An atlas of gas poisoning.

AN ATLAS OF GAS POISONING

SECOND EDITION

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FOREWORD

The present edition of this "Atlas of Gas Poisoning" is published by arrangement with the Medical Research Council. The first edition was produced in 1918 by their predecessors, the Medical Research Committee, for official circulation only, and was intended to supplement the memoranda on the nature and treatment of gas poisoning which had already been made available to medical officers serving in the British Expeditionary Force. The present edition reproduces substantially the same text and drawings as the first edition, but the text has been revised in minor details in the light of later knowledge.

The drawings illustrate only the chief features in the pathology of the lesions produced by gas, and their primary aim is that of general instruction for doctors who are not already familiar with the subject.

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1st edition, August, 1918 (*Medical Research Committee*)
2nd edition, October, 1937

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INTRODUCTION

Out of all the various substances which could be used in gas warfare, only two have been chosen for illustration of their effects in this Atlas (except for Plate IV). They are Phosgene ($COCl_2$) and Mustard gas or di-chloro-di-ethyl sulphide $(CH_2Cl.CH_2)_2S$.

Phosgene is the chief of the many gases and liquids that are used for their effects as *pulmonary irritants*. These pulmonary irritants are inhaled as gases or vapours. They may cause some watering of the eyes, but the chief effect noticed at once is a catching of the breath or a choking sensation so that the chest feels gripped and incapable of free respiration. Coughing and vomiting may follow, and then after some time, varying from a few minutes to several hours, an inflammatory reaction appears in the lungs with the development of an acute oedema that may commence insidiously and yet progress so rapidly as soon to be a menace to life itself.

The alveoli fill with oedema fluid, which then rises into the bronchial tubes and may appear in a most abundant thin, frothy expectoration. Aeration of the blood is seriously interfered with, because the air sacs are either drowned with oedema fluid or burst by the efforts of coughing. Moreover the circulation through the lungs is embarrassed, both by the pressure of the fluid on the capillary vessels and by the local thrombosis that occurs in many places in the smaller lung vessels. The blood is concentrated by the loss of serum so that the count may rise to even eight or nine million red corpuscles to the cu. mm. This change adds to the difficulties of the circulation.

The affected person can no longer get the oxygen he wants, and he either dies from obvious asphyxia with progressive circulatory failure, or he collapses as the result of some muscular effort that suddenly makes a greater call for oxygen and so reveals the deficiency of the supply. Death is the result of this inflammatory oedema of the lung, and it occurs chiefly on the first or second day after exposure to phosgene. A few cases may chance to develop secondary bacterial infections of the lungs and to succumb to a later broncho-pneumonia, but they are relatively rare.

The main clinical features of acute phosgene poisoning may be summarised as follows:

(i) Catching of the breath, choking, and coughing *immediately* on exposure to the gas.

(ii) Inability to expand the chest in a full breath after removal from the poisoned air.

(iii) Vomiting, hurried shallow respiration, and sometimes coughing with an abundant expectoration, follow. Pain is felt behind the sternum and across the lower part of the chest. Fine râles are heard in the axillae and over the back.

(iv) Cyanosis next appears, in association either with a full venous congestion or with the pallid face of circulatory failure. The development of these dangerous symptoms may occur after many hours' delay, and sometimes, as the result of muscular effort, with unexpected rapidity in an apparently slight case.

(v) Death, which may or may not be preceded by mild delirium or unconsciousness, rarely occurs after the second day.

Mustard gas is known as a vesicant. It may exert its irritant action either as a vapour concentration in the air or by direct contact with the liquid. The liquid or vapour clings to the clothing and slowly exerts its continuously irritant action on the body.

As a rule no irritant effect is felt on exposure, whatever the concentration, but after about two to six hours the skin and mucous membranes begin to react with a progressive inflammation that may result in local necrosis and desquamation of these covering membranes. There is intense conjunctivitis; the skin turns an angry red, and this erythema is soon followed by the skin blistering here and there over the face and body. The passage of the vapour down the respiratory tract may cause such severe injury to the mucous membranes lining the trachea and bronchioles that they are eventually destroyed and sloughed away. Bacterial infection then seizes upon these raw surfaces, and the patient may die from secondary septic broncho-pneumonia.

Death is never the direct result of the action of the poisonous vapour. and there is no initial pulmonary oedema. From the second day onwards through the first and second week severe cases may die, but only as the result of secondary bacterial infection. Mustard gas therefore differs entirely from the lung irritants such as phosgene, which kill directly and speedily by flooding the lungs with oedema fluid.

The main features of poisoning from mustard gas may be resumed as follows:

(i) *Delay* of the irritant effect for at least two to three hours, and then a comparatively slow development of the various inflammatory reactions.

(ii) Vomiting, and a sense of burning in the eyes, with discomfort in the throat, hoarse cough, and some retro-sternal pain.

(iii) Intense conjunctivitis that temporarily " blinds " the man.

(iv) Reddening of the exposed skin surfaces and of the moist areas in the axillae and groin, followed by blistering, excoriation, and brown staining.

(v) Inflammatory necrosis of the mucous membrane of the trachea and bronchi, with the secondary development of infective bronchitis or septic broncho-pneumonia.

(vi) Death is relatively uncommon: it occurs later than the first day and only as the result of septic complications.

1. Microscopic Section of Human Lung from Phosgene Poisoning

(See Plate I)

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 oedema fluid, and the walls of the air sacs are burst asunder in many places. The rounded edges of these torn walls can be recognised both in the areas of emphysema and in the parts that are flooded with oedema fluid. The bronchus is also filled with oedema 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 m[illegible] changes in the lung are:—

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

(ii) Abundant outpouring of inflammatory oedema 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 oedema fluid.

From the third day onwards the oedema fluid is either reabsorbed or expectorated, and the lung soon resumes its functions. Bronchopneumonic 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.

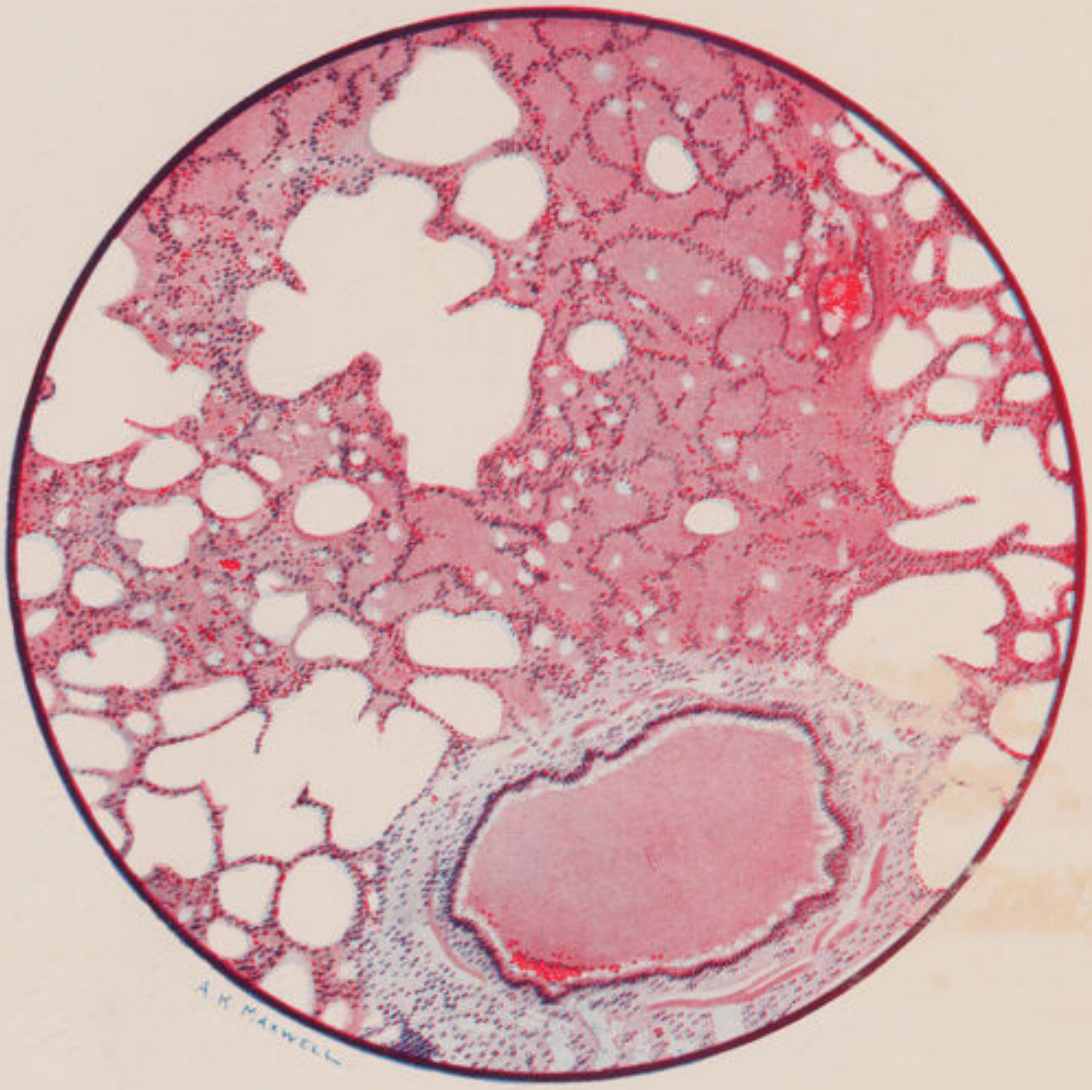
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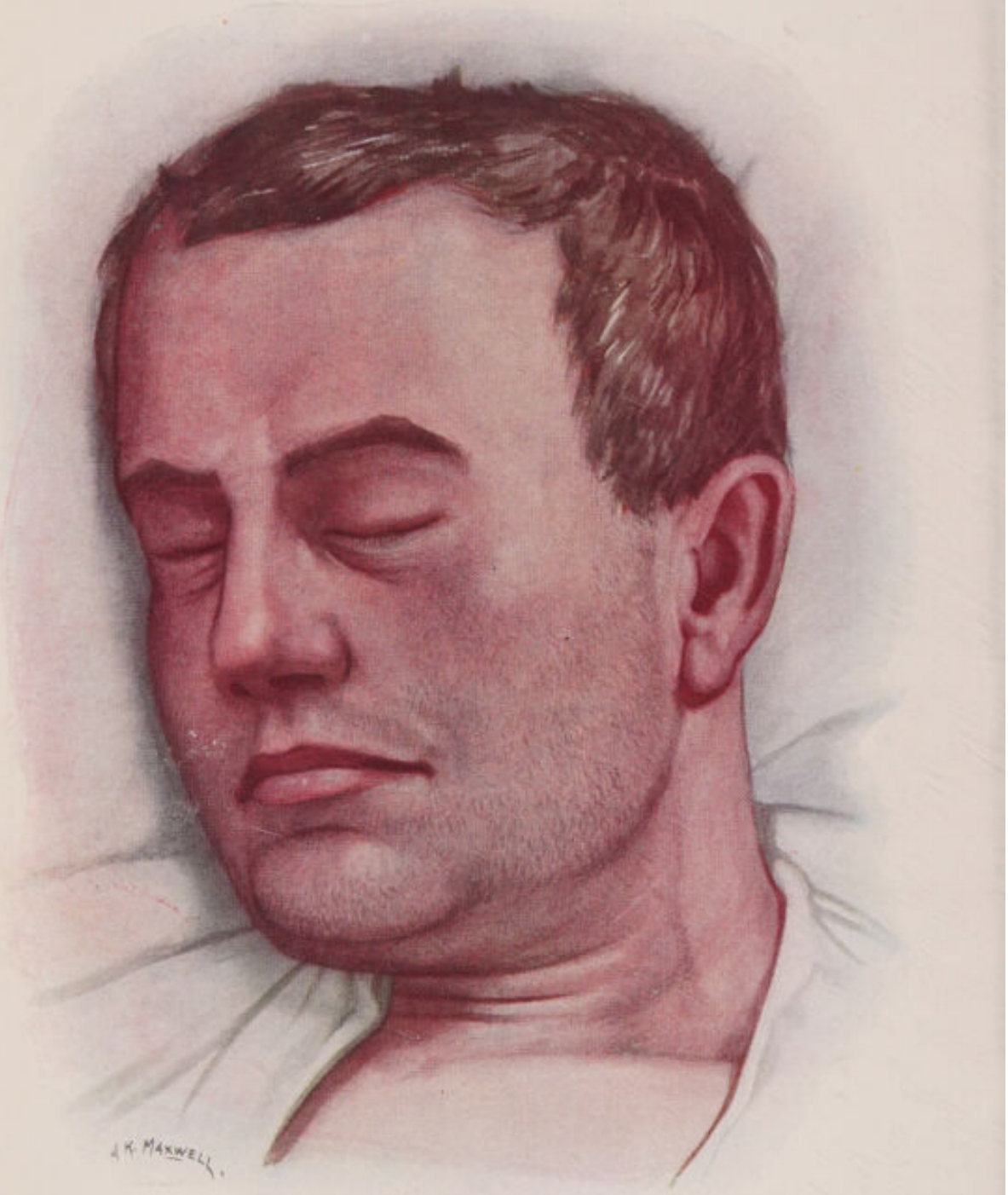
PLATE II.



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3. Pallid or Grey Type of Asphyxia from Phosgene Poisoning, with circulatory failure

(See Plate III)

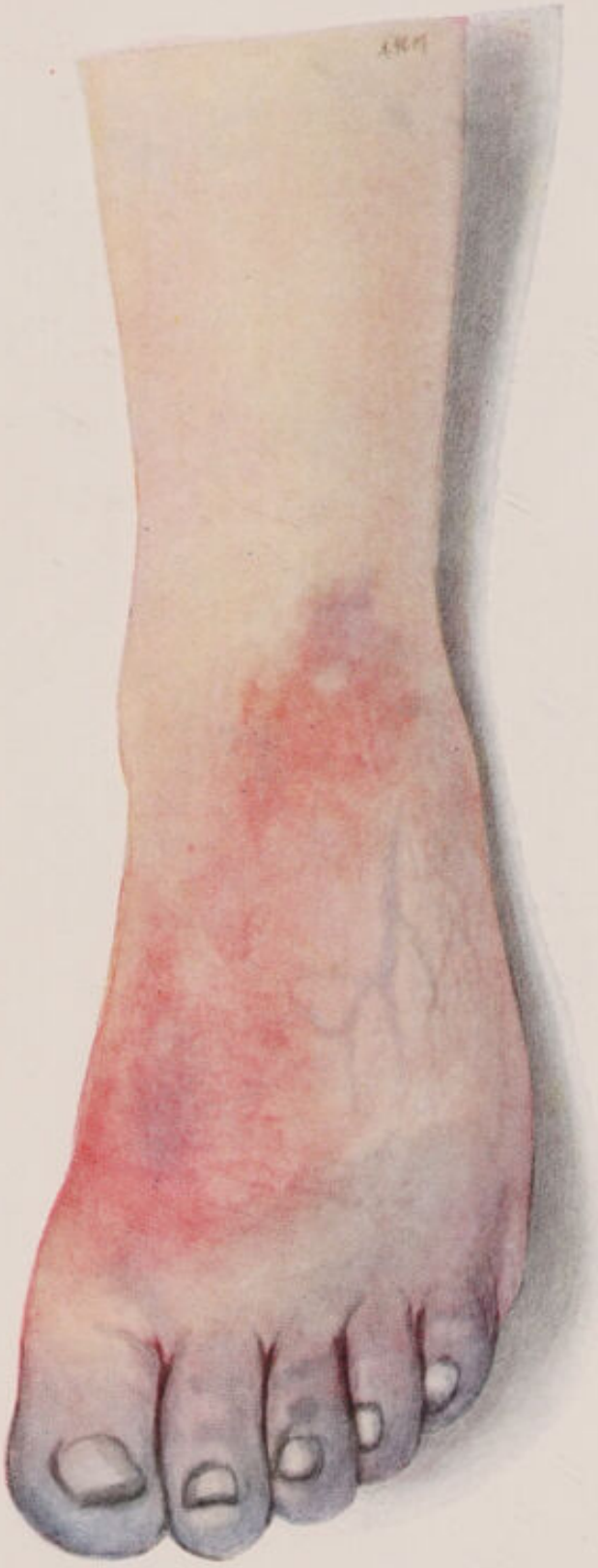
The cyanotic hue of the ears and lips, despite the general pallor caused by the failure of the circulation, indicates the intense want of oxygen from which the patient is suffering.

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-delirous, 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 axillae and over the back. In both cases the intensity of the pulmonary oedema is hidden from physical examination by the presence everywhere in the lungs of scattered islets of emphysema.

PLATE IV.



4. Gangrene of Foot caused by Vascular Thrombosis from Chlorine Poisoning

(See Plate IV)

History of the case. Gassed by chlorine in 1915 under conditions which could not have induced frost-bite. Severe dyspnoea from pulmonary oedema.

Drawing of foot made on fifth day. Both feet were then anaesthetic and stone cold: no pulsation could be felt in the dorsalis pedis artery. The right hand also was mottled, cold, and painful. The circulation was restored in a few days, with complete recovery, except that two toes became black and shrivelled.

Such arterial thrombosis, of slowly progressive onset, is quite uncommon in the extremities, though it is occasionally seen with phosgene. The obstruction is very rarely so complete as to cause gangrene and death of the tissues. But this drawing of a visible condition is introduced in order to emphasise the fact that an unseen vascular thrombosis of smaller vessels in deeper organs of the body is frequently found with phosgene poisoning. Such thrombosis is revealed by the microscope in fatal cases in the smaller lung vessels, in the kidney, in the mucous membrane of the stomach, and in the brain. Indeed in death with prolonged asphyxia from phosgene poisoning the white matter of the brain is often seen to be thickly sown with brownish-red petechial spots around each tiny arterial thrombus. This is, of course, not specific nor the result of the gas—it is evidence of a serious degree of anoxaemia and a similar picture is presented by anoxaemias due to quite other causes; to carbon monoxide poisoning for instance. The obstruction to the lung circulation has already been referred to; the kidney thrombosis does not appear to have any serious results; and, except where larger haemorrhages have burst in the brain, the scattered cerebral thrombi do not appear to be of grave clinical import. The petechial areas within the stomach may occasionally become the seat of a superficial ulceration. Large thrombi are sometimes found within the heart, but they also are associated with, rather than the cause of, the other changes that lead to death.

Note: Plate IV, which illustrates a case of chlorine poisoning, is retained mainly for its historical interest. It is of less importance than was formerly thought, because gangrene is a rare sequela of gas poisoning.

5. Erythema of Skin from General Exposure to Mustard Gas Vapour

(See Plate V)

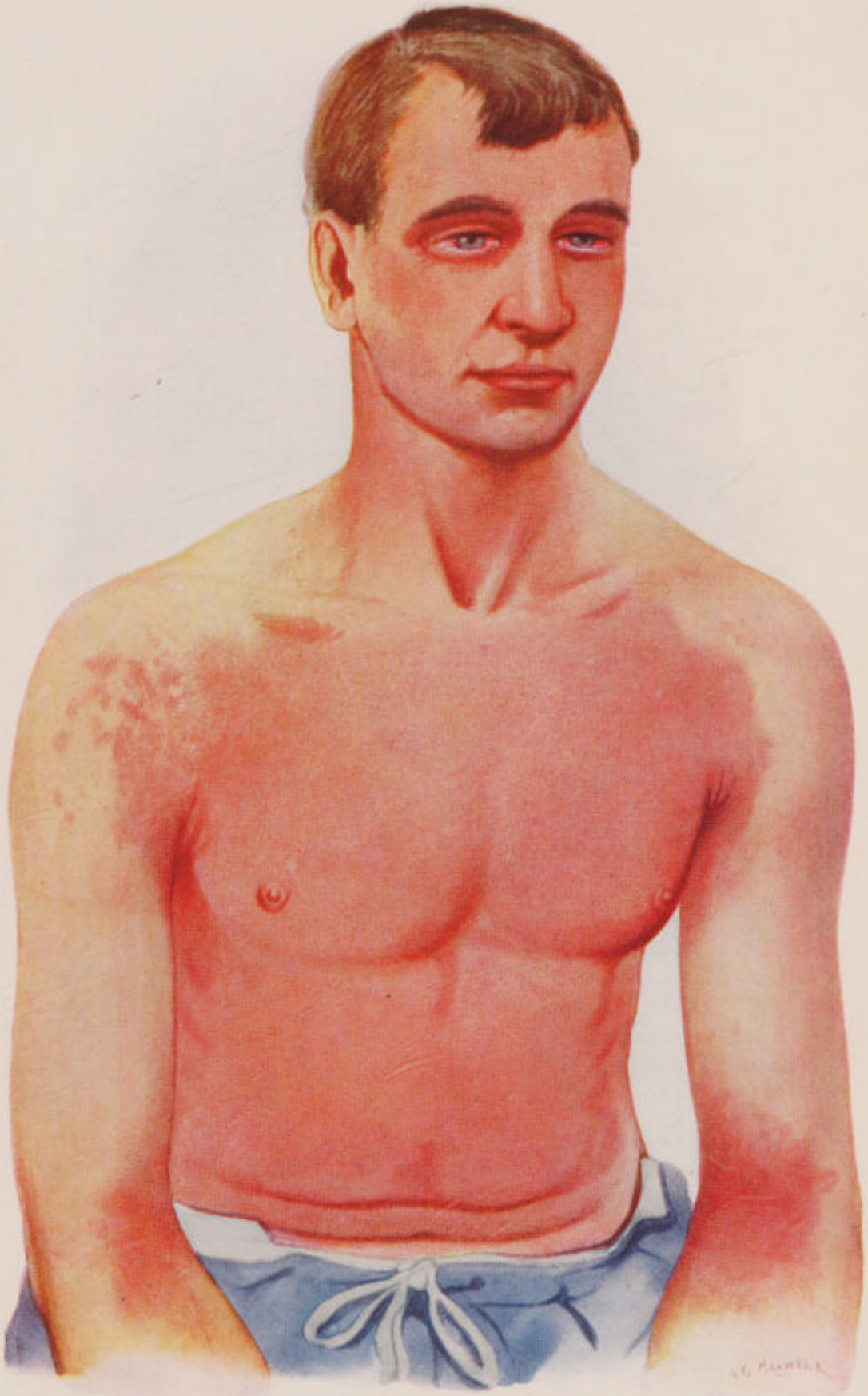
Dermatitis of this distribution, associated with conjunctivitis, forms a picture characteristic of poisoning by this vesicant.

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 axillae, the flexures of the elbows, and the perineum and inner surfaces of the thighs are particularly affected, that is, in the places where the skin is often sodden with fatty perspiration. This special distribution of the diffuse erythema characterises 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 oedema 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 oedema 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.



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6. Blistering of Buttocks by Mustard Gas

(See Plate VI)

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 bullae, 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.

7. Burning of the Perineum

The perineum is peculiarly liable to become inflamed after exposure to the vapour of mustard gas, and the external genitalia become oedematous 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.

8. Brown Staining from Mustard Gas

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.

9. Ulceration of the Trachea by Mustard Gas

(See Plate VII)

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 oesophagus has refused ingress to the toxic vapour, occurs also in the case of 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 débris 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 bronchopneumonia, localised 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 of 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.

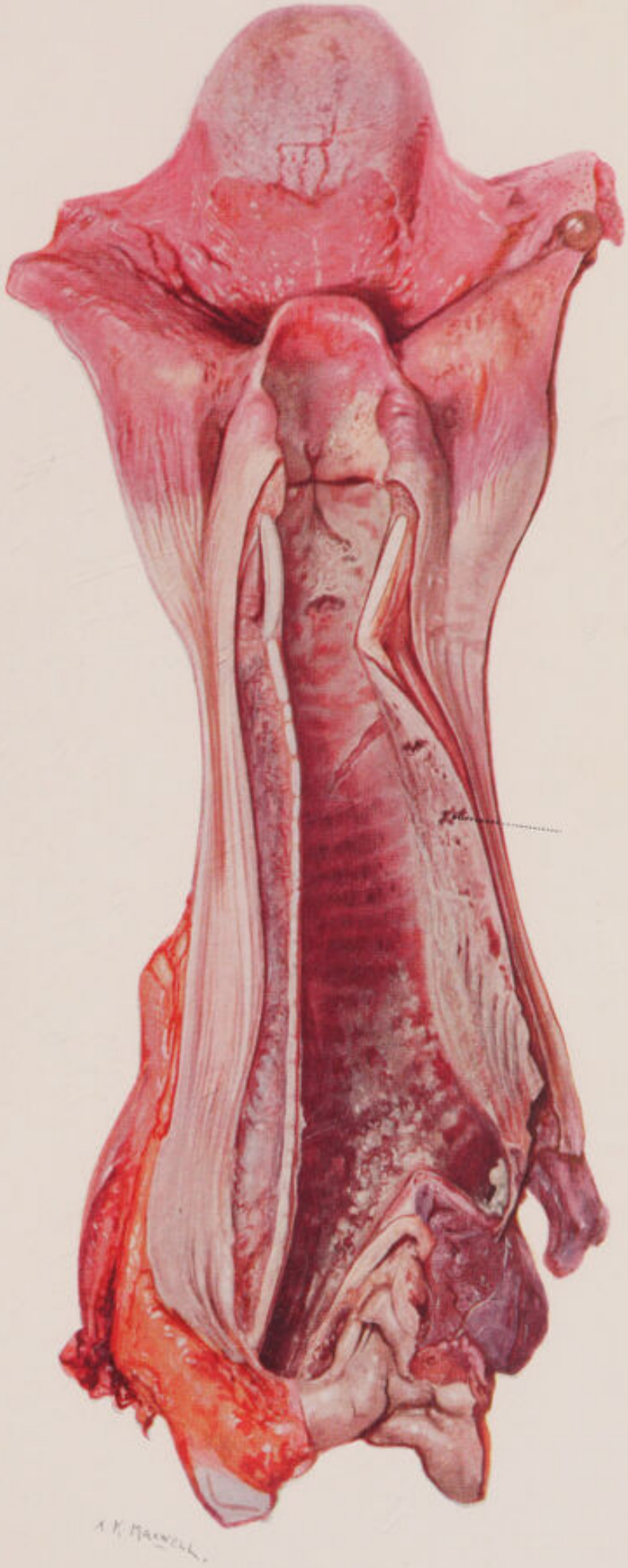
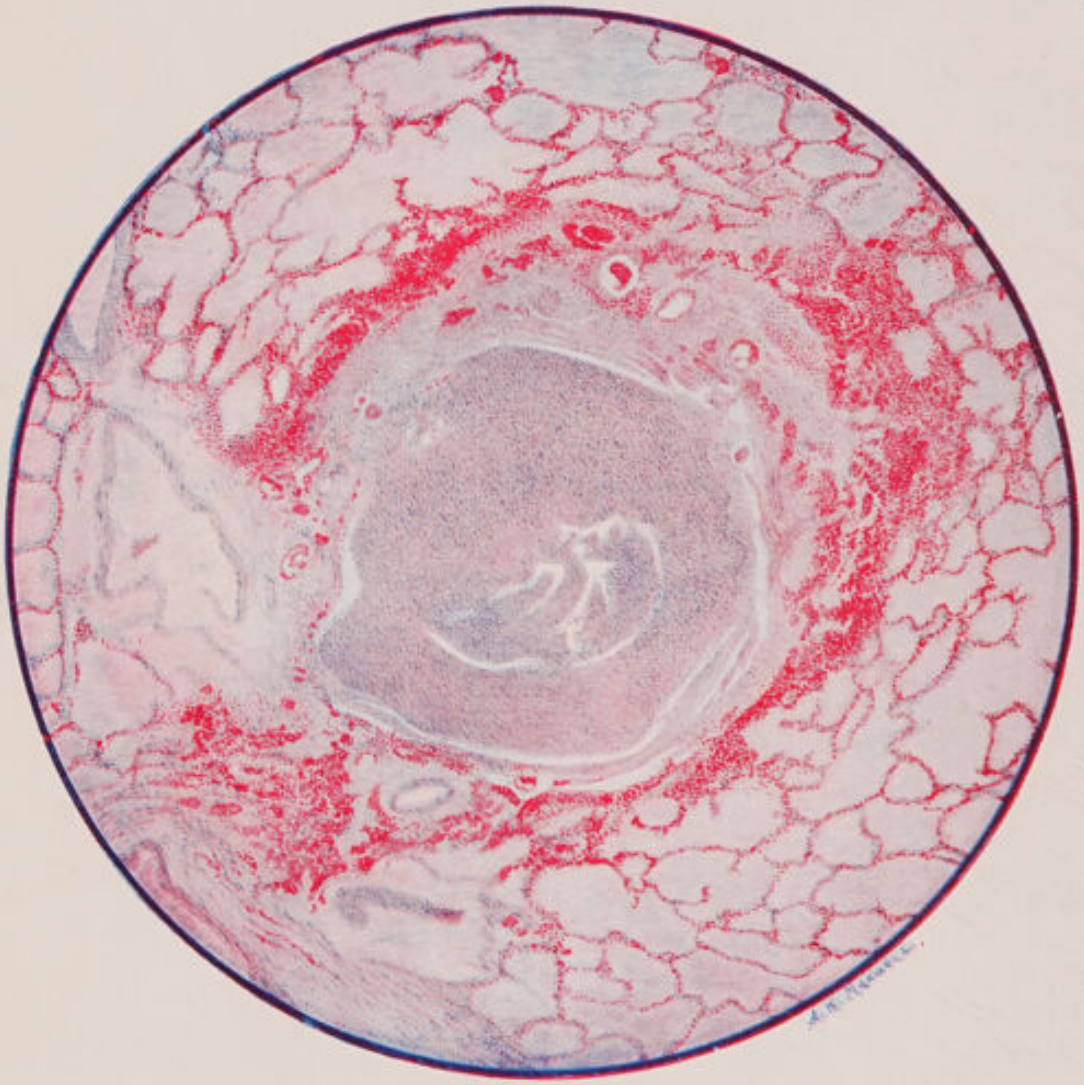


PLATE VIII.



10. Microscopic Section of Human Lung after Mustard Gas Poisoning

(See Plate VIII)

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 haemorrhage 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 generalised pulmonary oedema and no disruptive emphysema.

Mustard gas may cause some catarrhal desquamation of the pulmonary endothelial cells, but it rarely excites an outpouring of oedema 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 I). As infection spreads into the lung tissues, patches of septic broncho-pneumonia and small abscesses develop, and these often excite an inflammatory oedema 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.

11. Severely Burned Eye in the Acute Stage

(See Plate IXA)

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 oedematous eyelids, which may even be blistered, and there is often severe pain behind the eyes and in the forehead. The conjunctiva is swollen, oedematous, 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.

The majority of gassed eyes exhibit inflammation of a general character that is not illustrated in this Atlas. But examples continually occur in which the eye is more severely burned, and these may be recognised by certain characteristic features that are depicted in the drawing. 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 oedematous, serious injury from the burning is likely to have occurred.

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 oedema, which compresses the vessels and impairs the circulation, thus acting as a menace to the nutrition of the cornea. 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, which should always be used early in severe cases or where there is much pain and blepharospasm.

12. Slightly Later Stage of Acute Burning of the Eye

(See Plate IXB)

The swelling in the conjunctiva above and below has subsided, but the vascular injection remains, and the solid white oedema in the palpebral aperture is still well marked. The cornea is grey and lustreless in the exposed area.

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Contd. from page 14 (see Plate IXB).

With the lowering of the nutrition of the corneal epithelium, secondary infection is liable to take place. In this case an infiltrated corneal ulcer is seen associated with hypopyon. It is therefore important when there is conjunctival discharge, which indicates secondary infection, that in addition to the use of atropine the conjunctival sac should be cleansed by frequent bland irrigations and by the instillation of antiseptic drops so as to check infection of any corneal ulceration which may develop. Otherwise the infective progress which has led to hypopyon may progress to panophthalmitis.

History of the case. The casualty was caused by a mustard gas shell bursting close to the man when he was riding a restive mule, and his box respirator was momentarily displaced. A fine spray of the liquid must have splashed lightly over his right side, for cutaneous blisters developed on this side only of his neck, cheek, and forehead. The right eye showed serious burning with the central white band, while the left eye was only in the state of general red conjunctivitis.

13. Stage of Resolution after Severe Burning of the Eye

(See Plate XA)

The vascular injection is passing off, the interpalpebral zone of solid oedema is becoming absorbed, and the corneal epithelium has regained its normal lustre. At this stage the use of atropine should be discontinued.

14. Late Stage of Resolution

(See Plate XB)

The earlier vascular injection above and below the cornea has practically disappeared; the solid white oedema has been absorbed, and the conjunctiva in the palpebral aperture now shows definite injection, often of a bright violet tint. The entire picture has changed, so that the parts which were red in the acute stage are now white, and the part which was formerly white is now red.

At this stage the use of atropine and shades should be abandoned. Astringent drops should be instilled and photophobia combated with cold douching, etc., while fresh air and occupation will help to restore the general health of the individual and mitigate any tendency to neurasthenia.

This drawing of the late phase of recovery after a severe burn would serve equally well to illustrate the early stages of a mild burn, which is the commonest form of eye lesion after exposure to the vapour rather than to droplets of the liquid. Though discomfort may make the patient unable to open his eyelids, examination will in such mild cases reveal a lustrous cornea and a central band of red injection instead of the central zone of white oedema that characterises severe burns.

15. Drawing of the Cornea in the Acute Stage of Severe Burning

(See Plate XIA)

This corresponds with Plate IXA. The exposed central area shows grey haze and loss of lustre on its stippled surface, which gradually fades off to the bright lustrous normal surface in the part above that has been protected by the eyelid. Injection of the conjunctival vessels is seen only in relation to this upper and less burned area.

16. Drawing of the Cornea in the Stage of Resolution after Severe Burning

(See Plate XIB)

The cornea is now smooth and bright with a clear light reflex on its surface. But some grey superficial nebulae are seen in the centre, and these may persist for several weeks. The injection of the conjunctival vessels is now limited to the central band.

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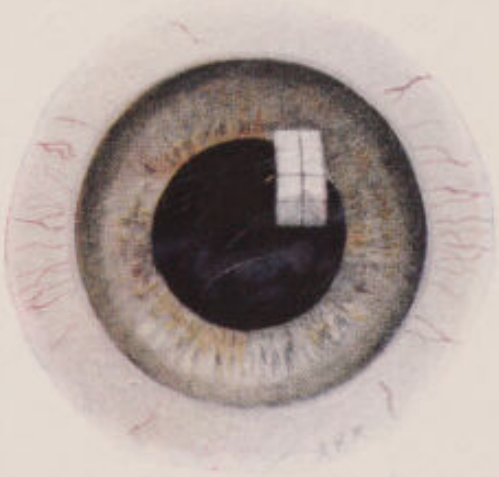

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MATERNAL MORTALITY

Two reports have recently appeared on this subject, one devoted especially to conditions in Wales where maternal mortality is high, the other to England. The documents are essentially practical and concentrate upon remedial measures in areas where mortality is highest. A working scheme is outlined whereby local authorities should co-ordinate the work of health visitors, midwives and doctors—whether in general practice, attached to hospitals and clinics, or specialists. Each report contains numerous statistics. Report for England, 353 pages. 5s. (5s. 11d.). Report for Wales, 156 pages. 2s. 6d. (2s. 9d.).

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