily soluble in a variety of organic solvents and in oils and fats. It is, however, rapidly hydrolyzed by water, especially in the presence of alkalis, to the corresponding hydroxide or oxide. These compounds are only slightly lipoid- or water-soluble and have a very low vapour pressure ; they prove in practice much less vesicant than lewisite itself although extremely toxic. Water contaminated with lewisite is poisonous and must not be used for drinking or washing.

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. Although the products formed by its hydrolysis are highly toxic, lewisite can be completely destroyed by strong oxidizing agents, the arsenic being finally left in the inorganic form.

Penetration.—Like mustard gas, lewisite is penetrative as well as persistent. Materials contaminated by it are therefore dangerous to wear or to handle.

24. Toxicity of lewisite.

Lewisite is more rapid than mustard gas in its action, producing more discomfort on inhalation and more irritation when placed on the skin. It is accordingly less insidious. Its extreme pungency and penetrative odour ensure its 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 lead to vesication of the skin; in the liquid form it penetrates the tissues rapidly, and after a short latent period produces severe blistering. Liquid lewisite in the eye entails immediate incapacitation.

Hydrolysis of lewisite gives rise to products which are highly toxic, and their absorption after a sufficient dose has been placed on the bare skin may cause acute arsenical poisoning in which many organs of the body are affected.

Acquired hypersensitivity to lewisite has not yet been encountered.

25. Methods of dispersion of lewisite.

Lewisite may be used for the same tactical purposes as mustard gas, namely: (1) to inflict casualties, (2) to harass troops, and (3) to deny ground to an enemy. These objects can be attained by the methods already detailed for mustard gas, subject to the following limitations:—

(*a*) The easy detection of lewisite as compared with mustard gas makes it less effective than the latter against trained personnel, except when used as a spray from aircraft.

(*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.

26. Dangers to be anticipated from liquid lewisite.

Theoretically, the most effective method of using lewisite in warfare is by spraying the liquid from aircraft, as the element of surprise accompanying such an attack will largely counterbalance the handicap of its early detection.

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. Contamination of the eye is the most pressing danger; but, whereas with liquid mustard gas a latent period of nearly one hour elapses before incapacitation results, with liquid lewisite the local irritation is immediate, and of such severity that the eye is at once rendered useless.

Although very small drops of lewisite on the skin are less damaging than similar drops of mustard gas, an aggregation of such drops on the bare skin, as on the face and neck, would probably produce casualties within one hour owing to the increasing severity of the irritation. Moreover, the penetration of lewisite into the skin is so rapid that preventive measures, unless almost immediate, are of little value.

27. Lesions produced by liquid lewisite.

(1) *Eye burns due to liquid lewisite.*

However small the contamination, liquid lewisite in the eye elicits immediate pain, spasm and lacrimation. The vesicant acts, subjectively, like a powerful caustic, and the victim becomes an immediate casualty. Within 15 minutes there is intense chemosis, œdema of the eyelids, subconjunctival hæmorrhages, and œdema of the cornea. Within 3 to 4 hours the eyelids adhere by their margins, the cornea is hazy and the conjunctiva is extensively ulcerated—the condition being much more distressing than that produced by liquid mustard gas in 24 hours.

The prognosis for saving the eye is almost hopeless if untreated.

(2) *Skin burns due to liquid lewisite.*

(*a*) *On bare skin.*—Like 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 intolerable if an extensive area or a sensitive surface be affected.

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.

The absorption of enough liquid lewisite from the bare skin may result in acute arsenical poisoning. Animals thus poisoned show hæmorrhagic inflammation of the gall bladder and bile duct, necrosis of the liver, inflammation and degeneration of the kidney, albuminuria, anæmia, increase in blood urea, and concentration of the blood as in shock. From such experiments it appears that 1 c.cm. of lewisite would prove a fatal dose for man by skin absorption and that 0·3 c.cm. would cause serious illness. Such heavy contamination of the bare skin might be caused by the bursting of a lewisite bomb close to a person, or by lewisite spray released from a low-flying aeroplane.

(*b*) *On clothed skin.*—Investigation shows that on *dry* clothing, as on bare skin, very small drops of lewisite are less dangerous than similar drops of liquid mustard gas ; whereas when the drops are large, lewisite can produce casualties more quickly and of a more severe type than liquid mustard gas.

With *wet* clothing the position is different. Under these conditions small drops of lewisite will be ineffective, while large drops will lose much of their efficiency owing to the rapid hydrolysis which the vesicant undergoes in contact with the moisture present in the garment. This is in marked contrast with the behaviour of the more stable liquid mustard gas which, as already noted, is usually aided in its penetration, rather than hindered, by moisture.

Unless the contamination be massive, or the clothing thin and scanty, the action of liquid lewisite on the clothed skin may be ascribed entirely to the vapour evolved.

28. Dangers to be anticipated from lewisite vapour.

The irritant character of lewisite vapour, even in low concentrations, ensures its speedy detection, and compels all personnel, unless disabled, to seek the protection afforded by a respirator. This should ensure immunity for the eyes and the respiratory tract, but in the absence of protective clothing the skin is liable to suffer through the action of the vapour absorbed by the garments, especially if these be dry.

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, with ill-trained personnel, may lead to a lack of confidence in the respirator.

29. Lesions produced by lewisite vapour.

(1) *Action on the eyes.*

The irritancy of lewisite vapour and the consequent immediate adoption of protective measures tend to minimize harmful effects on the eye, while the lacrimation which it induces is an additional factor of safety.

Exceptionally, as in the case of disabled personnel, severe burning may occur, analogous to that produced by mustard gas vapour but much more acute. In 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 naso-pharynx is affected within a few minutes, and symptoms of coryza, with salivation and laryngeal irritation, quickly supervene. These are followed by a generalized bronchitis which is well established in 24 hours and may lead to broncho-pneumonia and death.

(3) *Action on the skin.*

The vapour of lewisite is less effective, at ordinary temperatures, than mustard gas vapour in similar concentration. At low temperatures, however, when mustard gas may be inactive, lewisite vapour may still burn the skin, while at high temperatures it will burn the bare skin severely and rapidly.

It has already been noted that wet clothing is an effective safeguard against lewisite vapour.

Unless in high concentration on a hot, receptive skin, or on prolonged exposure (as when wearing contaminated clothing), the vapour causes no more than erythema. When vesication occurs, the skin presents an angry surface, bearing small, shallow, turbid blisters which may coalesce to form one large vesicle. The irritation is worse than with a corresponding mustard gas vapour burn.

30. Protection against lewisite.

Oilskins and oil-treated fabrics which will long resist penetration by liquid mustard gas are not so successful in keeping out liquid lewisite. On the other hand rubber and leather articles are more resistant to lewisite than to liquid mustard gas. It must not be inferred, however, that efficient protection against mustard gas implies inadequate defence against lewisite, or the converse. The only deduction permissible is that, whatever the type of contaminant, all protective garments must be discarded at the earliest possible moment after contamination.

As a protection against the *vapour* of lewisite, the standard protective clothing is subject to the same drawbacks that limit its usefulness in the case of mustard gas vapour, namely, the suction effect of body movements and the interference with aeration of the skin surface. The comparative ease with which lewisite can be neutralized, however, renders it unlikely that protective clothing will have to be worn so frequently or for such long periods as in the presence of mustard gas, except at decontaminating centres.

31. Preventive treatment after lewisite contamination.

With lewisite, even more than with mustard gas, speed is of the utmost importance.

Theoretically, lewisite may be neutralized locally by the application of water, especially if the latter be hot and contains an alkali ; it may also be removed by solvents, or it may be destroyed chemically by aqueous bleach,

as in the case of mustard gas. It should be noted, however, that the original bleach ointment (ointment, anti-gas, No. 1) is of no value in preventing burns by lewisite, so No. 2 or No. 3 must be used (*see* p. 17). Practically, it has been found that none of these methods will prevent a burn after liquid contamination of the skin unless applied within one minute.

Anti-gas ointments Nos. 2 and 3 and aqueous bleach cream act by changing lewisite (and arsenical liquids generally) into compounds which are relatively non-vesicant but are still toxic when absorbed. Hydrogen peroxide and hyperol oxidize trivalent arsenic to the far less toxic pentavalent arsenic, and are thus of considerable therapeutic value. If the skin is heavily contaminated with arsenicals, the danger of systemic poisoning can be lessened by *prompt* application of hydrogen peroxide, 10 vols., or hyperol (10 g. in 100 c.cm. water). The peroxide or hyperol should be freely applied to the affected area with a swab, and preferably three changes of swab, for 2–3 minutes ; and then a piece of lint, gauze, or cotton wool saturated with peroxide or hyperol should be left in close contact with the skin for 1–2 hours, or longer if possible.

This prolonged treatment, which can be used even if anti-gas ointment has been applied previously, will lessen the systemic poisoning, though it will not necessarily prevent vesication. Prompt removal of contaminated clothing is essential.

The most effective means of countering lewisite, however, is the application of " anti-arsenical " agents, which are capable of preventing damage to the eyes or skin.

32. Curative treatment for lewisite casualties.

Since the presence of lewisite vapour in the air can be readily detected, lesions of the eye caused by lewisite vapour should be less numerous and less serious than with mustard gas vapour ; but if liquid lewisite gets into the eye the spasm and rapid swelling of the eyelids, and pain, will render early treatment difficult. The immediate use of an anti-arsenical agent is essential.

Even when applied after erythema is evident, these agents may prevent vesication of the skin. Should blisters form, they must be evacuated as soon as possible, with removal of the epithelium and irrigation to lessen the danger of absorption of arsenic. If an anti-arsenical agent is not available, hydrogen peroxide, hyperol, or potassium permanganate may be employed.

Should symptoms of shock, accompanied by plasma loss and increased concentration of the blood, result from a heavy contamination of the bare skin with liquid lewisite, treatment by transfusion of plasma will naturally suggest itself ; but, since lewisite continues to exert its toxic action so long as it is being absorbed, this treatment is unlikely to be successful unless the area of contaminated skin has been treated at a sufficiently early stage with an anti-arsenical or an oxidising agent. Plasma transfusion has, in fact, been shown to be useless, indeed dangerous.

Signs of renal and hepatic congestion, gastric and intestinal disturbance, and the presence of arsenic in the urine, may afford evidence of arsenical absorption.

33. Lewisite contamination of equipment and supplies.

The general remarks in Section 21 are equally applicable to lewisite.

ETHYLDICHLORARSINE

34. Properties of ethyldichlorarsine.

This gas, familiarly known as " Dick," is a clear, oily liquid soluble in benzene. Its fruity smell may be imperceptible because it immediately irritates

the nose, even in low concentrations of vapour. It penetrates clothing and boots at about the same speed as lewisite, but is much less persistent than mustard gas. It is decomposed by water, but contaminates food or water with arsenic.

As a vesicant, ethyldichlorarsine is considerably less powerful than mustard gas, and as a nasal irritant considerably less powerful than diphenylcyanarsine. As a lung irritant it is rather less toxic than phosgene, and since the immediate irritation it causes will induce fit men to put on their respirators, serious effects on the lungs should be seldom seen.

The action of liquid and gaseous ethyldichlorarsine on the eyes is similar to that of lewisite, but the irritation and lacrimation induced by the vapour is evanescent. Drops of the liquid produce stinging and irritation of the skin with erythema in 1–2 hours and vesication in 4–8 hours. The vesicles resemble those of lewisite, and symptoms of arsenical poisoning may follow contamination of the skin. Vapour, too, may evoke erythema, though the danger is relatively small since the gas is unlikely to persist long in adequate concentration. The effect on the upper respiratory tract is transient ; but if no respirator is worn ethyldichlorarsine may produce tracheitis, bronchitis and later pneumonia.

The Service respirator gives complete protection to the eyes, nose, throat and lungs. Anti-gas ointments Nos. 2 and 3 are effective.

35. Treatment for ethyldichlorarsine casualties.

For lesions of the eyes and skin the treatment is the same as for lewisite. An anti-arsenical agent should be applied to the eyes or skin and blisters should be opened. Hydrogen peroxide or hyperol are useful alternatives for skin lesions. Pulmonary and nasal irritation should be dealt with as described in Sections 44 and 65.

OTHER VESICANTS

36. Besides mustard gas, lewisite and ethyldichlorarsine, there are other colourless or yellowish-brown liquids, capable of employment in chemical warfare, which are generally similar to these gases in their action, but are less readily detected by smell or sensory irritation. Their smells are relatively feeble and different people apply widely different descriptions to them, such as " fishy," " like soft soap " and " fruity." Smell or sensory irritation may be completely lacking in concentrations of vapour which would nevertheless be dangerous if exposure were prolonged. In liquid form, however, they give the usual blister gas reaction on the Service detector paint, and the vapour may be detected by means of special papers.

The general toxicological properties of these substances are similar to those of mustard gas, including systemic effects. In brief, it may be said that their effects on the skin are somewhat less than those of mustard gas or lewisite, while the effects of their vapour on the eyes are rather greater than those of mustard gas vapour. In liquid form their effects on the eyes are rather less severe and less immediately painful than those of *liquid* lewisite. Compared with mustard gas, the damage to the eye may be deeper, and iridocyclitis of hæmorrhagic type may be encountered. Ciliary spasm may be severe.

The treatment necessitated by exposure to these gases should be along the lines recommended for mustard gas. The following points should be noted :—

Liquid in the eye.—Immediate and copious irrigation is the best treatment so far discovered, but if delayed longer than two minutes will not prevent a

severe casualty. To relieve ciliary spasm, repeated dosage with 1 per cent. atropine may be necessary.

Vapour in the eye.—All cases with contracted pupils will require at least one dose of atropine.

Contaminated food and water.—Food and water suspected of contamination by these gases must be rejected.

CHAPTER III

THE LUNG IRRITANT OR "CHOKING" GASES

37. General description of lung irritant gases.

The chief members of this group are *Chlorine, Chloropicrin, Phosgene* (carbonyl chloride), and *Diphosgene* (trichlormethyl chloroformate). Chlorine and phosgene are true gases under normal conditions of temperature and pressure, while chloropicrin and diphosgene 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 Great 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 causes a violent and paroxysmal cough which persists after the cessation of exposure. It is of much lower toxicity than phosgene or diphosgene.

Chloropicrin (P.S.) 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 exposures to small doses of this gas may gradually lead to a greatly increased susceptibility with a liability to attacks of nocturnal asthma. It is a strong lacrimator.

Phosgene (C.G.) is the most important member of the group, and the most toxic. It is a liquid which boils at 8·2° C. (46·8° F.) with the evolution of a colourless gas. Although the least immediately irritant of the group, phosgene gas is readily detectable, even in concentrations which are harmless to the lungs, by its characteristic smell of musty hay.

Diphosgene is an oily liquid boiling at 128° C. (262·4° F.), and smelling like phosgene. Apart from its more pronounced lacrimatory power, the symptoms produced by this gas are practically identical with those caused by phosgene.

It will be recalled that vesicant vapours also attack the lungs and upper respiratory tract (Sections 15, 18, 29, and 34); but, unlike the "lung irritants," they do not induce acute pulmonary œdema.

38. Methods of dispersion.

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. Both chlorine and phosgene may be dispersed from cylinders

mounted in fixed positions or carried on moving vehicles; the emission of phosgene is hastened, in cold weather, by mixture with chlorine.

By whatever means these chemical agents are distributed, the resultant gas clouds are carried downwind. The sudden release of a heavy cloud of lung irritant gas in enclosed areas or in close proximity to personnel is fraught with danger even to men supplied with (but not wearing) respirators, since the intense spasm induced by a single breath of a heavy concentration of the gas may interfere with their adjusting them quickly.

39. Mode of action of lung irritant gases.

All lung irritant gases attack the alveoli of the lungs and the smaller bronchial tubes, and the great danger to be feared is acute pulmonary œdema. The rate of onset and the amount of œdema 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 lacrimators, especially chloropicrin and diphosgene; but they are far less powerful in this respect than the true lacrimators.

The toxicity of phosgene and diphosgene is approximately the same. In lethal concentrations chloropicrin is about four times, and phosgene practically ten times, more toxic than chlorine. While chlorine and chloropicrin cause more damage to the lining membranes of the respiratory passages, phosgene is more liable to produce pulmonary œdema.

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, and this varies as much with the concentration of the gas as with its particular nature.

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

40. Morbid anatomy. (*See* Appendix IV, Plate VIII.)

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

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

When death occurs later on the first day the lungs are voluminous, heavily œdematous 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 hæmorrhages 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.

After the second or third day there is aeration of the lungs, especially the lower lobes, and 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 œdema fluid is rapidly absorbed from the lungs from the second day onwards.

In severe cases the hæmoglobin percentages may rise as high as 140 with a corresponding rise in the red cell count. Associated with this concentration is 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 conspicuous in these cases than in phosgene poisoning. Subcutaneous emphysema was rarely seen in the Great War except after gassing by chlorine.

41. General symptoms and signs of lung irritant poisoning.

Casualties may be classified into two chief groups: Acute with violent onset, and Acute with insidious onset.

(1) *Acute with violent onset*

Exposure to an atmosphere containing phosgene causes immediate sensory irritation of the respiratory passages accompanied by smarting and watering of the eyes. This irritation of the respiratory passages causes catching of the breath, coughing and a sensation of tightness and constriction and pain in the chest. After the initial check, the breathing continues, but is gasping in character and interrupted by violent fits of coughing. After getting out of the poisonous atmosphere the respiration remains rapid and shallow, any attempt to draw a deep breath giving rise to painful discomfort and provoking a fit of coughing. Nausea, retching, and vomiting are prominent features in the early stages of poisoning and may make it difficult to wear a respirator. There is slight or profuse expectoration. Headache, and a sense of fatigue in all the limbs, often prostrate the patient.

As œdema develops in the lungs, the breathing becomes rapid and panting, but shallow—unlike the deep ventilation of a healthy man after running, and more resembling the fast respiration of a child with broncho-pneumonia. The sputum is frothy. The ears, lips and progressively the entire face assume a cyanotic, bluish-red tint which may deepen to the intense violet of fullest cyanosis, and there may be visible distension of the superficial veins of the face, neck or chest—especially in persons gassed with pure chlorine. 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 in the face, in which only the lips and tips of the ears reveal the asphyxial cyanosis that underlies the failure of the man to win his fight for life. While in the stage of cyanosis, whether "blue" or "grey," the patient is always restless and very apprehensive of the seriousness of his condition. The expression is anxious and distressed, with the eyeballs staring and the lids half closed. At this stage casualties can be divided into three types:—

(*a*) The milder case, with reddish flush in the face, with some hurry of respiration, and with pain in the chest and epigastrium which is increased by coughing.

(*b*) The severe case with "blue" cyanosed face, distended neck veins, and full, strong pulse of 100 (*see* Appendix IV, Plate IX).

(*c*) The severely collapsed case, with leaden "grey" cyanosis of the face, and rapid, thready pulse (*see* Appendix IV, Plate X).

The milder case is often drowsy and soon falls into a sleep from which he wakes refreshed. Coughing upon a deep breath, occasional vomiting after food or drink and a slight sense of rawness in the throat, together with general debility, may persist for a few days, after which the patient becomes convalescent. During the early days of convalescence there is often a considerable slowing of the pulse from vagus action, which may bring it down to about 50 or even 45 in the minute. Such early bradycardia is often seen also in recovery from severe poisoning; it has no serious import, but is rather a sign that the patient is beginning to convalesce.

Cases of severer cyanosis, if the depth of the reddish-blue colour is well maintained and the pulse does not exceed 100, tend to recover in two or three days, and their recovery is generally similar to that of the milder cases. Provided that the circulation and the activity of the respiratory centre can be maintained, the œdematous fluid in the lungs is soon absorbed, most of it vanishing by the fourth or fifth day. At any time, however, particularly if subjected to much physical effort, these cases may rapidly pass into the most dangerous condition of "grey" cyanosis and collapse. The pulse becomes rapid, thready and irregular. The patient, though obviously weaker, becomes more restless and slightly wandering in mind, or semi-comatose.

Even the worst of the "grey" cases may recover with proper treatment, but the mortality among them is always distressingly high. Recovery from this state of depressed circulation may be succeeded by broncho-pneumonia. When this infective complication develops, the sputum becomes purulent and the temperature rises. Death may follow rapidly.

In 81 per cent. of deaths due to poisoning by phosgene and chlorine the death occurred within 24 hours.

(2) *Acute cases with insidious onset*

Cases have been frequently reported in which men who have been exposed to gas have been able to carry on their work for an hour or two with only trivial discomfort, and even to march from their trenches to their billets, and then have become rapidly worse, with collapse and progressive œdema of the lungs. At other times men who have passed through a gas attack and have complained of only slight cough, nausea, and tightness of the chest, have collapsed and even died abruptly some hours later on making some vigorous muscular effort. A minor degree of the same effect has sometimes been shown when men who have been slightly gassed found on trying to walk down from the trenches that they got unusually "done in" and breathless, and were obliged to rest frequently. In these cases the deficiency of oxygen, the result probably of pulmonary œdema already developing, has not been felt until muscular exertion increased the need for oxygen.

This delay in the onset of serious symptoms is a striking feature in poisoning by the lung irritant gases, especially phosgene and diphosgene. It is not so evident in cases of poisoning by chlorine or chloropicrin when the violent paroxysms of coughing, the painful dyspnœa, and the vomiting convey the impression that the patient is seriously ill from the start.

42. Chest signs in lung irritant poisoning.

The percussion note may remain resonant over the chest, notwithstanding the existence of pulmonary œdema. The breath sounds are weakened, especially over the back; they may also be harsh in character, but never tubular. Fine râles 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 chest 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.

43. Prognosis of lung irritant cases.

Cases of the " blue " type which react favourably to the administration of oxygen usually do well ; the fluid in the lungs may be 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 patient lasts into the third week after gassing he may justly be expected to survive the acute infection.

In the Great War broncho-pneumonia was found to be more frequent and more 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 œdema since the margin available for respiration was correspondingly less.

44. Treatment of lung irritant cases.

It may be difficult to decide whether a man has really been gassed. The history of the case must be taken into account. But the benefit of the doubt should always be given to the patient, 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 returned to duty with little delay.

(1) *Treatment in the acute stage*

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

(*a*) *Rest.*—The importance of rest cannot be exaggerated ; in the earlier stages exertion may aggravate the symptoms, while in the later stages, when the oxygen supply is impaired by pulmonary œdema, any attempt to perform muscular work may have disastrous consequences.

All cases should, if possible, be evacuated lying down, with collars, belts and braces eased so as not to impede breathing. If this is not possible even the lightest walking cases should be given every assistance and should be relieved of all equipment so that they may be spared physical effort. As with every type of gas casualty, the patient's contaminated clothing should be removed on reaching the treatment centre. Those who show definite symptoms should not be allowed to leave their beds or stretchers for any purpose whatever.

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

(*c*) *Oxygen.*—Oxygen should always be given to casualties with serious pulmonary œdema—that is, to those with 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 the B.L.B. or Haldane mask, or an oxygen tent, that will ensure a suitable mixture with air (*see* Appendix II).

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. 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.

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

The experience of the Great 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 œdema.

(*d*) *Venesection.*—Deep cyanosis with venous engorgement is an indication for venesection, since this may relieve the right heart which has begun to dilate owing to lack of oxygen and obstruction of the pulmonary circulation. Some 300–400 c.cm. should be withdrawn through a large-bore needle or by cutting a vein. Relief of headache and dyspnœa often results, but if cyanosis still continues further venesection is advisable, up to a total of 700 c.cm.

Venesection is contra-indicated in cases showing a leaden grey colour with circulatory failure, very low blood pressure and a small, rapid or impalpable pulse.

Although animal experiments in the last war suggested that it would be an advantage if venesection were associated with infusion of isotonic salt solution, recent experiments have shown clearly that the infusion of saline, serum or plasma fails to restore the blood volume if hæmoconcentration has occurred, but does accentuate the pulmonary œdema and may thus accelerate death. Transfusions and infusions, beneficial though they may be in cases of surgical shock accompanied by hæmoconcentration, are therefore inadmissible in cases of pulmonary œdema caused by the lung irritant gases.

(2) *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. The patient's head should be turned sideways.

Expectorants should not be given to severe cases, at any rate during the first two or three days, for fear of increasing the tendency to cough and so augmenting the damage in the lungs.

No drugs were found to be of any special value in the Great War. Attempts to reduce the permeability of the lung tissue to fluid by administration of calcium chloride were unsuccessful, and recent experiments on animals have shown that raising the concentration of calcium in the blood by the injection of parathyroid extract or calcium gluconate (subcutaneously or intramuscularly) is just as useless. Large doses of calcium chloride or gluconate are damaging to the tissues and may cause sloughing of the skin. Atropine does not check œdema or relieve bronchial spasm, while morphia is dangerous and should be used only in small doses (gr. $\frac{1}{6}$), to control extreme restlessness. Camphor and Coramine may be useful in collapse, but strychnine, digitalis and alcohol are contra-indicated and adrenaline is ineffective. The relief of asphyxia is the best means of relieving the headache and the best cardiac stimulant is oxygen.

If pulmonary complications, such as infective bronchitis or broncho-pneumonia, develop, 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. Sulphonamide drugs should be given.

(3) *Treatment in 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 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 long 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 contra-indicate this ; but men with abnormally rapid or slow pulses 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, however, must be carefully studied, and exhaustion must be rigidly guarded against ; for effort syndrome may develop and delay recovery for weeks or months.

45. Protection against lung irritant gases.

The protection afforded by the respirator is complete. Moreover, all the members of the group are obvious to the senses in concentrations that can be breathed without serious danger. It follows, therefore, that unless an enemy can exploit an asphyxiant cloud in a concentration that is effective before personnel have time to adjust their respirators, casualties from gases of this group should be infrequent.

46. 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 sequelæ of poisoning by the lung irritant gases in the Great War were, contrary to popular belief, much less grave than was anticipated. The great majority of men were restored to good health. Some, however, remained incapable of severe muscular effort or even of moderate exercise, showing tachycardia and a rapid shallow type of breathing. Recurring frontal headache, generally worse after exercise, and transient epigastric pains were frequent ; and, while pain in the chest was variable, a mild bronchitis continued in an appreciable proportion of cases.

Two important, though not common, sequelæ which prolonged invalidism were effort syndrome and nocturnal " asthma."

Effort syndrome, with præcordial pain, a sense of exhaustion, dyspnœa and persistent tachycardia after exercise, but with no sign of organic heart disease, was the commoner and more persistent. A small proportion proved intractable, and there was evidence that this form of disability increased when 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 3 to 30 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 hæmoglobin 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.

Patients who have had infective broncho-pneumonia should always be considered as a group apart.

47. Invalidism after lung irritant poisoning.

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 œdema 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 dyspnœa, 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 Great War was made, and it soon became evident that, in spite of what was being written in certain countries, there was no reason to suppose that gassing induced tuberculosis. On the other hand, as would be expected, it is unquestionable that gas aggravated any pre-existing tuberculous condition.

CHAPTER IV

PARALYSANT GASES

48. 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 Great War in the open field, where they caused but few casualties. Nevertheless, a study of the gas is essential as it may be used again in different circumstances.

Mode of action.—The gas 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 low concentration it may be detoxicated in the body as quickly as it is absorbed.

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

Attention may be drawn to a danger in the use of hydrocyanic acid gas when it is employed for the destruction of vermin, such as rats in ships, or in the disinfection of rooms. A suitable respirator eliminates the danger of its inhalation, but since this gas may be absorbed by the skin it is dangerous to remain long, even with a respirator, in the high concentrations employed against vermin.

Owing to the ease with which the gas dissolves in water, the skin absorption danger is greatly increased if the weather be hot 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.

Protection.—Ordinary charcoal respirators give only limited protection against hydrocyanic acid gas, but special containers containing pads impregnated with caustic soda, etc., are available which give effective protection. These depend on the neutralization of the gas by chemical action, and are fitted with indicating devices which give timely warning when the useful life of the respirator is nearing its end.

An indirect form of protection, applicable to confined spaces, is the employment of 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.

Treatment.—Treatment must be immediate, and the primary, urgent necessity after removing the victim from the poisonous atmosphere is to reduce the concentration of the gas in the circulation. This can be achieved by artificial respiration, preferably in conjunction with the administration of oxygen with an admixture of 5 to 7 per cent. of carbon dioxide to stimulate the respiratory centre. Coramine may be tried in desperate cases, 5 c.cm. of 25 per cent. solution being injected slowly into a vein.

Various prophylactic and antidotal methods have been suggested based on the laboratory neutralization of hydrocyanic acid gas by chemicals such as sodium thiosulphate, methylene blue, and glucose; but very little success has, so far, followed their adoption.

49. Hydrogen sulphide (Sulphuretted Hydrogen).

Physical and chemical characteristics.—Hydrogen sulphide is a colourless gas with a fœtid 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 irritating to the eyes and throat, may be unrecognizable by smell. The gas, which is inflammable, is heavier than air, and may form explosive mixtures in tunnels, cellars, dug-outs, and other confined spaces. Hydrogen sulphide was tried in the Great War as an offensive gas, but its use was abandoned.

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.

In high concentration the gas produces unconsciousness with the same dramatic suddenness as hydrocyanic acid, both causing paralysis of the respiratory centre in the brain. Moderate concentrations give rise to pulmonary œdema. There is no evidence that abnormal combinations with hæmoglobin are formed.

Symptoms.—Usually the first signs of acute poisoning are panting respiration, pallor and rapid unconsciousness ; breathing soon ceases and there are often convulsive movements. The heart continues to beat for some minutes, but will fail unless the victim is extracted from the gassed area and artificial respiration is begun immediately. In less acute cases violent irritation of the eyes and severe inflammation of the respiratory tract, which may prove fatal, are the most prominent symptoms.

The subacute poisoning met with occasionally in industrial life is not likely to be seen frequently in war time ; it gives rise to general ill-health, with chronic conjunctivitis and affections of the respiratory and digestive tracts.

Protection.—Adequate protection against its use in the field is provided by the respirator. If concentrations of the gas are suspected in dug-outs, cellars or other closed spaces, they 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.

Treatment.—As in the case of poisoning by hydrocyanic acid gas, treatment must be prompt. It consists essentially in the immediate removal of the victim from the poisonous atmosphere and the administration of artificia respiration, preferably with inhalation of air or oxygen mixed with 5 to 7 per cent. of carbon dioxide. 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. The effectiveness of this has been proved by industrial practice.

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

CHAPTER V

ARSENIURETTED HYDROGEN (ARSINE)

50. General.

A colourless, inflammable gas, which, when liquefied, boils at about −60° C. It is formed, together with hydrogen, when acids react with metals containing arsenic as an impurity, and is readily evolved by the action of water on calcium, magnesium and sodium arsenides, or of dilute acid on other metallic arsenides. In addition, therefore, to its potential chemical warfare use as a gas in cylinder attacks, or as a non-persistent charging in shell and bombs, there is also the possibility that one of its progenitors, e.g. calcium arsenide, might be disseminated in the form of a dark-grey heavy powder, which in contact with atmospheric and soil moisture would slowly generate arseniuretted hydrogen in situ, over a period of hours or days, according to conditions.

In moderately strong concentrations arseniuretted hydrogen possesses a nauseating garlic-like odour (frequently described as reminiscent of that of acetylene or phosphorus), and gives rise to a flat metallic taste in the mouth ; but when largely diluted with air, although possibly present in toxic concentrations, it may be imperceptible to the senses. It is non-irritant to the eyes,

the respiratory passages and lungs, and the skin, and exerts its poisonous action only after absorption through the lungs into the body. The most characteristic feature of its action is the destruction of the red corpuscles of the blood, with the consequent appearance of hæmoglobin in the urine and the development of jaundice and anæmia, but the kidney and liver are also directly affected by the absorbed arsenic. Exposure for a few minutes to a high concentration of arseniuretted hydrogen may be rapidly fatal. Even extremely low concentrations, however, can cause symptoms of poisoning, though slow in onset and mild in degree, provided that the exposure is long enough, i.e. many hours or even days.

The presence of arseniuretted hydrogen in the atmosphere can be detected by means of test-papers impregnated with mercuric chloride or bromide which develop a yellow or orange colour when exposed for some time even to very low concentrations of the gas. Such papers are liable to give a similar colour on exposure to air in industrial areas owing to reaction with the hydrogen sulphide present, and under such conditions the test for arseniuretted hydrogen should be confirmed with a differential detector in which the interfering gas is removed by passage through lead acetate granules.

51. Symptoms.

In *severe cases* due to the inhalation of a relatively high concentration of the gas, symptoms develop rapidly—shivering, headache, pain in the epigastrium and over the kidneys, vomiting, breathlessness, weakness and giddiness, the patient gradually becoming comatose with a steady deterioration of the pulse. Evidence of serious destruction of the red corpuscles in the blood is soon afforded by the passage of a deeply coloured urine, but in the worst cases the urine may be very scanty and there may even be anuria with an intensification of the pain over the kidneys. If the patient survives, intense jaundice accompanied by enlargement of the liver soon ensues.

In *cases of moderate severity* the symptoms are of the same general type—shivering, weakness, giddiness, nausea and vomiting, headache often associated with insomnia, renal and hepatic pain, dyspnœa on exertion, and often diarrhœa. Headache and repeated vomiting seem to be an almost constant feature. Hæmoglobinuria soon makes its appearance, the colour of the urine varying from deep red (port wine colour and sometimes almost black) to reddish brown, the difference probably depending on the relative amounts of unaltered hæmoglobin, methæmoglobin, bile pigments and perhaps acid hæmatin (in an acid urine) that are being excreted. The urine also contains albumin and casts. Jaundice becomes apparent a little later on.

In *mild cases* anæmia may develop gradually, with a progressive fall in the red cell count and the hæmoglobin in the blood, without the appearance of hæmoglobin in the urine and with little or no jaundice, this anæmia being sufficient to account for symptoms of lassitude, headache and general malaise as well as undue breathlessness on exertion.

In the earlier stages the occurrence of tingling, or pins and needles, in the hands and feet, which has been reported by some authors, may possibly be due to the toxic effect of arsenic on the nervous system ; but such a phenomenon should be distinguished from a true arsenical neuritis which occasionally develops at a later stage, causing pain in the nerve trunks, particularly of the extremities, and some dulling of sensation.

52. Pathology.

Arseniuretted hydrogen is taken up chiefly by the red blood corpuscles, from which it is released by hæmolysis ; the hæmoglobin set free, some of which is converted to methæmoglobin, is partly excreted by the kidney and

partly converted by the liver into bile pigments which are formed in excessive amounts and give rise to jaundice. Hæmolysis may continue for several days after exposure to the gas and anæmia is therefore progressive. While hæmolysis is taking place a simple estimation of the hæmoglobin percentage in the whole blood will give an erroneous idea of the extent of red cell destruction since much of the hæmoglobin may be in solution in the plasma and not in the corpuscles. A red cell count will make the situation clear.

In the absence of a centrifuge, hæmolysis may be recognized by making equal dilutions of the patient's blood and of normal blood and comparing the supernatant fluids after sedimentation. The following technique is convenient :—

Prick the finger of the patient and allow three drops of his blood to fall into twelve drops of 3·8 per cent. sodium citrate solution in a watch glass or small tube. Mix well and draw up the mixture into a glass tube of length 20 cm. and internal bore 2–3 mm., the end of which has been slightly tapered, as for a pipette. Close the lower end of the tube with a short length of narrow rubber tubing that has been stoppered with a piece of glass rod or clipped. Prepare a precisely similar tube with the blood of a *normal* person to serve as a control. Stand both tubes vertical for half an hour or longer so that the corpuscles may sediment. If there is any hæmolysis in the suspected blood it will be shown by the tint of the supernatant fluid when compared with that of the control.

Even in the early stages of poisoning there is evidence of increasing damage of the kidneys, liver and spleen, and these organs are intensely congested. This is soon followed by tubular degeneration in the kidneys, with excretion of blood casts and toxic degeneration and necrosis of the liver. With incipient recovery the excretion of blood pigments ceases, though albuminuria may continue for a considerable time ; the blood count reaches a minimum value which may be well below 2 million red cells per cubic millimetre ; blood regeneration is manifested by poikilocytosis and anisocytosis as well as by the appearance of nucleated red corpuscles and reticulocytes ; and the damage to the kidney and liver is slowly repaired. When hæmoglobinuria is very severe there is some risk that actual blood clots may form in the bladder.

Death in the most severe cases may take place within 48 hours of exposure and may be preceded by complete anuria. Although it has been suggested that such anuria is caused by blockage of the capillaries of the kidney by the debris of red cells, and of the tubules by cellular debris and casts, it is more probably attributable to acute arsenical poisoning of the kidney, coupled with a failing circulation, and rapid death must be attributed as much to the poisonous effects of arsenic on the organs of the body as to the results of acute blood destruction.

53. Treatment.

(1) *Blood transfusion* is indicated whenever severe anæmia is shown either clinically, by percentage of hæmoglobin, or by the number of red cells, and certainly if the hæmoglobin falls to 60 per cent. or the red cells to 4,000,000 per c.mm. The risk of hæmolysis of the transfused blood by the gas is apparently slight after an interval of two to three hours after exposure. Transfusion may need to be repeated since hæmolysis may continue for several days. For a single transfusion 500 c.cm. is an advisable quantity. Owing to the general toxic effect of the gas the destruction of red cells which it causes cannot be regarded in the same light as the loss of an equal quantity of blood by a simple hæmorrhage, and transfusion should, therefore, be done slowly, careful observation being kept on the general circulation and the blood pressure, lest over-filling of the circulation should damage a heart already poisoned.

(2) *Diuresis* is beneficial, and liquids should be given freely by the mouth or if necessary per rectum. The quantity should be about four pints in 24 hours. Arsenic is largely excreted through the kidneys and a good flow of urine will lessen the risk of blockage of the renal tubules by cellular debris. It must, however, be remembered that in arsine poisoning there is no loss of fluid from the circulation ; so both intake and output must be watched to guard against retention of fluid leading to œdema.

Alkalis (*see* below) will aid diuresis, but organic mercurial diuretics should not be employed.

(3) *Administration of alkalis.*—Potassium, or preferably sodium, citrate should be administered in doses sufficient to maintain an alkaline urine. A dosage of three or four drachms in 24 hours will usually be sufficient. It is known that with hæmaturia the hæmoglobin excreted may be converted to acid hæmatin in the tubules if the reaction of the urine is acid, and it has been suggested that this pigment may crystallize and thus block the tubules. Experimentally, there is little indication that such crystallization of acid hæmatin occurs in cases of arseniuretted hydrogen poisoning. Nevertheless the administration of alkalis may be a wise precaution for the present.

(4) *Administration of glucose.*—Glucose should be given freely from the onset, either with or without insulin. It is especially indicated if there is evidence of necrosis of the liver and if necessary may then be injected intravenously.

(5) *Oxygen administration.*—Moderate anæmia does not cause anoxæmia, but oxygen may be useful when anæmia is far advanced. In arseniuretted hydrogen poisoning the lungs are practically unaffected and there is no true cyanosis, and there may still be a considerable amount of functional hæmoglobin in the blood even if much of it is in solution in the plasma. Consequently, it is unlikely that oxygen administration will have the dramatic effect it has in acute pulmonary œdema due to the lung irritant gases. Anoxæmia may, however, occur in the absence of cyanosis.

(6) *Special risks.*—In the wounded, watch must be kept for secondary hæmorrhage. In choosing anæsthetics the probable liver damage needs consideration.

(7) *Convalescence.*—It must be borne in mind that arseniuretted hydrogen causes necrosis of the liver and other organs. Light diet is indicated so long as the urine contains evidence of this toxic action.

The anæmia will need full doses of iron and treatment with liver extract may be useful.

Cases must be kept under observation until both the blood and the urine are normal.

54. Differential diagnosis.

The diseases which may possibly be confused with arseniuretted hydrogen poisoning are blackwater fever and paroxysmal hæmoglobinuria. A history of residence in the tropics, malaria, and quite recent use of quinine, taken in conjunction with the high temperature which is usual, will suggest the former ; while association with exposure to cold, as well as the previous history, will suggest the latter. Such diseases are of course a most improbable cause of hæmoglobinuria and anæmia developing in a group of people simultaneously. Nor should there be any great difficulty in differentiating jaundice due to arsine poisoning from jaundice due to other causes.

CHAPTER VI

GASES USED PRIMARILY AS HARASSING AGENTS

55. General description of harassing agents.

This group includes certain gases or irritant smokes which, as met with in the open field, cause symptoms of distress without any subsequent physical injury. Typical examples are the following :—

(1) *Lacrimators* or " tear gases," such as :—

Ethyliodoacetate (K.S.K.).
Bromobenzyl cyanide (B.B.C.), and
Chloracetophenone (C.A.P.).

(2) *Nasal irritants* or " nose gases," organic arsenical compounds such as :—

Diphenylchlorarsine (D.A.),
Diphenylamine-chlorarsine (D.M.), and
Diphenylcyanarsine (D.C.).

The primary object in the employment of these compounds is to harass troops by causing acute, though temporary, distress, thereby compelling them to wear respirators, possibly for prolonged periods. This tends to reduce efficiency, and, if anti-gas training be defective, to have a lowering effect on morale ; this applies particularly to the more insidious nasal irritants.

These harassing gases have several important features in common, viz. :—

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

(ii) The effects of the lacrimators are immediate, but transient. The nasal irritants have a delayed action of a few minutes when compared with the lacrimators, and the discomfort is more lasting ; in each case, however, withdrawal from the hostile atmosphere is followed by recovery.

(iii) They are effective in extremely low concentrations, but they never give rise to permanent disabilities. It is very unusual to incur any physical injury from these irritating gases.

56. Lacrimators or " tear gases."

There are many compounds, both liquid and solid, which may be used as lacrimators in wartime. The following are typical examples :—

(*a*) *Ethyliodoacetate.*—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.

(*b*) *Bromobenzyl cyanide.*—In the pure state this is a yellowish crystalline solid, stable at ordinary temperatures and melting at 24·8° C. (74·6° 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 at 242° C. (467·6° F.) ; it is more stable, and has a lower vapour pressure than ethyliodoacetate, hence it persists longer than the latter as an effective lacrimator when spread on the ground.

(*c*) *Chloracetophenone.*—A colourless, crystalline solid melting at 54° to 59° C. (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.

Chloracetophenone is a very stable compound which does not decompose on heating or detonation ; its lacrimatory effects, however, are soon lost by reason of its condensation to the solid, inert state soon after the initial dispersion, and it is therefore classed among the non-persistent gases. It is used extensively in gas training schools.

57. Methods of dispersion of lacrimators.

The liquid lacrimators may be dispersed by shell or bomb, or by mechanical spraying for purposes of ground contamination. The solid C.A.P. can be dispersed from generators by means of heat, or it may be sprayed, in solution, from the exhaust pipes of internal combustion engines. It is, however, unsuitable for use in shell.

For training purposes, small capsules containing solid C.A.P. are heated over a flame to evolve the gas, while in certain countries effective use of this compound is made by the police against mobs. A variety of more or less harmless weapons such as lacrimatory pistols and hand bombs are employed for this purpose.

58. Action of lacrimators.

Exposure to any of these lacrimators gives rise to an immediate, acute and localized 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 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.

Ethyliodoacetate is less irritating to the respiratory tract than chloroacetophenone. The latter, however, unlike the liquid lacrimators, will irritate the bare skin, especially if it be hot and moist.

As a rule, persons exposed to lacrimators do not exhibit more grave symptoms than those described above, as the very high concentrations necessary to produce lung lesions are not met with in the field. In confined spaces, however, where the actual liquid may be splashed on the skin, or the gas inhaled in high concentration worse 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.

59. Protection against lacrimators.

The respirator affords complete protection to both eyes and lungs against all concentrations of tear gases likely to be met. 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.

The irritant action of C.A.P. and other solid lacrimators on the hot, moist skin is not sufficiently intense in temperate climates to necessitate the use of special protective garments.

60. Treatment of tear gas casualties.

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 is likely to be necessary, and there should be

no after-effects. In the rare cases where conjunctivitis or respiratory affections develop, treatment should be symptomatic and follows general principles. If the eyes be contaminated by the actual liquid from aerial sprays, bursting bombs, etc., treatment should follow the lines laid down for mustard gas (Section 18).

61. Nasal irritants or "nose gases."

These sensory irritants are solid organic arsenical compounds which can be dispersed by heat or detonation in the form of a very fine, almost invisible, particulate cloud or smoke.

They were employed in the Great War in the belief that they would penetrate the respirator then in use and cause such distressing symptoms that the men would 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, however, meet with any striking success, because at that time the methods of release were unsatisfactory.

The following are typical examples of the nasal irritants :—

(*a*) *Diphenylamine-chlorarsine.*—A yellow, almost odourless, crystalline solid melting at 196° 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.

(*b*) *Diphenylcyanarsine.*—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.

It may be noted that diphenylchlorarsine and diphenylcyanarsine have slight vesicant properties. They are chemically related to chlorovinyldichlorarsine (lewisite) and ethyldichlorarsine, which are also sensory irritants though their vesicant action is of greater practical importance.

62. Methods of dispersion of nose gases.

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

They may be heated by means of special incendiary generators which are placed in position by hand or dropped from vehicles, or the solid compound may be incorporated in shell. By these methods it is possible to emit, over a wide front, a cloud of extremely fine particles.

63. Action of nose gases.

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

Their effectiveness depends on the amount inhaled during the few minutes before symptoms come on. Even if the affected person withdraws from the poisonous atmosphere (or puts on his respirator) directly the irritant effects are felt, the symptoms continue to increase in severity for some time before they begin to subside. Whereas the tear gases cause immediate irritation which diminishes as soon as the eyes are protected, the arsenicals have a delayed action which may cause its maximum of distress a little while after the respirator has been adjusted. Unless personnel are trained to expect this, they may lose faith in the respirator, with disastrous results.

The symptoms are characteristic, and consist in acute pain in the nose

and accessory sinuses with a sense of " fullness " in the head and with repeated sneezing (hence the term " sternutators " 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 lacrimation, and aching of the gums are common, while salivation, nausea and even vomiting, are important. Mental distress is very marked in severe cases, in which the patients feel and look utterly miserable. This condition of intense discomfort is very alarming to the inexperienced.

The effects, however, are transitory, and affected men should not leave their units as the symptoms usually disappear within one to four hours. During the Great War cases were reported showing paralyses of limbs, which suggested a central toxic action of the gas ; but recovery took place within 24 hours. Various transient paræsthesiæ developing later were regarded as probably functional, and no lasting organic lesion is likely to follow exposure to these gases in the open.

64. Protection against nose gases.

The respirator charcoal, so effective in arresting the lethal gases, has little protective value against the particulate clouds of these arsenical compounds. Special filtering devices must be employed to trap the arsenical dust in the air, and all modern respirators for use against war gases give adequate protection.

Symptoms of true arsenical poisoning may occur through men using water drawn from 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. It is important, therefore, that stringent orders be issued that no water from shell craters be employed for drinking, cooking or washing.

65. Treatment of nose gas casualties.

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 relief from the medical officer, when the inhalation of a little chloroform will be found useful. A five per cent. solution of sodium bicarbonate for nasal irrigation or as a gargle will help to allay the irritation of the nose and throat. Alcohol and Benzedrine (amphetamine) have been recommended for depression.

CHAPTER VII

CORROSIVE AND INCENDIARY SUBSTANCES

66. Offensive use of corrosive acids.

Corrosive acids might possibly be sprayed from aircraft or projected in jets from armoured fighting vehicles. For example :—

1. *Oleum.*—Sulphuric acid with added sulphur trioxide. Gives off dense white fumes with a sulphurous smell, and has been employed to make screening smoke.
2. *Hydrofluoric acid.*—A colourless liquid forming a mist with moist air.
3. *Nitric acid.*—Yellow liquid changing to reddish brown.

Eyeshields will protect the eyes from acid, and anti-gas capes will also be effective though the oilskin will be damaged. The vapour is not likely to be dangerous in the field, and does not necessitate the wearing of a respirator.

The immediate treatment of an acid burn of the skin is to wipe off the liquid and wash with plenty of running water. Ideally, the skin should then be dried by gentle mopping with swabs of cotton wool, and covered with a moist alkaline dressing (e.g. 3 per cent. sodium bicarbonate solution). Failing this, an ordinary dry dressing can be applied. Anti-gas ointment should not, of course, be used.

The treatment of the eyes requires special consideration.

67. Injury to the eyes by corrosives.

The essential primary measure is prompt and copious lavage : if lavage is immediate and efficient the damage may be greatly mitigated, but its value diminishes with every second that is allowed to pass. Compared with the time factor, the nature of the solution used is unimportant, so long as it is bland. Sodium bicarbonate (1 per cent.) is theoretically ideal, normal saline is good, but water (e.g. from the water bottle) does very well.

If the eyes are spasmodically closed they must be opened ; and if this proves impossible without using undue force no time must be lost in securing skilled assistance so that the lids may be parted and lavage begun. In trying to open the eyes it is useful to interpose a piece of material between the lids and the fingers, so as to minimize slipping. If the lesion seems to be severe a drop of atropine alkaloids should at once be instilled, or (if available) a drop of 1 per cent. water solution of atropine. Lavage should not continue after it is thought that all free chemical has been washed out. Bandaging of the eyes should be avoided when possible ; if the eyes can be opened dark glasses and/or large shades are preferable.

Thereafter treatment consists in the maintenance of cleanliness and the avoidance of secondary infection, with the aid of a mild antiseptic such as Albucid, and the use of a mydriatic. The greatest danger in all caustic burns is symblepharon, and a glass rod may be passed daily between the lids and the globe to break down adhesions. Liquid paraffin, olive oil, or cod-liver oil drops may be instilled to prevent these adhesions forming, *but they should not be employed in the first aid treatment.*

If the lids and face are burned no coagulant such as tannic acid or aniline dyes should be applied within 2 in. of the lid margins, for contraction of the skin in this region may cause ectropion and the eye may be lost through the effects of exposure. Burns of the lids are most safely treated with simple gauze packs lightly laid on and constantly kept moist. If the burn is severe, involving much scarring and loss of tissue, and if the eye itself does not need much attention, the lids should be sewn together at the earliest possible stage, to protect the globe, and skin-grafting should be done at the first appearance of granulations. The first graft may be lost but can easily be replaced. If tarsorrhaphy is impracticable the globe should be temporarily protected by a contact glass.

68. Screening smokes.

Smoke may be used for screening important positions or the movement of troops ; it may also be employed to mask a gas cloud, or to extend its flanks so as to conceal its actual frontage. Such screening smokes may be generated from solids dispersed from shell or bombs, or from liquids sprayed from aircraft or land vehicles.

Screening smokes are irritating when inhaled in close proximity to their source, but they are not toxic in the concentrations that render them effective as screens ; under ordinary conditions troops can operate in them without wearing respirators, while in higher concentrations they may be irritant yet not produce toxic effects.

A dangerous and possibly asphyxiating concentration, however, may arise if a smoke shell burst at, or close to, the entrance of a dug-out, while proximity to a bursting phosphorus smoke shell may result in very severe burns from flying particles of burning phosphorus (*see* Section 72).

Apart from these possibilities, the chief danger associated with the use of screening smokes 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 cause severe ulceration or burns. With a view to preventing such accidents, operators should wear protective goggles or respirators, thick gloves, rubber boots, and special coats of oilskin or rubber.

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

69. 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 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 C.S.A. generates intense heat and acid may be scattered in all directions. Treatment follows the lines laid down in Sections 66 and 67.

70. Titanium tetrachloride (F.M.).

This is a yellow, non-inflammable and corrosive fluid which, on contact with damp air, gives off a heavy dense white cloud. This property is made use of by aircraft for the production of vertical smoke curtains extending down to the ground or sea level. The smoke consists 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 eye. The usual precautions must be taken when handling the liquid, and contamination of the eyes or of the skin should be treated as described above.

71. Stannic chloride (K.J.).

Stannic chloride is a fuming, straw-coloured, corrosive liquid which produces a heavy white cloud on contact with air, and is therefore sometimes utilized by aircraft for the production of vertical smoke curtains. The dangers attending the handling of it are similar to those associated with titanium tetrachloride, and the treatment is the same as for other corrosives.

72. Phosphorus.

At ordinary temperatures white phosphorus is a solid which can be handled safely in water but, when dried in air, burns fiercely with a dense white smoke. It may be used by an enemy as an incendiary or as a smoke filling in bombs or in shell, and flying fragments or melted particles of the burning substance may become embedded in the skin of persons close to the bursting missile. These fragments continue to burn unless flooded or smothered. Burning clothes may be ripped off and flames extinguished with water, blankets, sand or earth.

First-aid treatment should consist in immersion of the affected part in water, or, in the absence of enough water, in the application of a thick pad soaked in water. This purely temporary measure should be followed at the earliest opportunity by removal of all phosphorus from the skin, according to the following directions :—

(1) Bathe the affected part in sodium carbonate or bicarbonate solution (two tablespoonsful of washing soda or bicarbonate of soda to the pint of water) for a few minutes, to neutralize phosphoric acid, which is formed when phosphorus burns on the skin, and to allow of the removal from the skin of any visible phosphorus. Fragments otherwise unobserved may be seen in the dark.

(2) Immerse the injured part in a solution of copper sulphate (one heaped teaspoonful to a pint of water) for some seconds, after which all dark-coloured deposits which are formed by the action of the sulphate or any remaining phosphorus should be removed, with the aid of forceps, in clean water. If this removal is incomplete and free phosphorus is left, even in minute amounts, on the skin—detected by fuming and the typical smell of white phosphorus—these two stages should be repeated.

Sulphanilamide powder, as a paste in water or dry, is a good first-aid application. The treatment, after a final wash in weak Dettol or other antiseptic, can then be as for any other thermal burn.

Daily exposure of 1–1½ minutes' duration to ultra-violet light is of value in the treatment of a phosphorus burn from its inception ; more rapid healing is promoted, and the great pain always associated with skin lesions due to phosphorus is lessened.

If the eyes are affected, treatment should be as described in Section 67, except that, where practicable, initial lavage should be performed with a 1 per cent. solution of copper sulphate. The particles rendered visible by copper sulphate may be picked out after instillation of cocaine.

Symptoms of acute poisoning, such as restlessness, thirst, abdominal swelling and nasal discharge, may be shown by animals grazing on land contaminated by phosphorus bombs or shell. Post-mortem examination reveals fatty degeneration of the liver, and acute congestion, or even necrotic patches, in the kidneys and small intestine.

73. Thermite.

Thermite (aluminium and ferric oxide) is also used in incendiary bombs and in attacks on armoured fighting vehicles.

In the first-aid treatment of burns of the skin, the field or shell dressing may be thoroughly soaked with water and applied as a pad, keeping it moist. Armoured fighting vehicles are supplied with morphine (Tubunic ampoules), which should be given in all severe cases. As soon as possible any obvious particles of thermite should be removed under water. The burn should then be treated as an ordinary thermal burn.

74. Fuel oil and creosote.

Burns known to be caused by fuel oil or creosote mixtures, which are sometimes used for incendiary purposes, should also be treated as ordinary thermal burns. Contamination of the skin with unignited fuel oil, however, deserves special notice, since people with highly sensitive skins (e.g. many of those with fair hair) may be rapidly affected. For removing the oil, soft soap is preferable to powerful solvents such as benzol and carbon tetrachloride, neither of which

must be applied more than once to the same area of skin. Petrol is unsuitable. After cleaning, a bland ungent such as lanoline should be employed without a dressing. Hands soiled with fuel oil should never be allowed to come into contact with mucous membranes (conjunctiva, mouth, or nose).

CHAPTER VIII

DANGEROUS GASES NOT USED FOR OFFENSIVE PURPOSES, BUT ENCOUNTERED IN WAR

75. Carbon monoxide.

Carbon monoxide is a colourless, odourless non-irritant gas which cannot be recognized by the senses. It is formed wherever combustion of carbonaceous material is incomplete, and is therefore commonly met under normal conditions of everyday life. It burns with the characteristic blue flame so often seen flickering over a coke or smouldering coal fire.

Occurrence.—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 collieries.

In war, carbon monoxide may be met in dangerous quantities under the following conditions :—

(*a*) *Mining operations.*—Whenever a blasting charge is exploded, as in mines or camouflets, the resulting gases contain large quantities of carbon monoxide, which are liable to find their way to adjacent galleries, trenches or dug-outs ; moreover, pockets of gas may occur which may be tapped when new galleries are being driven in the vicinity. Carbon monoxide is also produced in large quantities when the timbering of mine galleries catches fire.

(*b*) *Heavy gun fire.*—A high-explosive shell penetrating the soil, and bursting in close proximity to confined living quarters such as dug-outs, may liberate sufficient carbon monoxide to poison the occupants.

(*c*) *Gun emplacements, pill-boxes and ships' turrets.*—Carbon monoxide gas may accumulate rapidly in gun emplacements, pill-boxes or turrets of ships, especially when firing into the wind, owing to the blow-back of muzzle gases or when the breech of the gun is opened ; in the case of ships' turrets, if the air-blast in the barrel be defective, high concentrations of the gas may accumulate within the turret.

(*d*) *Underwater explosions.*—At sea, following underwater explosions, large quantities of carbon monoxide may be trapped within the hull of the ship and may gain access to inner, inhabited compartments.

(*e*) *Interior of tanks.*—If ventilation be defective, dangerous concentrations of carbon monoxide may be found in tanks from leakage of engine exhaust gases and from blow-backs from the guns.

(*f*) *Burning buildings.*—Carbon monoxide gas is generated by the burning of material in closed spaces, owing to insufficiency of oxygen.

(*g*) *Coke and charcoal fires.*—Poisoning has often occurred through the use of coke or charcoal braziers or stoves in insufficiently ventilated billets.

(*h*) *Internal combustion engines.*—The exhaust gases of internal combustion engines contain from 1 to 10 per cent. or more of carbon monoxide ; the use of such engines in confined spaces without adequate safeguards may have serious results.

(*i*) *Fractured gas mains.*—The leakage of coal gas into closed spaces, such as billets, following fracture of a gas main (e.g. after a bombardment) may cause poisoning.

76. Mode of action of carbon monoxide.

Carbon monoxide owes its poisonous properties to the fact that it combines with hæmoglobin to form a dissociable compound, just as oxygen does, and that its affinity for the hæmoglobin is about 300 times that of oxygen. But for this property of combining with hæmoglobin, carbon monoxide would be a physiologically inert gas like nitrogen or hydrogen.

When air containing carbon monoxide is breathed, the relative amounts of oxygen and carbon monoxide present in the atmosphere determine the proportion in which carboxy-hæmoglobin is found in the blood. When the amount of oxygen is 300 times that of carbon monoxide, half of the hæmoglobin can combine with the carbon monoxide and half with the oxygen ; this is about the degree of blood saturation at which unconsciousness occurs.

As the concentration of the gas in the air rises, the saturation of the hæmoglobin with carbon monoxide increases, and the oxygen-carrying capacity of the blood progressively diminishes until symptoms of anoxæmia (oxygen want, in this instance without cyanosis) make their appearance. In this sense the gas is cumulative in 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 symptoms of oxygen want.

The consequences are due to anoxæmia alone. There are no pathological changes in the lungs such as follow the action 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 hæmoglobin reaches about 70 to 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.

77. Symptoms of carbon monoxide poisoning.

The great danger in carbon monoxide poisoning is the risk that it may not be suspected until too late. The first symptoms may be a loss of power in the limbs, which makes escape 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 is gradual and insidious. The first symptoms may be 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 prevent 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 be left in the poisoned atmosphere.

The colour of the face may vary with the rapidity of the onset or the degree of anoxæmia. 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 a vivid carmine.

Individual susceptibility to the gas varies, and experience has shown that acute or chronic alcoholism, and cardiac or respiratory disorders, accentuate the severity of carbon monoxide poisoning.

Recovery from the initial symptoms may be followed by mental confusion and slow cerebration, while headaches, often of a severe or migrainous type, are characteristic. Among other after-effects may be mentioned cardio-vascular disorders, especially tachycardia and dyspnœa which may continue for months, and a predisposition to pneumonia. Disturbances of the central nervous system range from a simple neuritis to paresis and even mental derangement, usually of temporary duration, but sometimes the result of damage to the brain by protracted anoxæmia.

78. Protection against carbon monoxide.

The respirator does not afford any protection against carbon monoxide. If it is necessary to enter an atmosphere in which the gas is present or suspected, it is essential that a special carbon monoxide respirator * be used (e.g. mask with filter consisting of catalysts which oxidize the carbon monoxide to the dioxide) or some form of self-contained oxygen-breathing apparatus such as the Proto or Salvus set, or, in the case of H.M. ships, the Davis S.E. apparatus, which is more familiar to the personnel.

In the absence of special oxygen sets, a useful apparatus can be readily extemporized by means of an ordinary respirator facepiece to which is attached a suitable length of non-collapsible tubing of 1·5-inch diameter, the far end of which is left out in the open. Such an apparatus can only be worn for short periods, owing to the absence of an inlet valve and the rapid accumulation of carbon dioxide in the tubing ; the insertion of a valve, however, at the inlet end of the facepiece valve holder will enable a man to remain in a contaminated atmosphere almost indefinitely. Such an apparatus (with special valve) is already standardized in H.M. Navy, and is known as B.A. Pattern 230.

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

79. Test for the detection of carbon monoxide in blood.

The simplest method of detecting carbon monoxide in the blood is as follows : Dilute a sample of the suspected blood in a test tube (of internal diameter of about 1·5 cm.) to 0·5 per cent. with distilled water to which a trace of ammonia has been added (e.g. 0·1 c.cm. blood diluted to 20 c.cm.) ; this will give a perfectly clear solution. Dilute similarly a sample of normal blood.

Compare the tints of the two test tubes by transmitted daylight, when it will be seen that the tint of the suspected blood, if carbon monoxide be present, is definitely more pink than the yellowish-red of the dilute normal blood.

This test is purely qualitative ; the actual saturation in any suspected specimen can be determined either by the carmine method or by means of the reversion spectroscope.

80. Treatment of carbon monoxide poisoning.

The majority of cases of carbon monoxide poisoning recover with prompt treatment, although relapse, or even sudden death, is possible later. Uncon-

* Carbon monoxide respirators must be used with great care since oxygen deficiency is generally associated with the presence of carbon monoxide, and these respirators give no protection against this danger.

sciousness may last 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, which may have to be continued for many hours.

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 B.L.B. or Haldane (*see* Appendix II), which may be set to deliver 8 to 10 litres a minute, or one of the types which allow the administration of a mixture of carbon dioxide with either oxygen or 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 c.cm. of 25 per cent. Coramine solution is useful, while blood transfusion has been found of great value in desperate cases. Venesection is valueless.

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

81. 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 of a mixture of oxides of nitrogen.

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.

Occurrence.—Under war conditions, nitrous gases may be met with in mining or tunnelling operations when detonation of the blasting charge is incomplete, in gun pits, armoured cars, and tanks, and in magazines of ships when propellant charges are set on fire. 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.

Although no serious cases of poisoning by these gases were recorded on land in the Great War, this may have been partly due to the fact that when nitrous fumes are formed in large quantities in mining operations, carbon monoxide, with its more rapid action, is also generated in lethal proportions. Another possible factor was the moist condition of the surroundings, which may have helped to reduce the concentration of nitrous gases.

At sea, the virulence of the nitrous gases was well illustrated in the Great War by the death-rolls which followed the sinking of H.M. Ships " Russell " and " Britannia," when a large proportion of the officers and men who had been exposed to the fumes of burning cordite succumbed to their effects.

82. Mode of action of nitrous fumes.

The action of nitrous fumes on the lungs closely resembles that of phosgene. They are particularly dangerous because they do not produce much sensory irritation, and men may therefore fail to realize 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, with inflammation and œdema. This usually overshadows the systemic effect of the alkaline nitrites, which, however, contribute to the clinical picture by their enfeebling action on the circulation. Through the formation of methæmoglobin, they may also diminish the oxygen-carrying capacity of the blood.

With the 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 may vary from 2 to 24 hours or more, according to the conditions of exposure, its usual duration being between 10 and 24 hours. 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.

83. Symptoms of poisoning by nitrous fumes.

The symptoms on exposure are slight irritation of the eyes, nose and throat, accompanied perhaps by a little cough—symptoms which are seldom at all conspicuous 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 associated with a profuse mucopurulent 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 œdema which may be hæmorrhagic in character. Restlessness is extreme, and in fatal cases consciousness is retained almost to the end, the patient struggling vainly for breath while, with bloodstained fluid trickling from his mouth and nostrils, he drowns slowly in the fluid exuded in his lungs.

84. Protection against nitrous fumes.

The ordinary charcoal respirator affords limited protection against nitrous fumes, but none against the carbon monoxide which is nearly always generated simultaneously. It is essential, therefore, that an oxygen or air breathing apparatus (such as the Proto, Salvus, D.S.E.A. or the Naval B.A. Pattern 230) be worn in such atmospheres whenever possible, and reliance should not be placed on a respirator save in 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.

85. Treatment of poisoning by nitrous fumes.

Treatment follows the same lines as those outlined for phosgene poisoning, stress being laid on complete physical rest from the time of exposure and on administration of oxygen as soon as cyanosis develops.

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

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

86. Ammonia.

Ammonia, which is widely used for industrial purposes, is dangerous in high concentration. In the present war 47 persons were admitted to hospital with ammonia poisoning after a bomb had hit a brewery cellar.* The mild cases (9) showed catarrh and slight œdema of the upper respiratory passages, with hoarseness and conjunctivitis ; they recovered rapidly. In the 27 " moderate " cases these signs were exaggerated, with more or less shock, moist sounds in the chest, dyspnœa, dysphagia, severe cough, and blood-stained tenacious sputum ; broncho-pneumonia developed in 9 and corneal ulceration in 2. The 11 most severe cases showed pulmonary œdema, and the total number of deaths was 13.

Treatment should combat shock. Morphia and oxygen are useful. Locally, a mouth-wash of vinegar (1 part) and water (4 parts) can be given, and the throat sprayed with cocaine and adrenaline. The eyes are washed out with saline, and liquid paraffin and atropine alkaloids are instilled.

The protection afforded by the Service respirator is considerable. In its absence the nose and mouth may be guarded with a cloth soaked in water.

87. Dangers associated with fire fighting.

(1) *Noxious products of combustion.*

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 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, at 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 10 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

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* Caplin, M., *Lancet*, 1941, 2, 95.

respirator cannot compensate for oxygen deficiency, it is essential that, when fighting fires where a free dilution of the atmosphere or a free escape for noxious gases is not possible, an oxygen or air breathing apparatus should be worn.

(2) *Chemical fire extinguishers.*

The following are two types in common use, sold under various trade names :—

(*a*) *Carbon tetrachloride.*—This is a volatile liquid, boiling at 76·7° C. (170° 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 greater 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 with a dangerous emergency, to leave a burning room. In these circumstances a very real danger arises from the continued inhalation of vaporized carbon tetrachloride or its products.

Recent experience has shown that exposure to the fumes of carbon tetrachloride 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 idiosyncrasy 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, varying from a trivial rise in blood pressure to an acute uræmia. Evidence of liver damage may also be seen in the jaundice, the slow pulse, the abdominal pain and hæmorrhage from stomach and bowel so characteristic of the toxic jaundice caused by the organic halogens.

(*b*) *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. Owing to its rapid vaporization 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, epileptiform seizures and paralysis, both motor and sensory.

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

CHAPTER IX

ANOXÆMIA

88. 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 the oxygen percentage in the atmosphere may be reduced. Apart from high-altitude flying or climbing (where the deficiency is of partial pressure and not of composition, though the effect is the same), oxygen deficiency may be met with :—

(*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 it with residual nitrogen welling out of the surrounding strata.

(*b*) In air-tight compartments such as double-bottoms and " blisters " 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 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 oxidizable or oxygen-absorbing substances, such as grain, fruit, potatoes or coal, are stored.

89. Symptoms of anoxæmia.

In healthy adults, the percentage of oxygen in the air breathed must be reduced by about a third before any symptoms, such as hurried breathing, 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 anoxæmia from whatever cause it may arise, namely : headache, visual disturbance and mental dullness, loss of muscular power and of co-ordination; dyspnœa and weakened cardiac action ; the power of judgment may be seriously impaired, while loss of memory is common. With extreme reduction in oxygen acute anoxæmia results in immediate loss of consciousness.

90. Treatment of anoxæmia.

Immediate removal to fresh air is imperative, and, if the breathing has stopped, artificial respiration should be started at once. This should be supplemented, if possible, by the administration of oxygen (*see* Appendix II) ; 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.

91. Protection against oxygen deficiency.

The precautions may be summarized as follows :—

(*a*) Test the suspected atmosphere by means of a lighted candle or oil lamp, **after excluding the possible presence of explosive or combustible gases.** A flame is much more sensitive than the human body 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 possibly 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, the other end of the line being held by observers outside.

(*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, such as the Proto, or the Salvus, or the Naval Breathing Apparatus, Pattern 230, or a valved air pipe drawing its supply from pure atmospheric air at a distance.

CHAPTER X

PROTECTION AGAINST GAS AND AIR RAIDS

92. Individual protection.

The most important item in the protection against vapour is the respirator, which is an article of personal equipment.

The hygiene of the respirator is a unit responsibility, but medical officers should bear in mind that periodical cleansing or disinfection is necessary and they should satisfy themselves that these procedures are being carried out efficiently and as laid down by the regulations.

Cleansing is required immediately after wear and at least once a month, whilst disinfection is authorised: (1) at least twice yearly in units; (2) whenever the facepiece changes ownership; (3) before facepieces are despatched to, or after their receipt from, store; and (4) during epidemics, when the facepieces of all personnel suffering from the disease and those in contact with them (as ordered by the medical officer) will be treated.

Respirators of personnel suffering from pulmonary tuberculosis or any other infectious disease must be destroyed if the medical officer considers this advisable.

93. Collective protection.

The respirator is the first line of defence against gas. Where, however, its use might interfere with the efficiency of the personnel—e.g. in control rooms, telephone exchanges, and transmitting stations in H.M. ships—it is necessary to render the compartment or shelter gas-proof.

Gas-proof shelters may also be required to avoid the necessity of wearing respirators for long periods and to enable men to take food and rest.

In estimating the number of persons that can be accommodated in a protective shelter without serious discomfort or risk to health, the temperature and humidity prevailing in the shelter, as well as the content of oxygen and carbon dioxide in the air, must be taken into account.

A normal man when at rest will give out heat amounting to about 100 kilocalories (400 B.Th.U.) * per hour, and will during the same period produce about 0·6 cubic feet of carbon dioxide and absorb about 0·75 cubic feet of oxygen, but these figures may be substantially increased by restlessness or bodily movement. Heat is lost from the body by radiation and convection and by the evaporation of moisture (moisture in the expired air, evaporation of sweat from the surface of the body).

As the temperature of the environment increases, a smaller proportion of

* B.Th.U.=amount of heat required to raise 1 lb. of water 1° Fahrenheit.

the heat produced by the body will be lost by radiation and convection and a larger proportion by evaporation. In warm air which already has a high moisture content the rate of evaporation will be slowed down and the condition may be such that it is no longer possible to lose heat from the body as fast as it is formed. Under such circumstances the body temperature will begin to rise. The wet-bulb thermometer (i.e. a thermometer whose bulb is covered by wet muslin so that it is subject to cooling by evaporation) will show by comparison with the reading of the ordinary dry-bulb thermometer the extent to which an object can be cooled under the particular circumstances merely by the evaporation of water from the surface, and it has been found that in still air the body temperature can no longer be kept constant when the wet-bulb temperature exceeds 88–89° F., no matter whether the dry-bulb thermometer shows the same temperature (air completely saturated with moisture so that no evaporation can occur) or a higher temperature (air incompletely saturated with moisture). The wet-bulb temperature can, therefore, be used as an index to show when temperature conditions are becoming intolerable, and in a crowded shelter a wet-bulb temperature of 80° F. should be regarded in practice as the figure that must not be exceeded in temperate climates.

In an *unventilated shelter* the heat produced by the occupants can only be dissipated by conduction through the walls, roof and floor, and the rate of dissipation will be governed by the temperature of the walls and their surroundings, being high, for instance, in a deep shelter whose walls are in close contact with the surrounding cool soil. It is clearly inadvisable to choose as a shelter a room in the vicinity of boilers, steam pipes or other sources of heat. The moisture given off by the occupants will be in part condensed on, and possibly absorbed by, the walls if these are cool, and the remainder will stay as vapour in the air, thus increasing the humidity and causing a rise of wet-bulb temperature. The carbon dioxide concentration will rise and the oxygen concentration will fall at a rate determined by the number of occupants, and it is inadvisable in a crowded shelter that the carbon dioxide concentration in the air should exceed 2 per cent. by volume for any length of time.

In a *ventilated shelter* the dissipation of heat by conduction through the walls, roof and floor is still material. Owing to the low specific heat of air no great quantity of heat will be removed from the shelter by warming up the air current, unless this is very large, but more will be lost as the latent heat of evaporation by the removal of some of the water vapour given off by the occupants. The removal of this water vapour will also help to keep the wet-bulb temperature, the importance of which has been emphasized above, at a reasonably low figure. At the same time the ventilation will compensate for the increase of carbon dioxide and deficiency of oxygen that would otherwise occur.

The permissible number of occupants is, therefore, much less in an unventilated than in a ventilated room. In a warm room, whether ventilated or unventilated, discomfort can be greatly reduced by promoting free movement of the air, e.g. by means of electric fans.

On the above basis the number of permissible occupants for protective shelters of different types and sizes is discussed below.

(*a*) *Accommodation in unventilated shelters.*—For periods of occupation up to three hours under normal English summer conditions an allowance of 75 square feet surface area (floor, ceiling and walls), 160 cubic feet of air space and 6 square feet of floor space per person is necessary.

For periods above three hours the allowance should be not less than 100 square feet per person.

The following figures may be taken as a guide :—

Dimensions of room	No. of persons permitted up to 3 hours
10 feet by 10 feet by 8 feet high	7
15 feet by 10 feet by 8 feet high	9
20 feet by 15 feet by 10 feet high ..	17
30 feet by 15 feet by 12 feet high ..	26
50 feet by 20 feet by 15 feet high ..	55

(*b*) *Accommodation in ventilated shelters.*—The following table gives the allowance per person which must be made for varying rates of ventilation.

Period of occupation	Shelters above ground		Shelters underground	
	Total surface area per person in square feet	Ventilation per person in cubic feet per hour	Total surface area per person ih square feet	Ventilation per person in cubic feet per hour
Up to 3 hours.	30	450	20	480
	40	150	20	150
Indefinite.	50	450	25	450

To ascertain the number of persons who can be accommodated in any shelter, add the surface area of the floor, ceiling and four walls together, and divide by the minimum surface area per person shown in the table above : e.g. the total surface area of a room 20 by 10 by 8 ft. high is 880 sq. ft. :—

	Square feet
Floor and ceiling surface=twice 20 by 10	400
Two walls 10 by 8 =twice 10 by 8	160
Two walls 20 by 8 =twice 20 by 8	320
Total surface area	880

It will be seen that for a shelter above ground of this size, with a rate of ventilation per person of 450 cubic feet per hour, 29 persons may be accommodated for three hours and 17 persons for an indefinite period.

It follows of course that where the number of occupants of a compartment is greater than that laid down, the period of occupation of an unventilated shelter must be reduced and the air flow in a ventilated shelter must be increased. The same remarks also apply to shelters in the tropics and it is for this reason that an allowance of 40 cubic feet of air a minute for each man is provided in H.M. ships which may have to operate in every climate.

94. Cleansing centres.

A Gas Cleansing Centre, when available, is intended for cleansing unwounded personnel contaminated either by a liquid vesicant or by exposure to its vapour. Such a centre is a unit responsibility and is staffed by personnel of the unit.

Casualties who are both wounded and contaminated by a vesicant will be treated at a First Aid Post where they will be cleansed if this has not already been done. In addition, any casualty showing actual symptoms of gas poisoning, whatever the type, will be treated at a First Aid Post.

CHAPTER XI

RECOGNITION AND EARLY TREATMENT OF GAS CASUALTIES

A suitable introduction to this chapter is the following quotation from the "Official History of the War—Medical Services," Vol. II (1923), page 317; it refers to the period of the battle of Loos (25th–27th September, 1915) when chlorine 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 poisoning 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."

In this chapter an attempt is made to indicate and deal with the difficulties of medical officers 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 prevent 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 lacrimatory or sensory irritant gases, it is absolutely essential that all medical officers should be familiar with the symptoms of gas poisoning.

It is also of the first importance that, when gas is used by the enemy, the fullest and earliest information possible be given by the military authorities to medical units, not only to facilitate the correct diagnosis of individual cases, but also to permit of arrangements for their reception being made on an adequate scale.

Further, in order that men only temporarily distressed by a harmless gas should not be sent unnecessarily to medical units, it is equally important that all officers commanding troops in the field should be familiar with the various types of war gases.

95. History of the case.

The patient will generally be able to give a definite history of exposure to gas, and to describe the smell of the gas and his initial and subsequent symptoms. If he is unconscious a history of his exposure may sometimes be obtained from others. In both cases the medical officer's questions should be directed on the following lines:—

(1) Under what conditions did exposure to the gas take place? (e.g. position in the field, whether exercising actively or at rest, whether in the open or in a dug-out, etc.).
(2) What did the gas smell like?
(3) For how long (minutes or hours) was the gas breathed?
(4) What effects did it produce at the time?
(5) Did symptoms persist after adjustment of the gas-mask?
(6) What symptoms appeared later?
(7) What period of time has elapsed since gassing—hours or days?

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

96. Mustard gas vapour.

The characteristic symptoms are: conjunctivitis, laryngitis, and skin burning, but these only develop after some hours.

Should a respirator have been worn during exposure, reddening and even vesication of the skin in the more sensitive regions may have occurred without damage to the eyes or respiratory tract.

The immediate treatment of such cases is the removal of the clothing which is impregnated with the vapour of mustard gas.

97. Arsenical vesicant vapours.

Lewisite and other arsenical vapours irritate the respiratory tract, and personnel will probably adjust their respirators before the eyes or lungs are damaged. High concentrations will cause skin burning. The eyes or skin will require treatment with an anti-arsenical agent.

The clothing of persons exposed to lewisite vapour will, unlike clothing worn in mustard gas vapour, generally have a strong unpleasant smell. Clothing smelling of lewisite should be removed as early as possible.

98. Vesicant liquids.

The Medical Services are not usually responsible for the recognition of vesicant liquids nor for the cleansing of contaminated personnel unless they have become casualties. Contamination may vary from a few drops, which may readily escape notice until skin burns appear, to a thorough drenching from a successful low-altitude spray attack, or from a bursting bomb.

The preventive treatment necessary for such cases of gross contamination should have been carried out in the unit immediately after contamination, viz. removal of the clothing and application of ointment, anti-gas, to the areas of skin affected, followed if possible by washing with water. Anti-arsenical agents should be used in the treatment of casualties due to arsenical vesicants.

99. Lung irritant gases.

Sometimes it is not easy for the medical officer to decide whether or not a man has really been gassed. The delayed action of the lung irritants introduces another factor of uncertainty.

In doubtful cases the patient's own account is of great importance, for he may describe the choking character of the gas, the cough and tightness of the chest, and there may have been definite objective symptoms such as vomiting. He may have been unduly exhausted by trying to walk to the aid post, and have had to be helped down; evidence in support of this may possibly be furnished by the pallor of his face and by a rapid pulse. Examination should be made to see if there is any lacrimation, any sign of cough, or unduly short and rapid breathing. It should be ascertained whether he can take a deep breath without discomfort or coughing.

The benefit of the doubt must be given to the patient, but if no objective symptoms have arisen within 48 hours the gassing must have been very slight, and he can be returned to duty with little delay.

All gas cases must be evacuated as soon as possible to the point at which arrangements have been made for effective treatment. Rest and warmth are essential. Whilst waiting for evacuation care should be taken that the men do not undertake any undue muscular exertion. All except the slightest cases should, as far as possible, be evacuated lying down, and walking cases should be given every assistance; they should not carry their own equipment. If any walking case shows increasing symptoms (breathlessness, palpitation, loss of power in the limbs, or a feeling of being "done in") arrangements should at once be made to carry him.

Should pulmonary œdema develop, with accompanying cyanosis and frothy expectoration, the essential treatment is oxygen administration and, in suitable cases, venesection.

100. Paralysant gases.

Hydrocyanic acid gas and hydrogen sulphide gas in high concentration paralyse the respiratory centre, quickly producing unconsciousness and failure of respiration. In weak concentration they are not harmless, but in comparison with other gases their action is mild.

The recognition of casualties caused by these gases presents no difficulties; a history of exposure to one or other of them, followed by unconsciousness and cessation of breathing, with or without convulsions, will be sufficient. Treatment, to be effective, must be prompt; it consists in immediate artificial respiration, combined with the administration of oxygen and carbon dioxide if available.

101. Arseniuretted hydrogen (arsine).

The outstanding symptoms are: shivering, nausea, vomiting, headache, renal and perhaps hepatic pain, hæmoglobinuria, weakness and at a later stage jaundice and anæmia. The gas exerts no irritant action on the eyes or respiratory tract.

Treatment in the early stages will involve measures to promote diuresis, and, for severe anæmia due to the hæmolysis, blood transfusion.

102. Lacrimatory gases.

These are easily recognizable by the *immediate* and severe lacrimation and the spasm of the eyelids that they produce.

As met with under war conditions they are otherwise relatively harmless, and personnel against whom they are used 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 treatment of eyes so affected should consist in copious and immediate lavage, followed by such other symptomatic treatment as may be called for. The condition is seldom serious and clears up quickly, in sharp contrast with that resulting from eye contamination by drops of a liquid vesicant.

103. Nose irritant gases.

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; coughing, 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. The treatment consists of a brief rest coupled with the assurance that recovery will be rapid and complete.

104. Carbon monoxide.

The recognition by a medical officer of cases of carbon monoxide poisoning in war time should not be difficult if he be familiar with the possible sources of such poisoning (as detailed in Chapter VIII) and if he can obtain a history.

In the milder cases the symptoms are headache, giddiness, breathlessness on exertion, weakness of the legs and inco-ordination of movement, mental

irritability or confusion. In the severer cases unconsciousness will be profound. A blood examination may help to decide the diagnosis.

The immediate treatment should be—removal to fresh air, the administration of oxygen containing 5–7 per cent. of carbon dioxide (if the apparatus is available), and artificial respiration if the breathing shows signs of failure. Care should be taken to keep the patient at rest and warm, and to prevent him from accidentally injuring himself whilst mentally confused.

105. Nitrous fumes.

Poisoning by these gases has an insidious onset. The incomplete detonation of a charge in a mining gallery or the breathing of vapour arising from the spilling of nitric acid on a wooden floor may evoke a little coughing which soon subsides; hours later, however, œdema 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 the case 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 œdema should develop its treatment follows closely the lines laid down for phosgene gassing.

106. Corrosives and incendiaries.

Phosphorus is solid, and burning fragments on the skin or clothing must be put out at once, preferably by immersion in water or envelopment in cloths soaked in water. If available apply copper sulphate solution (1 or 2 per cent.).

The first aid treatment for personnel splashed by smoke-producing compounds or other corrosives is the same as that required for persons contaminated with any acid, namely, the rapid removal of the soiled clothing and the flooding of contaminated skin areas with water.

APPENDIX I

SUMMARY OF GASES, PROPERTIES, METHODS OF RECOGNITION AND FIRST AID TREATMENT

Type	Properties	Methods of recognition	Effects	First aid treatment
Choking Gases				
Phosgene (C.G.)	Almost invisible gas. Corrodes metals. Rain renders less effective. Non-persistent.	Smell of musty hay. A trace of phosgene in the air makes smoking unpalatable.	Highly lethal. First effects —cough and lacrimation ; pain in chest, dyspnœa ; later pulmonary œdema. Effects appear in 0 to 24 hours.	Complete rest essential. Warmth.
Chlorine	Greenish coloured gas. Corrodes metals. Repeated exposure rots clothing. Non-persistent.	Smell similar to bleaching powder.	Like phosgene but more irritant and less toxic.	Ditto.
Diphosgene	Colourless liquid, invisible gas. Semi-persistent.	Resembles phosgene.	Like phosgene.	Ditto.
Chloropicrin (P.S.)	A colourless volatile liquid. Semi-persistent.	Pungent odour.	Like phosgene but more irritant and lacrimatory and less toxic, though more toxic than chlorine. Cumulative.	Ditto.
Nasal Irritant Gases				
Diphenylchlorarsine (D.A.) Diphenylamine - chlorarsine (D.M.). Diphenylcyanarsine (D.C.).	Colourless or bright yellow crystals. When heated give off odourless smoke, invisible except near source. Non-persistent.	By onset of effects.	Produce sneezing, burning and aching pain in chest, throat, nose, gums. Later mental depression. First effects 0 to 5 minutes.	On removal to an atmosphere free from the gas recovery is usually rapid without treatment, but effects may increase for the first few minutes in fresh air.

Tear Gases				
Chloracetophenone (C.A.P.).	Colourless crystals. Invisible in gaseous state. Non-persistent.	Onset of, effects; smell of 'Ronuk' or cheap scented soap.	Stinging of eyes. Immediate and profuse lacrimation. Blepharospasm. Skin irritation of exposed areas.	Recovery usually rapid without treatment.
Ethyliodoacetate (K.S.K.).	Dark brown liquid. Invisible in gaseous state. Persistent.	Onset of effects; fruity smell.	Ditto. Irritates nasal mucosa but not skin.	Ditto.
Bromobenzyl cyanide (B.B.C.).	Pure—yellowish brown crystals. As used—brown liquid. Persistent.	Onset of effects; pungent penetrating smell.	Ditto.	Ditto.
Blister Gases				
Mustard gas (H.)	An oily liquid varying in colour from dark brown to straw yellow. Soluble in oil, fats and organic solvents. Penetrates rubber, leather, wood, cloth, etc. Very persistent. Neutralized by chlorine (e.g. bleaching powder).	Smells of garlic, horseradish, onions, or mustard.	(i) *Vapour effects*— *Eyes.*—Inflammation with swelling within 24 hours. High concentration causes tears earlier. *Skin.*—Redness, irritation, blisters. Damp parts specially affected. *Respiratory passages.*—Aphonia and cough. Later, bronchitis and possibly pneumonia. (ii) *Liquid effects*— *Eyes.*—No immediate sensation. Gradual onset of severe symptoms in a few hours. *Skin.*—Erythema in 2 hours, blister in 12 to 24 hours.	1. Remove clothing. 2. Wash patient with soap and water, if possible. 1. *Eyes.*—Immediate flushing out of eyes with any available water. 2. *Skin.*—Ointment, A/G No. 2 or No. 3, bleach paste or solvent. 3. *On clothes.*—Remove clothing, apply ointment or bleach paste to affected skin. Wash skin with soap and water or use solvents.*

* Cleansing materials stated in order of preference, but if skin is already reddened solvents are preferable.

SUMMARY OF GASES, ETC.—*continued*

Type	Properties	Methods of recognition	Effects	First aid treatment
Lewisite	A colourless liquid when pure ; brown in the crude state. Gives off an invisible gas. Is rapidly destroyed by water and any alkali. Great power of penetration. Very persistent but less so than mustard gas.	Smell of geraniums.	(i). *Vapour.*—Causes irritation to nose. Hence respirator likely to be adjusted immediately so that there will be no permanent effects on eyes, nose or lungs. Is less effective on skin than mustard gas vapour. (ii) *Liquid*— (*a*) In eyes ; immediate and grave effect. (*b*) On skin ; blisters develop more rapidly than with mustard gas.	Apply an anti-arsenical agent to eyes or skin. Hyperol or H_2O_2 is useful for skin. Water more effective with lewisite than with mustard gas. Blisters should be opened antiseptically and drained.
Ethyldichlorarsine ..	Clear oily liquid rapidly destroyed by water. Great power of penetration. Not very persistent.	Nasal irritation may make fruity smell unrecognizable.	1. Nasal irritation immediate. 2. Coughing, choking and later bronchitis. 3. Eye and skin effects like lewisite but less severe.	1. As for nasal irritants. 2. As for phosgene. 3. As for lewisite.
Paralysant Gases				
Hydrocyanic acid	A colourless volatile liquid. Non-persistent.	Smell of bitter almonds.	Small amounts of vapour cause giddiness and headache—larger doses cause unconsciousness and death.	*Urgent.*—Artificial respiration. Oxygen inhalations, or better oxygen containing 5–7 per cent. carbon dioxide.
Hydrogen sulphide	A colourless gas. Non-persistent.	Smell of bad eggs.	Small doses cause irritation of eyes and nose and bronchitis ; large doses cause unconsciousness and death.	*Urgent.*—Ditto.

3* *Arseniuretted hydrogen* Arsine	A colourless, odourless gas. Non-persistent.	Special test papers.	Weakness, headache, vomiting, hæmoglobinuria, jaundice, anæmia.	Require hospital treatment.
Other Gases				
Carbon monoxide	A colourless, odourless gas. Non-persistent.	Practically no warning before onset of symptoms.	Causes giddiness, muscular weakness, then unconsciousness and death.	*Urgent.*—Artificial respiration. Oxygen inhalations, or better oxygen containing 5–7 per cent. carbon dioxide.
Nitrous fumes	Reddish brown fumes. Non-persistent.	Pungent smell.	Irritation of nose, throat and lungs; later pulmonary œdema.	Similar to phosgene.

APPENDIX II

ADMINISTRATION OF OXYGEN

In the treatment of certain pulmonary conditions *accompanied by cyanosis or blueness of the face*, and also in carbon monoxide poisoning (*see* page 51), administration of oxygen may be necessary for long periods. The aim should be to relieve the cyanosis, and there is no advantage in increasing the flow of oxygen beyond what is necessary to do this. Whilst providing the optimum concentration for the patient, economy in the expenditure of oxygen must be exercised, particularly in forward areas, or elsewhere where supply may be difficult. It is desirable, therefore, that some efficient method of administration should be employed, and useless or wasteful methods, such as the glass funnel, avoided.

Several types of apparatus are available. The following features are common to them all :—

Oxygen is supplied from cylinders in which the gas is compressed. The cylinders are fitted with—1. A master tap. 2. A pressure gauge to indicate how much gas is present in the cylinder. 3. A reducing valve, to reduce the pressure to manageable proportions and allow it to remain constant until the cylinder is almost empty. In some apparatus a by-pass to this valve is an additional fitting. 4. A fine-adjustment tap which may be graduated to show the rate of delivery of oxygen in litres a minute. As an alternative a bobbin flow meter may be used for determining the rate of delivery.

B.L.B. Apparatus.

This apparatus, described by Boothby, Lovelace, and Bulbulian, consists of a mask and a connecting device which joins the mask to a reservoir breathing bag (Fig. 1).

The mask.—Two types of interchangeable rubber masks have been designed : (i) a nasal type which leaves the mouth free, and (ii) an oro-nasal type which meets the requirements of cases in which adequate breathing through the nose is impossible. All normal adults can be fitted with one or other of the two sizes, small and large, of each of these two types. The nasal mask is provided with two hollow tubes which pass on each side of the mouth and unite over the lower part of the chin into a single tube. These tubes are in open communication with the rubber bag at all times through the connecting device. The mask is held in position on the face with an adjustable rubber strap, which is placed above or below the ears according to the comfort of the patient. Slight adjustments to the shape of the mask may be made by bending the malleable strips. A small hole is provided at the base of the nasal mask for the insertion, when required, of a gastric or a duodenal tube.

The connecting device.—On the side of the metal connecting device (Fig. 2) is an expiratory valve with a spring under slight tension, which permits the escape of any excess of expired air above that just sufficient to distend the bag, without causing any appreciable resistance to expiration. On the other side of the connecting device is an inlet connected with the oxygen cylinder ; the oxygen enters through this inlet and is delivered through a tube into the lower part of the rubber bag. The upper part of the connecting device is provided with an air-regulating mechanism which consists of three small holes

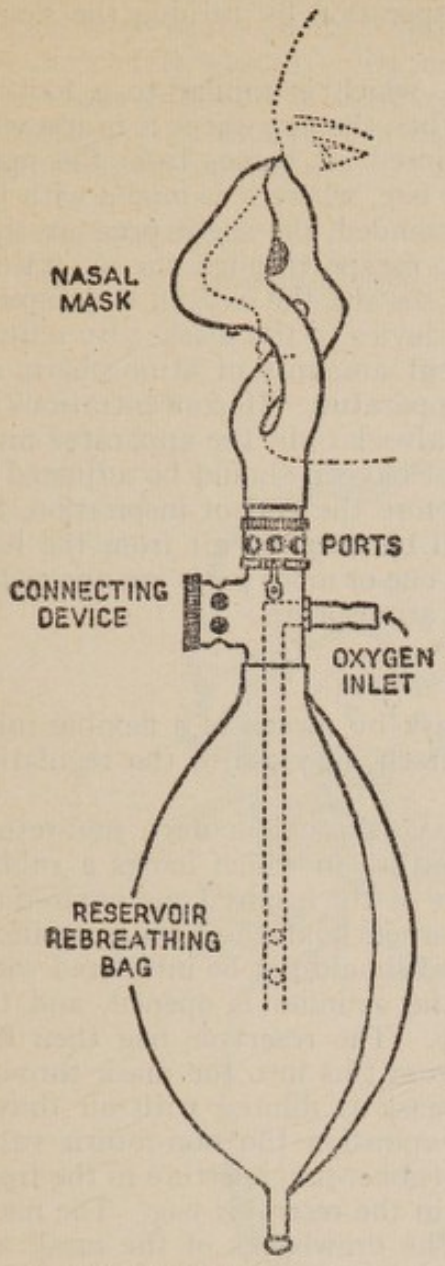

FIG. 1.—The B.L.B. inhalation apparatus.

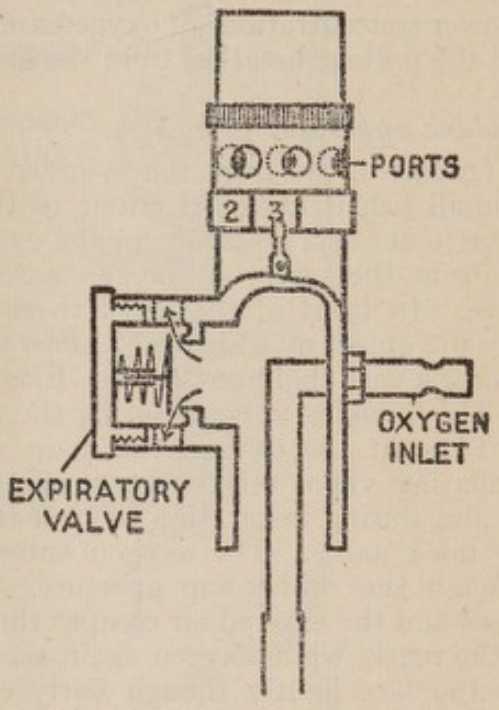

FIG. 2.—The connecting device.

Table to show effect of alterations in flow of oxygen and of opening or shutting ports upon concentration of oxygen in alveolar air.

Flow of Oxygen (litres per min.)	Ports open	Oxygen in alveolar air (%)	Carbon dioxide in bag (%)	Flow of Oxygen (litres per min.)	Ports open	Oxygen in alveolar air (%)	Carbon dioxide in bag (%)
3	3	46	2·04	6	0	87	1·42
4	3	56	1·51	7	0	90	0·99
5	2	69	1·22	8	0	91	0·39
6	2	76	0·89				

For details *see Oxygen Administration ; Indications, Methods and Types of Apparatus.* Ministry of Health Emergency Medical Service Memorandum No. 5. London (H.M. Stationery Office), 1940.

or ports, over which is mounted a rotating sleeve with three similar ports, one or more of which may be brought into operation by turning the sleeve. If desired, all the ports may be closed.

Reservoir breathing bag.—The rubber bag, which is similar to a football bladder, has a capacity of about 700 c.cm. When the apparatus is in use with all the ports closed, the first part of the expired air passes from the mask through the connecting device into the rubber bag, where it is mixed with the incoming oxygen. When the bag becomes distended, the slight pressure then produced permits the excess of expired air to escape through the expiratory valve. On inspiration the expiratory valve closes and the oxygen and expired air pass from the bag through the connecting device to the mask. By adjustment of the air-regulating mechanism different amounts of atmospheric air are mixed with oxygen in the inhalation apparatus. If concentrations of 80–90 per cent. of oxygen are required in the alveolar air, the apparatus must be used with all the ports closed. The flow of oxygen should be adjusted so that the bag does not collapse completely before the end of inspiration, for under these conditions the patient obtains all his inspired air from the bag. If lower concentrations of oxygen are required, one or more ports are uncovered and the patient breathes from the bag and the atmosphere.

Haldane apparatus (Fig. 3).

Oxygen is led from the cylinder to the mask by means of a flexible tube. A small bag is attached either to the mask itself, or close to the regulating valve to act as a reservoir for the oxygen.

From the reservoir oxygen enters the mask through a mica non-return valve. In front of the mask there is an aperture in which hangs a rubber flap, the object of which is to allow the passage of the inspired and expired air against a very slight resistance. Elastic head harness holds the mask in position.

The reducing valve is set by the makers and should not be interfered with.

To start the oxygen the main valve of the cylinder is opened, and the regulating valve set to the required delivery. The reservoir bag then fills up and during inspiration oxygen is drawn from this into the mask through the mica valve. The oxygen entering the mask is diluted with air drawn through the rubber-flap aperture. During expiration the non-return valve closes and the expired air escapes through the rubber-flap aperture in the front of the mask, while oxygen again accumulates in the reservoir bag. The mask fits the face lightly though fairly closely. The drawbacks of the mask are obvious. A casualty suffering from embarrassed breathing dislikes the idea of having anything put over his nose and mouth which may still further hinder his breathing.

Very often the patient's objection to the mask can be overcome by gaining his confidence. It is worth while spending a few minutes trying to do this, holding the mask a short distance away from the face and gradually bringing it up into position. In this connection there is a practical point worth noting. When the mask is first applied the patient is apt to take a deep breath. If he encounters any undue resistance his discomfort will be increased, and he will then probably refuse to give the mask a further trial. To overcome this difficulty the reservoir bag should always be completely filled with oxygen before the mask is applied. The initial gasp will give relief and the patient will be encouraged to proceed.

To avoid waste the minimal current of oxygen should be used ; the actual amount depends entirely on the individual case, and must be gauged by the disappearance of the cyanosis and the improvement of the pulse. The general condition of the patient and the colour of the lips are the best guide to the length of time and the amount of the flow.

Haldane's apparatus has the advantage that the oxygen flow can be readily gauged and regulated, and that, in addition, what flows during expiration is not completely wasted. The importance of cutting down waste is vital where continuous administration of oxygen over many hours, or even several days, is needed. The transport or handling of heavy oxygen cylinders ought to be reduced to the minimum.

Novox apparatus.

The Novox apparatus used in the Naval Service is also suitable.

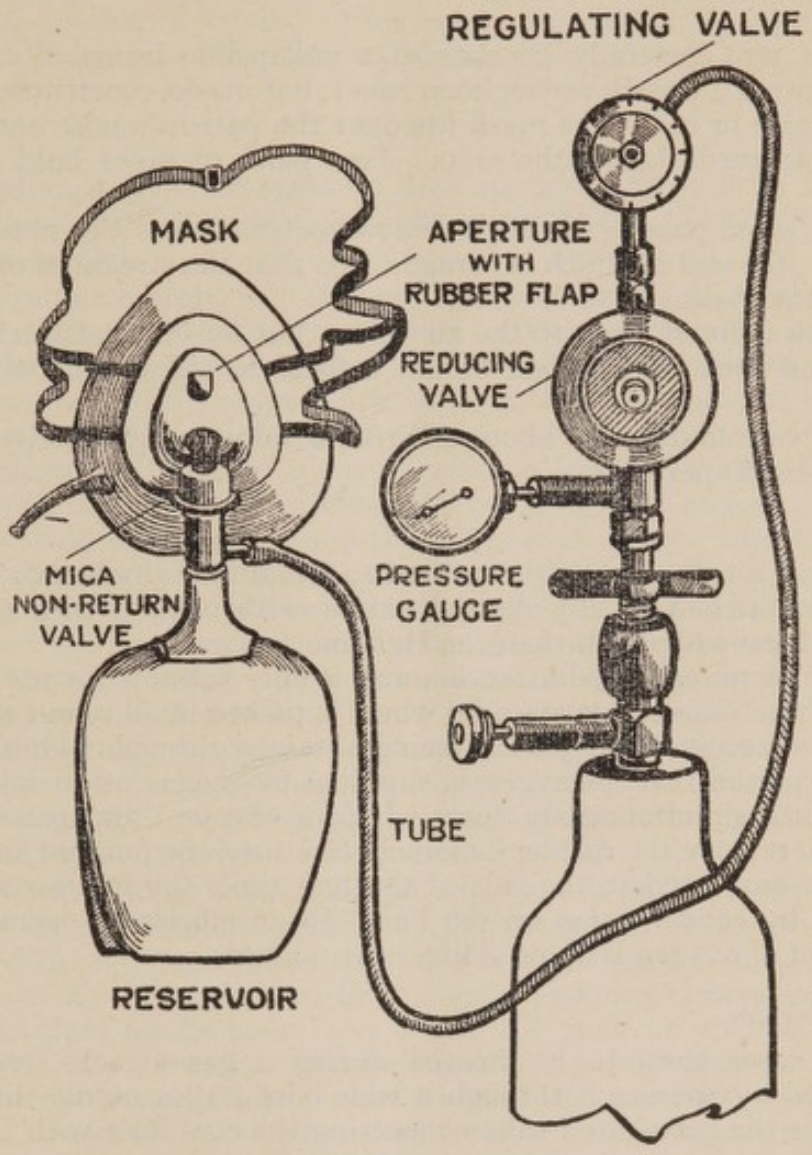

FIG. 3.—Haldane apparatus.

Nasal catheters and forked nasal tube.

Oxygen can be administered by means of a catheter passed up the nose, a Jacques rubber No. 9 being suitable. To be effective it is necessary for the tip to be passed up the nostril for a distance of approximately one and a hal inches.

A more satisfactory method whereby the oxygen is delivered through two catheters attached to a forked tube is also employed.

In this method the oxygen is conveyed in a rubber tube from the cylinder, fitted with the usual gauge, etc., to a metal tube fixed in a vertical position by means of a brow-band on the front of the patient's head.

The lower end of this tube, which is bifurcated, turns up opposite the nares. Two rubber catheters are attached to these ends, and pass into the nostrils. To get the best results a clear airway is essential.

An alternative form of catheter carrier has been described by Tudor Edwards.* In this type the carrier is incorporated in a spectacle frame, and in a modified form the device can be clipped on to the normal spectacle frames if these are already worn.

Oxygen consumption with the forked tube is about twice as great as with the Haldane apparatus.

Face tent.

The face tent generally consists of a collapsible frame, 6×4×3 inches, on which is fitted a closely woven linen cover, but masks constructed of cellulose acetate are also in use. The mask fits over the patient's nose and mouth and projects downwards below the chin. Two pairs of tapes hold the mask in position.

Oxygen, after passing through a flow meter, enters the mask through a rubber tube, the end of which is turned up so that the stream of oxygen strikes the roof of the mask.

The mask is freely open to the air below, but for efficient working must fit closely round the nose; a pad of lint suffices to secure this without undue pressure.

Oxygen consumption is about 6 litres a minute for an alveolar oxygen pressure of 40–50 per cent.

Oxygen tent.

The use of a tent permits the patient to remain for any length of time in an atmosphere enriched to any desired extent with oxygen with much greater comfort and less restriction than the Haldane apparatus.

The tent requires skilled attention and is only suitable for use in hospitals. It is made from impervious material which is tucked in all round the bed under the mattress, access to the patient being obtained through wide sleeves in the upper part of the tent. Oxygen is supplied by means of an injector on the cylinder which simultaneously draws air from the tent and passes it through soda lime to remove the carbon dioxide and returns the purified air to the tent. A metal ice-box fitted to the side of the tent cools the interior and moisture is removed by condensation on the box. In an efficiently operated tent the consumption of oxygen is about 3 litres per minute.

Service respirator.

Should cases have to be treated *during* a gas attack, oxygen can be administered by passing it through a wide-bore serum needle thrust into the lower end of the corrugated tube connecting the container with the facepiece.

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* *Lancet*, 1938, **2**, 680.

APPENDIX III

GAS CONTAMINATION OF FOOD AND WATER

The unit gas officer, in consultation with the medical officer, passes judgment on suspected food. The medical officer decides whether water is fit for use. Briefly: (1) In solid or liquid form *all* war gases make food or water unsafe. (2) In vapour form mustard gas makes fatty foods unsafe, while other vesicants, and arsenical smokes in high concentration, may poison any food or water. (3) In vapour or smoke other gases do not make food or water dangerous, but may make them unpalatable.

Food

Contamination may be revealed by dark spots or splashes, by smell, or by detectors. The Case, Water-testing, Poisons may help. But often evidence is only circumstantial.

Liquid.—Complete protection is given by sealed cans and stoppered bottles and glazed jars, good protection by corked bottles and barrels, and fair protection by lined three-ply tea-chests. Soft wood and cardboard are rapidly penetrated, but food well wrapped in waxed paper, Cellophane, or even greaseproof paper is usually safe if quickly and carefully removed. Salt-packed bacon may sometimes be saved if speedily taken from its wooden case, and so may dried fruit, which should afterwards be aired and cooked. Food contaminated by vesicants other than mustard gas must not be used till analysed. Meat, bacon, fish and bread in bales contaminated by mustard gas are also dangerous, but may if necessary be consumed after free removal of contaminated parts, airing for 24 hours, and, where applicable, prolonged boiling. (Roasting and frying are no safeguard.) Fatty foods are risky, but the rate of penetration of mustard gas into butter is less than ½ inch in 24 hours. Following slight contamination by mustard gas peas, beans, fresh fruit and vegetables, and eggs, all of which are relatively non-absorbent, are usable after hosing, airing and cooking. Sacks of flour may be soaked for an hour and dried; the flour inside the crust can then be tipped out and aired.

Vapour.—Unprotected foodstuffs exposed to vesicant vapours other than mustard gas, or to arsenical smokes in high concentration, are dangerous. Those exposed to high concentrations of mustard gas vapour should be treated as if sprayed by liquid; but after light mustard gas contamination meat and bacon may be aired for 24 hours and boiled, fish may be washed and boiled, while bread can be eaten when its smell becomes normal after airing. Fatty foods exposed to mustard vapour can be used after careful removal of the whole surface to a depth of 1 inch. Milk exposed to any vapour needs at least 15 minutes' boiling. Other food exposed to non-persistent gases should be aired for 24 hours: if then palatable it can safely be eaten. (N.B. This applies only to non-persistent gases, excluding concentrated arsenical smokes.) The taint of phosgene in tea or flour can be masked by mixing with fresh stocks.

Water

Contamination by mustard gas and lewisite is dealt with in " Water Supplies in the Field " (p. 43) which describes tests for poisons (Amendment No. 1, p. 3). Note that neither water-truck filters nor chlorination remove mustard gas. Any arsenical may render small quantities of water dangerous (Section 64). *See also* Section 36.

APPENDIX IV

PLATES

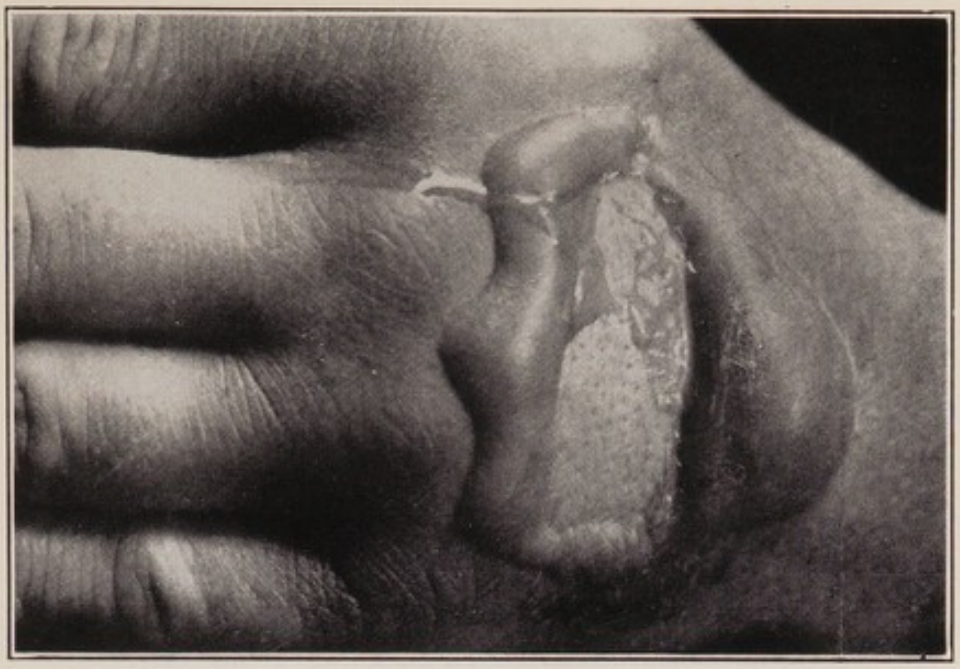
The following photographs and drawings illustrate the effects of poisoning by phosgene and mustard gas. Plate VII is taken from a paper by Dr. Ida Mann, F.R.C.S., and Dr. B. D. Pullinger in the *Proceedings of the Royal Society of Medicine* for January, 1942 (vol. **35,** No. 3), and is reproduced by courtesy of the Honorary Editors of that journal. The other coloured plates formed part of the " Atlas of Gas Poisoning," originally produced in 1918 by the Medical Research Committee, and republished in 1937 and 1938 by arrangement with the Medical Research Council.

Plate I.—Effects of Liquid Mustard Gas on the Skin

These photographs show the results of wearing, for three hours, a leather glove whose back had been accidentally contaminated with mustard gas.

Photos 1 and 2 were taken a day after wear. Photo 3 was taken a week later.

Plate I.



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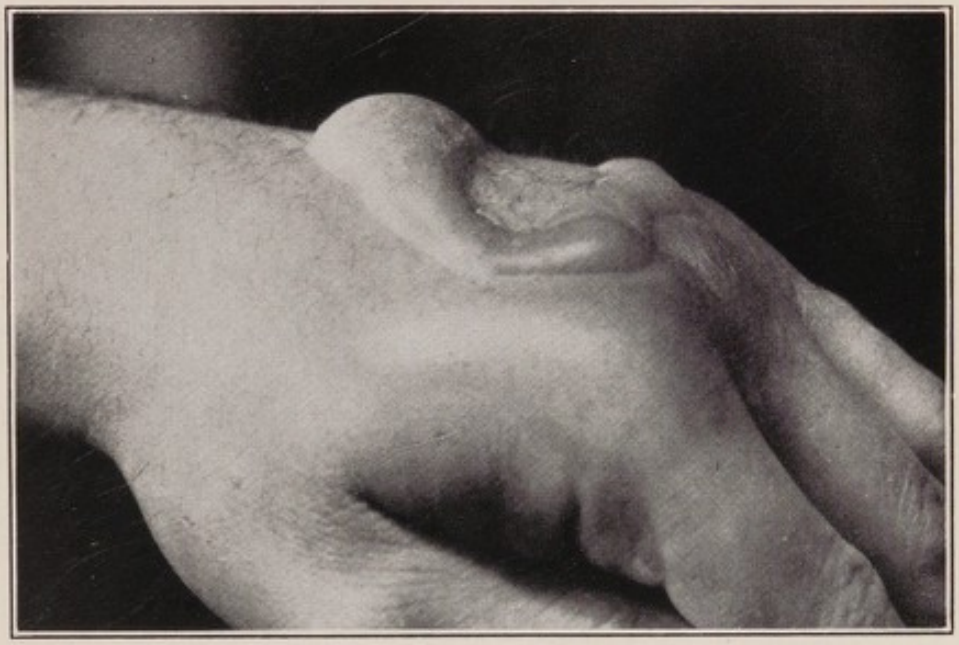

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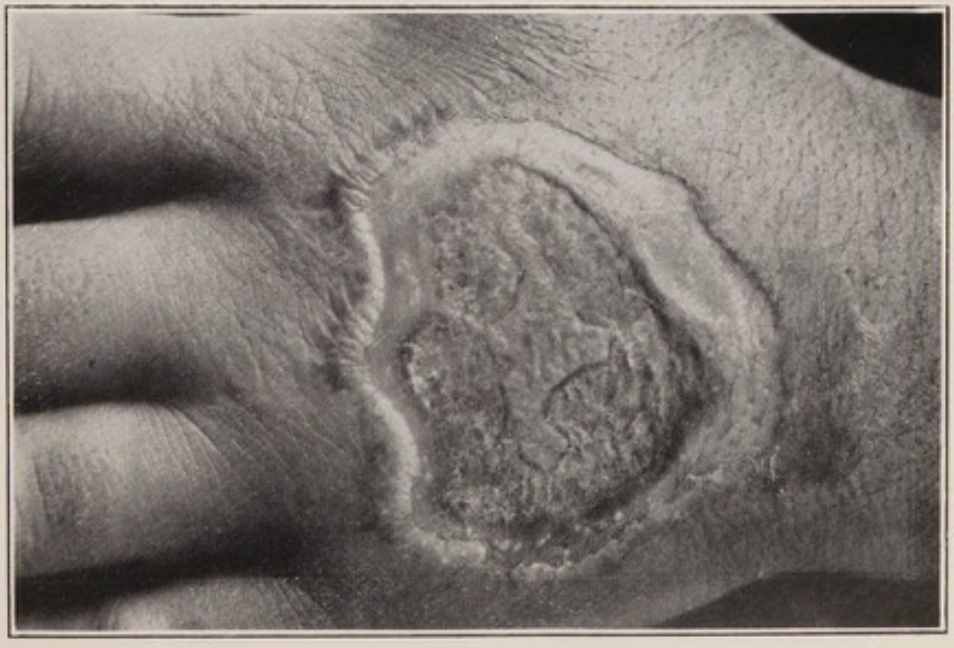

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PLATE II.

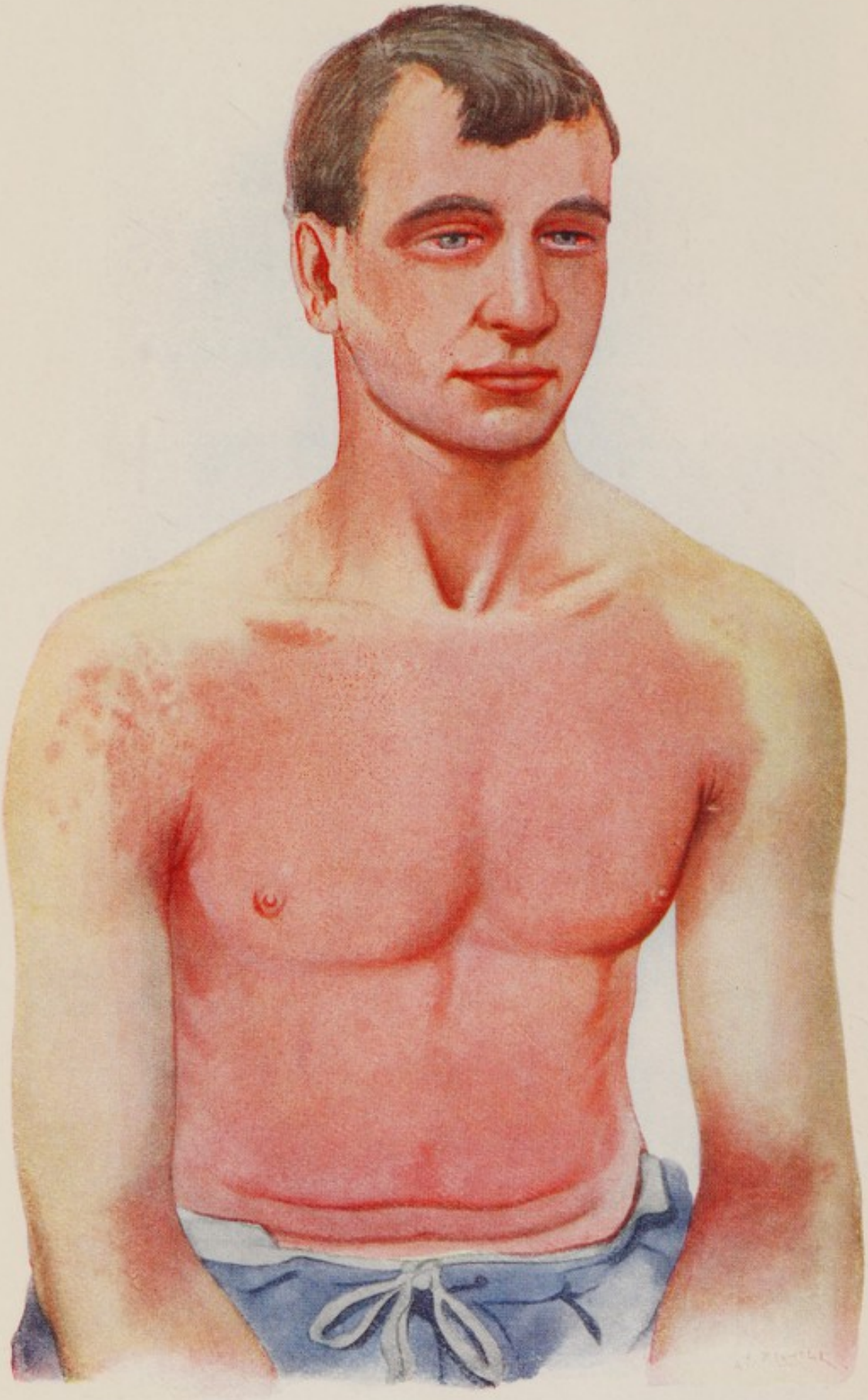

Plate II.—Erythema of Skin from General Exposure to Mustard Gas Vapour

Dermatitis of this distribution, associated with conjunctivitis, forms a picture characteristic of poisoning by this vesicant.

History of the case. Exposed to mustard gas at Ypres on the 12th July, 1917, when this substance was first employed by the enemy. Wore box respirator for only 30 minutes, so that he was exposed without any protection for nearly four hours. No symptoms were felt until some hours later, when severe vomiting commenced and conjunctivitis developed.

Drawing made on the fifth day. The laryngitis and bronchitis were slight, so that the poisonous vapour must have acted only in low concentration. But the reddening of the skin was fairly intense because the man had been sweating freely when exposed to the gas, and he was not washed afterwards nor was his clothing changed. The erythema was succeeded by staining in the same areas of the skin.

This reddening, as though the skin had been scorched or deeply sunburned, is the first cutaneous reaction to mustard gas, though it sometimes may not appear until several days after exposure. It is accompanied by only a slight feeling of warmth, but the irritation is frequently severe. In addition to the face and arms, which are directly exposed to the vapour in the air, the moist surfaces of the axillæ, the flexures of the elbows, and the perineum and inner surfaces of the thighs are particularly affected—that is, the places where the skin is often sodden with fatty perspiration. This special distribution of the diffuse erythema characterizes the general dermatitis of mustard gas vapour, but the reaction may be limited to a smaller area in any part of the body—for example, where the clothing may have chanced to be splashed by the liquid.

This inflammatory reaction is chiefly superficial, and is not accompanied by much œdema of the subcutaneous tissues except in the eyelids and over the penis and scrotum. Later the dusky-red colour deepens, and patches of cyanotic or whitish œdema may arise amid it. Blisters then appear, and the cuticle becomes excoriated; or the skin may be retained while the erythema fades and a brown staining slowly darkens the original area of irritation.

Plate III.—Blistering of Buttocks by Mustard Gas

History of the case. The man sat down on ground that was contaminated by the gas and the vapour passed through his clothing, causing inflammation of the buttocks and scrotum. A diffuse reddening appeared twenty-four hours later, and this was followed by an outcrop of superficial blisters. On the eighth day the erythema began to be replaced by a brown staining. The drawing was made on the eleventh day during this change of tints. Infection of the raw surface was avoided, and the healing was complete in three weeks.

The blisters in this case were probably aggravated by pressure, for inflamed skin becomes very fragile, so that the surface layer is readily loosened by pressure or rubbing. The blisters may be very tiny bullæ, as on the eyelids, or they may coalesce into areas many inches across, covering a collection of serous fluid which perhaps contains enough of some irritant to injure adjacent skin if it is allowed to flow over and remain on it.

The blisters are usually quite superficial and almost painless in their development. But the raw surface that is left after the blister has burst becomes most acutely sensitive to all forms of mechanical irritation. Deeper destruction of the dermis may be caused by spreading necrosis where the substance attacks the skin locally in high concentration, or when secondary infections are implanted on the raw surface. Chronic and painful sores then result, and in this event the skin does not regenerate completely, so that thinly covered scars for a long time will mark the site of the burn.

The perineum is peculiarly liable to become inflamed after exposure to the vapour of mustard gas, and the external genitalia become œdematous as well as reddened. Balanitis and pain on micturition may be troublesome. When the skin is excoriated, secondary infection of the raw surface is very likely to develop unless adequate precautions are taken to prevent sepsis. But with careful cleansing of the skin, and protection from contact with dirty clothes after exposure to the vapour, inflammation of the perineum can be reduced to a comparatively trifling incidence.

A purplish-brown, brown or brownish-black tint usually appears in areas that were first inflamed and red, but it may arise without such preceding erythema. Its distribution is in the same areas as those in which erythema occurs, such as over the exposed skin surfaces of the neck and hands, or on the sheltered, moist flexures of the body. It may appear any time after the fifth or sixth day, and will persist for several weeks until the stained cuticle desquamates. There is no deep pigmentation.

PLATE III.

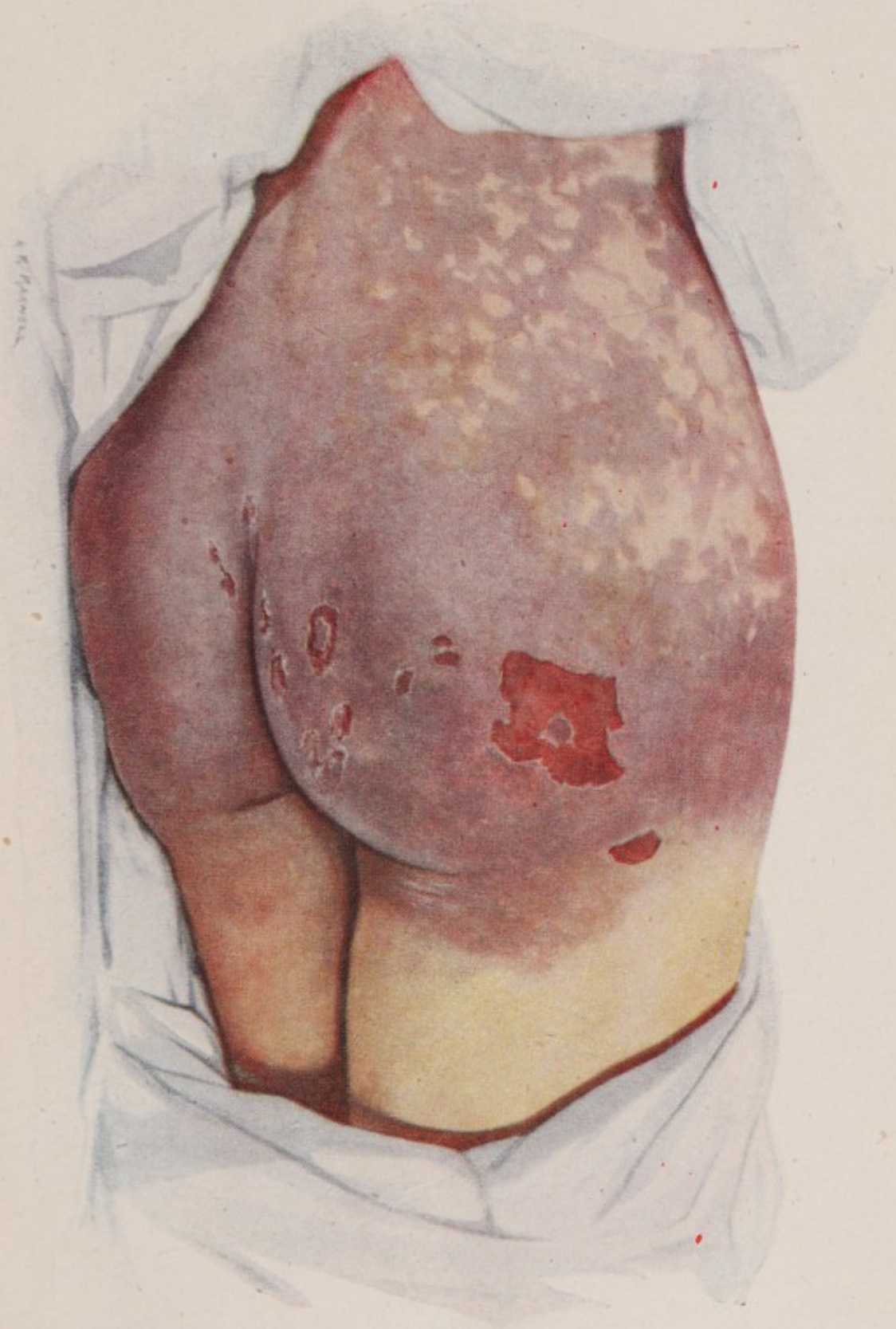

PLATE IV.

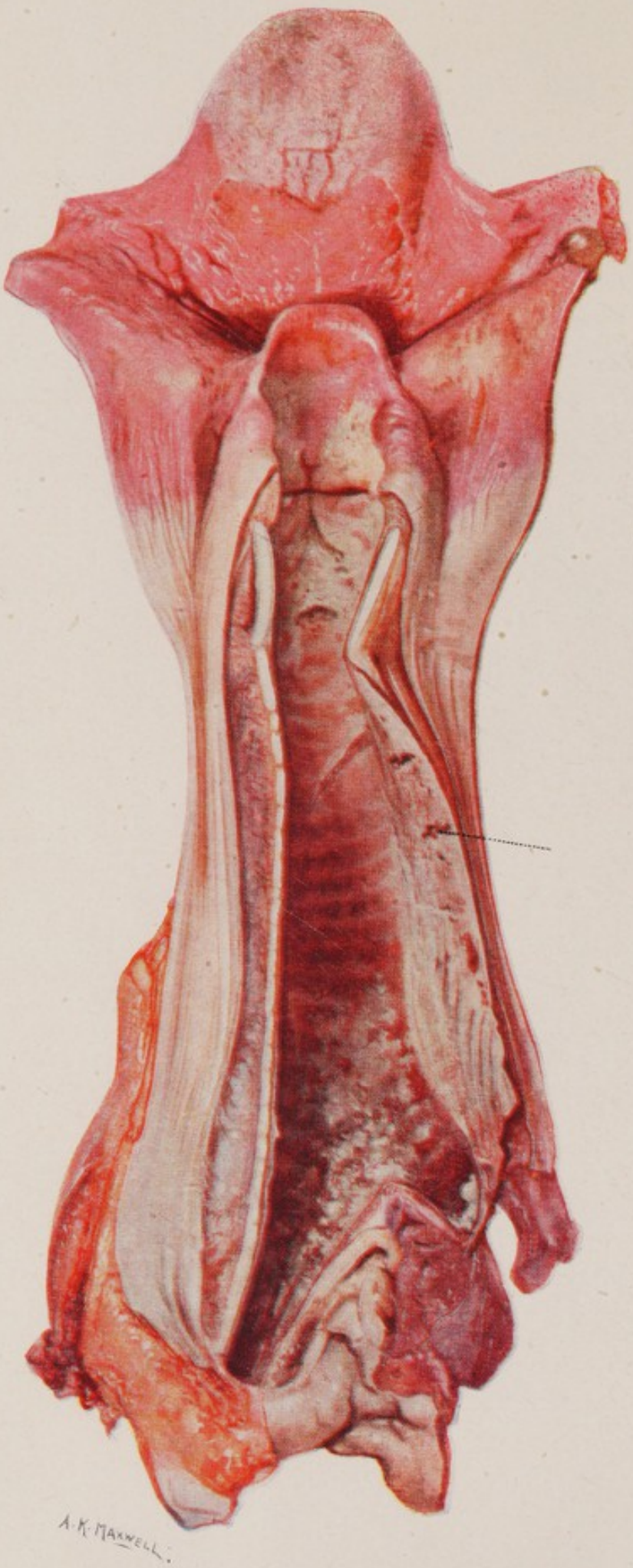

Plate IV.—Ulceration of the Trachea by Mustard Gas.

The characteristic feature is the sloughing of the tracheal mucous membrane. The reddening of the base of the tongue, and the pharynx, with a sharp delimitation where the œsophagus has refused ingress to the toxic vapour, is also seen after poisoning by chlorine and the other lung irritant gases. But the pharyngeal inflammation with mustard gas may proceed further to a local ulceration that will cause dysphagia for many days.

The mucous membrane of the trachea and bronchi is affected by mustard gas in much the same way as the skin. It reacts with an intense inflammation, and death of the surface layers soon results. The mass of necrotic tissue, exuded fibrin, and pus cells may form a yellowish-grey slough in which secondary infections can flourish. Subsequently this false membrane comes away in patches or in entire casts from the raw surface of the bronchial wall.

Meantime the infected debris and secretions tend to accumulate in the bronchial ramifications at the bases of the lungs, and infection may spread from them into the lung alveoli and tissues. Septic broncho-pneumonia, localized abscesses, superficial pleurisy, and even empyema or pyopneumothorax then develop and cause death.

The drawing is of a trachea on the twelfth day after gassing. The base of the tongue, and the pharynx, show characteristic inflammation. Yellow necrotic sloughs lie on the larynx and at the bifurcation of the trachea. Between these the trachea is red and glistening, because it is now completely denuded of both mucous membrane and slough. The dotted line points to a little group of ulcers on the posterior wall from which bleeding had occurred. The trachea and bronchi contained an abundance of thin yellow pus.

Plate V.—Microscopic Section of Human Lung after Mustard Gas Poisoning

Death occurred at the end of the second day after exposure (i.e. in 40 hours).

The bronchiole is filled with fibrin and pus cells, and its lining epithelium has been completely destroyed. The inflammation has caused a characteristic ring of hæmorrhage in the tissues around the bronchial tube, and infection is beginning to appear in the alveoli nearest to these inflamed tissues. But there is no generalized pulmonary œdema and no disruptive emphysema.

Mustard gas may cause some catarrhal desquamation of the pulmonary endothelial cells, but it rarely excites an outpouring of œdema fluid from the pulmonary vessels. The pathological changes in the bronchioles and in the alveoli are therefore in the sharpest contrast with those caused by phosgene (*see* Plate VIII). As infection spreads into the lung tissues, patches of septic broncho-pneumonia and small abscesses develop, and these often evoke an inflammatory œdema around them.

If the patient lives, his bronchial mucous membrane is slowly regenerated ; and during this time he is naturally subject to reflex spasms of coughing or even to a protracted bronchitis.

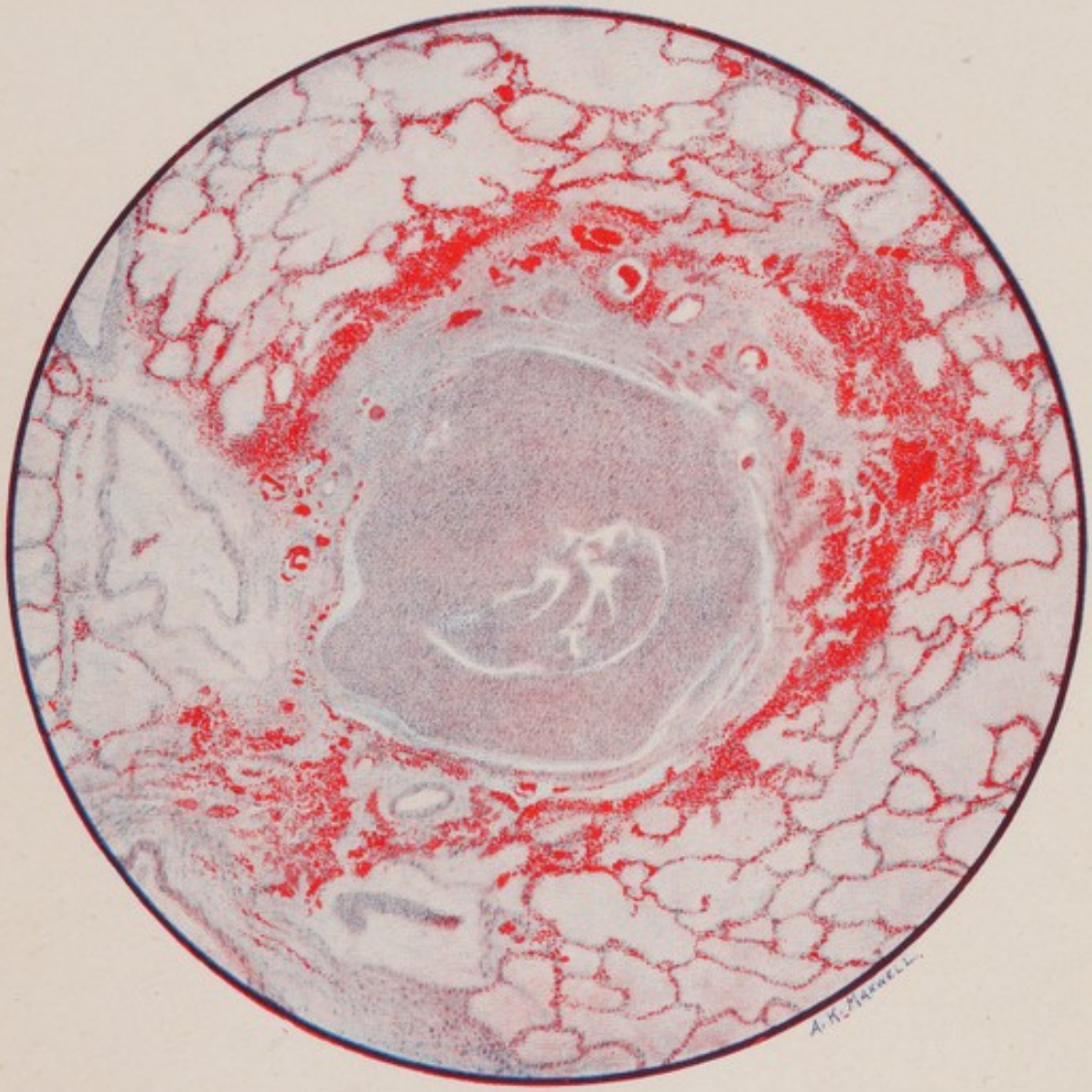

PLATE VI.

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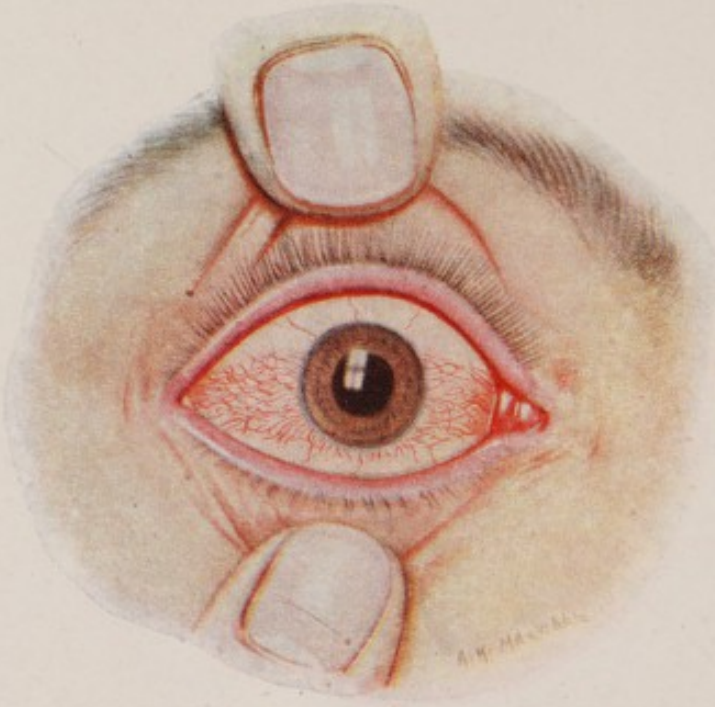

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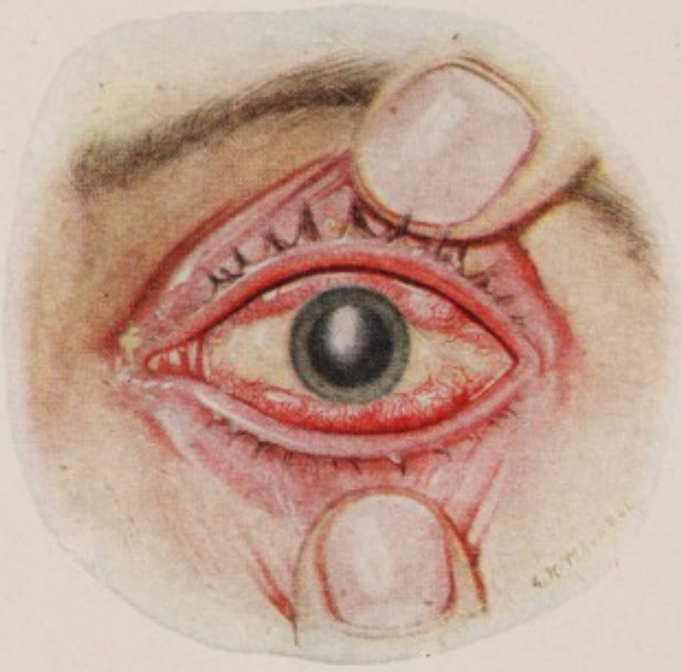


Plate VIA.—Mustard Gas Vapour : Mild Case

Early stages of a slight burn following exposure to mustard gas vapour. Though discomfort may prevent the patient from opening his eyes, the only lesion is injection of the conjunctiva.

Plate VIB.—Severely Burned Eye in the Acute Stage

Relatively early after exposure to mustard gas vapour the eyelids and the external surface of the globe show an intense inflammatory reaction. Tears stream from between the closed œdematous eyelids, which may even be blistered, and there is often severe pain behind the eyes and in the forehead. The conjunctiva is swollen, œdematous, and bright red from injection of the blood vessels. The injury to the cornea, even when severe, is not so obvious, and careful examination is of great importance for its detection. Photophobia and blepharospasm render examination of the eye difficult.

Whenever a dead white band crosses the exposed area of the conjunctiva, while the parts of this membrane covered by the upper and lower lids are red and œdematous, serious injury from the burning may be presumed.

In the case illustrated, the caustic effect of the vapour is seen chiefly in the interpalpebral aperture. On each side of the cornea there is a dead white band due to coagulative necrosis. The swelling in the region of this white band is slight, while the protected conjunctiva above and below it is greatly swollen and injected and may even bulge between the lids.

The exposed portion of the cornea is grey and hazy ; it has lost its lustre, and when viewed with a bright light and a magnifying glass it shows a blurred " window reflex " and a typical " orange-skinned " surface. The haze gradually fades off above in the region of the protected part of the cornea where the surface is usually bright and smooth. The pupil is at first contracted as the result of irritation and congestion. In this drawing it is shown as artificially dilated by atropine. A mydriatic should always be used early in severe cases or where there is pain and blepharospasm.

Plate VII.—Effects of Mustard Gas on the Eye

Fig. 1. Severe lesion 3 weeks after exposure to mustard gas vapour, showing a small patch of hyperæmia in the palpebral conjunctiva (A), a triangular white necrotic patch at the inner side of the limbus (B), and a slight corneal haze in the palpebral aperture (C).

Fig. 2. Severe lesion after exposure to a spray of liquid mustard gas, showing subconjunctival hæmorrhages, dilated thrombosed vessels and partial destruction of limbal capillaries in exposed areas which were formerly necrotic and opaque ; and in the cornea, epithelial bedewing with œdema of the substantia propria below.

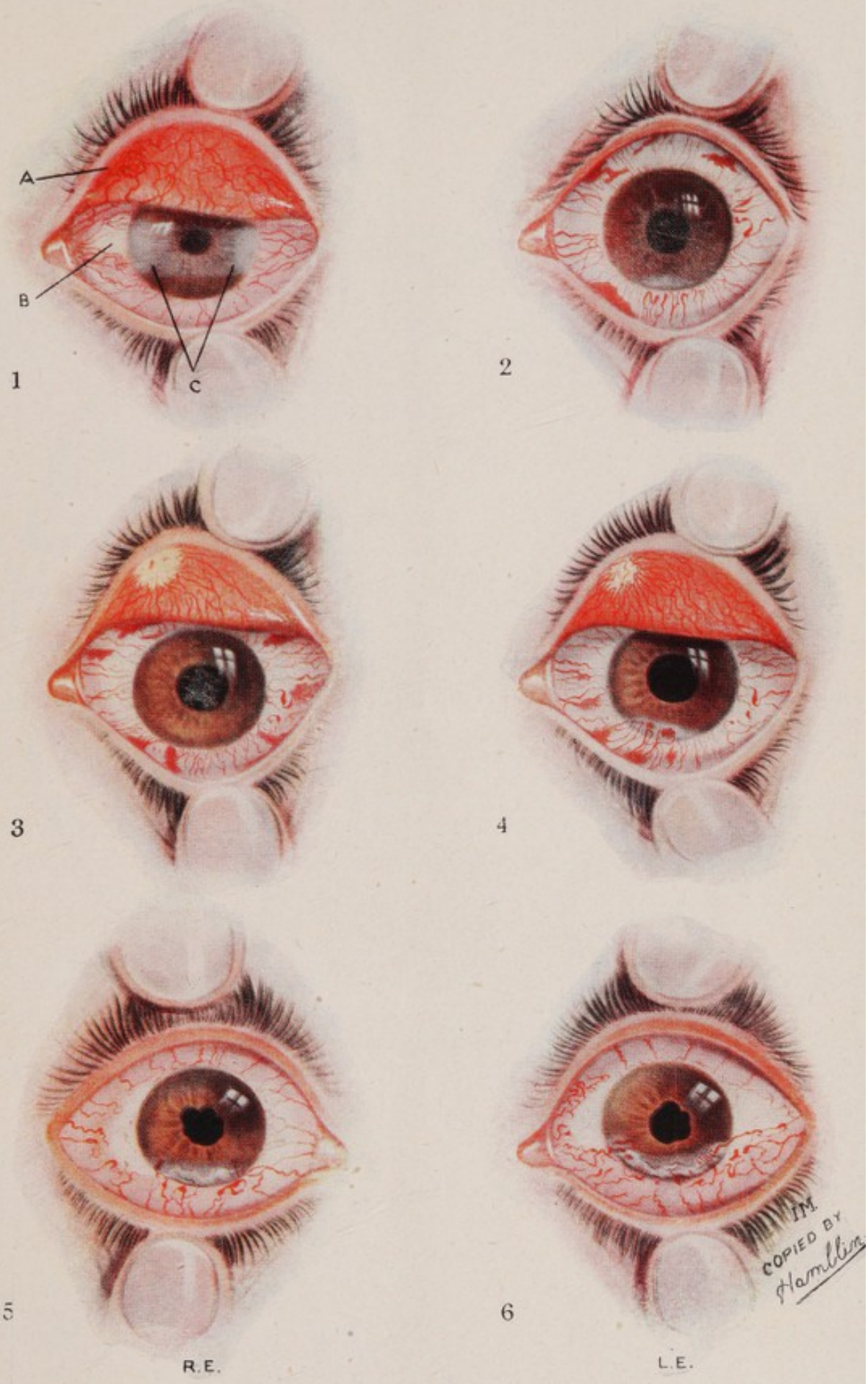
Fig. 3. Severe lesion 5½ weeks after exposure to a spray of mustard gas (the same eye as Fig. 2), showing an area of necrosis on the palpebral conjunctiva, hæmorrhages in the ocular conjunctiva with an area of necrosis at the limbus below, some bedewing of the corneal epithelium and clear substantia propria. (Stage of subsidence of primary œdema.)

Fig. 4. Severe lesion 8 weeks after exposure to a spray of mustard gas (the same eye as Figs. 2 and 3) showing partial vascularization of the necrotic patch in the palpebral conjunctiva, hyperæmia of the ocular conjunctiva around and in the former necrotic white area, reappearance of corneal œdema below, and vascular invasion of the cornea with thrombosis and hæmorrhage in this region.

Figs. 5 and 6. Very severe lesions (so-called delayed keratitis) in both eyes, 23 years after exposure to mustard gas and 13 years after the first attack of delayed ulceration.

R.E. Fibrous plaque of ocular conjunctiva at outer side ; abnormal varicose conjunctival vessels in outer and lower part ; gross scarring of lower part of cornea and limbus. Posterior synechiæ.

L.E. Triangular fibrous plaques at inner and outer side of limbus, tortuous and varicose conjunctival vessels, dense corneal scarring with vascularization and posterior synechiæ.



From A Study of Mustard Gas Lesions in the Eyes of Rabbits and Men.
By Ida Mann and B. D. Pullinger
(*Proc. R. Soc. Med.*, 1942, **35**, 229).

PLATE VIII.

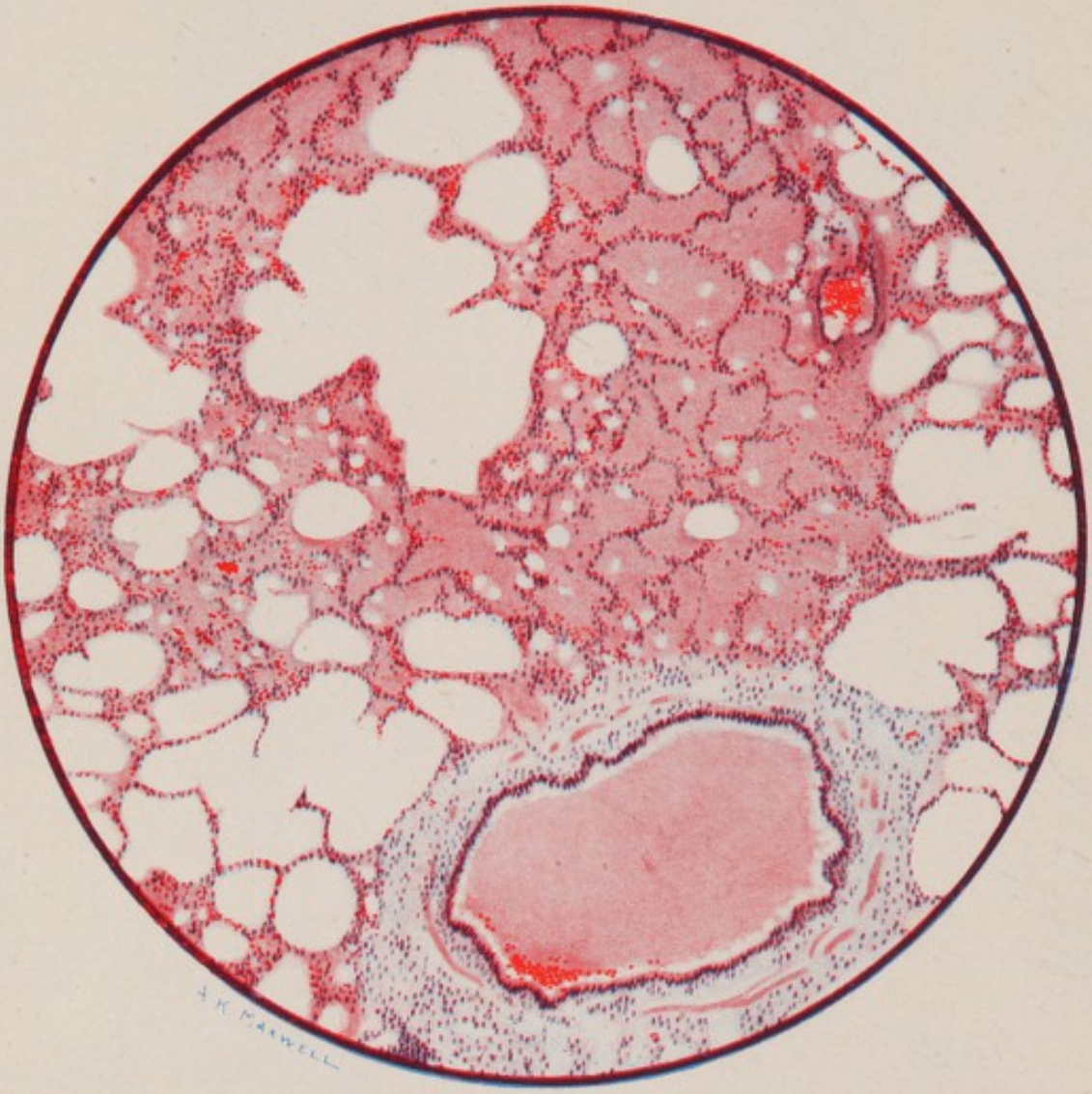


Plate VIII.—Microscopic Section of Human Lung after Phosgene Poisoning

Death occurred at the nineteenth hour after exposure to the gas.

The piece of lung shown is almost entirely useless for aeration of the blood. Most of the pulmonary alveoli are filled with œdema fluid, and the walls of the air sacs are burst asunder in many places. The rounded edges of these torn walls can be recognized both in the areas of emphysema and in the parts that are flooded with œdema fluid. The bronchus is also filled with œdema fluid, but it should be noted that its lining epithelium is intact and pus cells have not accumulated in the secretion. The blood vessels of the alveolar network are congested; intravascular thrombosis is frequently found in these smaller vessels, though it is not actually shown in the area of this section.

The main changes in the lung are :—

(i) Congestion, and occasional thrombosis, of the network of pulmonary blood vessels.

(ii) Abundant outpouring of inflammatory œdema fluid both into the tissues and into the air spaces of the alveoli and bronchi.

(iii) Disruptive emphysema of the weakened lung tissue.

The result of these changes is that the blood circulation through the lungs is impeded, and the respiratory exchange of gases between the blood and the air in the lung is seriously diminished. The affected person is in danger of death by asphyxia so long as his lung is drowned in œdema fluid.

From the third day onwards the œdema fluid is either reabsorbed or expectorated, and the lung soon resumes its functions. Broncho-pneumonic complications may develop from secondary infections, but they are not common.

The recovery of the lung, even after severe gassing, appears to be functionally good. In the earlier stages of convalescence there may still be signs of persisting oxygen want, so that tachycardia with excessively rapid respiration is the result of even slight physical effort. Later these disabilities vanish. The microscopic examination of lungs in these stages of recovery has not been made.

Plate IX.—Blue Type of Asphyxia from Phosgene Poisoning, with intense venous congestion

History of the case. Drawing made early on second day after exposure when there was copious frothy sputum, frequent cough, and hurried shallow respiration of 40 to 48 with temperature of 101° and pulse 100. The patient was bled 15 oz. and oxygen added to the air that he breathed. He soon made a complete recovery.

Such intense venous congestion was more frequent with chlorine than with phosgene poisoning. It is associated with a full strong pulse at the outset, though later the pulse may fail and the asphyxia change to the pallid type shown in Plate X. The patient as a rule is fully conscious and complains chiefly of headache and pains in the chest; he turns restlessly to and fro in extreme general discomfort, and his hurried breathing is interrupted from time to time by short bursts of coughing and expectoration. The lung is in the œdematous state shown in Plate VIII.

Oxygen, when given by an efficient apparatus, will at once change the blue tint of the face to a full pink colour, showing that it can still be absorbed by the blood through the lungs. Venesection relieves the discomfort felt by the patient, and lessens the embarrassment of the circulation.

PLATE IX.

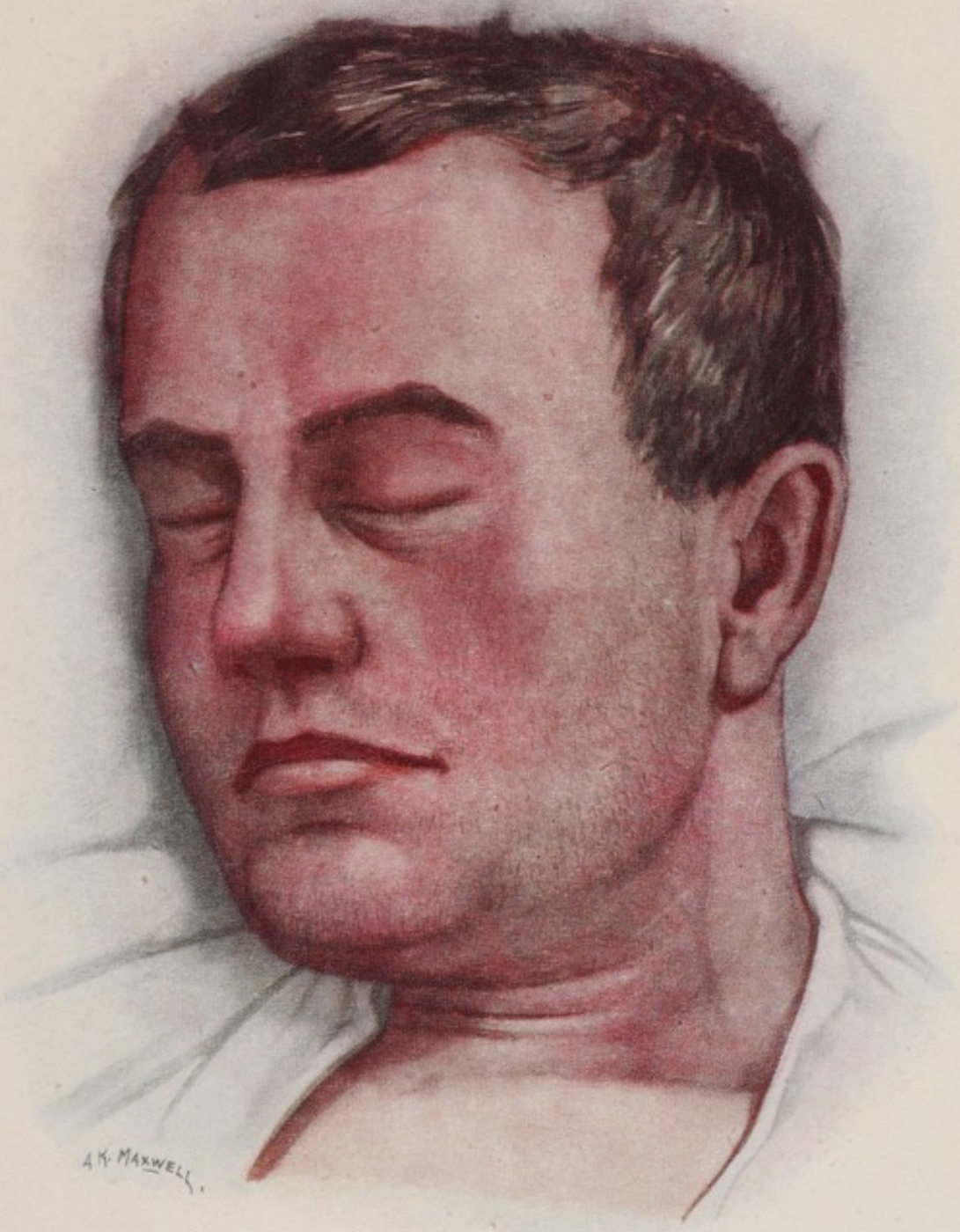


PLATE X.

Plate X.—Pallid or Grey Type of Asphyxia from Phosgene Poisoning, with circulatory failure

The cyanotic hue of the ears and lips, despite the general pallor caused by the failure of the circulation, indicates the intense want of oxygen from which the patient is suffering.

History of the case. Drawing made on second day after exposure, when there was profuse frothy expectoration, hurried shallow breathing of 50 a minute and a rapid running pulse of 132. The patient died two hours later.

This pallid or leaden-hued type of asphyxia is characteristically frequent after phosgene poisoning, and it may either develop at once, with a rapidly progressive failure of the circulation, or follow a stage of venous congestion.

The patient is restless, often semi-delirious, and his skin may be dry and hot, or cold in the final collapse, though it is not often damp with perspiration. The hurrying small pulse and the panting rapid shallow breathing, often with sounds of fluid in the trachea, are both characteristic. Examination of the chest finds physical signs very similar to those of the blue congested type, a little dullness on percussion and numerous fine râles and rhonchi, especially in the axillæ and over the back. In both cases the intensity of the pulmonary œdema is hidden from physical examination by the presence everywhere in the lungs of scattered islets of emphysema.

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