MEDICAL ASPECTS OF CHEMICAL WARFARE

By

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I have been assigned the duty of skipping lightly over the whole field of the medical aspects of gas warfare; which means, I hope, that almost everything I say this morning will be taken up in somewhat more detail by subsequent speakers. Perhaps the most useful thing I can do is to compare the several gases and emphasize the differences in the various problems they raise, rather than to go into detail about any one.

May I start by reading two quotations, to get ourselves into the proper atmosphere of this subject. The first is from Col. Waitt's book "Gas Warfare". He says:

"The adoption of chemicals as weapons was as logical as it was inevitable in a world war in which all the principal combatants were nations highly developed in science. When the German advance was halted after the Battle of the Marne in 1914, both sides firmly entrenched themselves. A dead-lock existed; neither side could gain any material advantage. Machine guns had increased firepower to such an extent that only by a mass superiority in manpower could ground be captured. The attack had failed, and mobility for both the Germans and the Allies had been lost.

" It became apparent that the issue could be determined only by exhaustion, and the odds were that the Germans, completely surrounded, would be exhausted first. The Germans recognized the situation and tried to find some agency to reach the enemy behind his sandbags and down in his dugouts. They looked for something which would rob the defender of the superiority which the machine guns and rifles fired from behind entrenchments had given him. Thus chemical warfare was born. The gas particles that could shoot around corners and into holes were given the job of driving the defender out of the dugouts into which steel particles had driven him, and from which artillery shells had failed to blast him."

He goes on then to describe the first gas attack.

"The weather was perfect, a glorious spring day. During the morning there had been somewhat more than the daily artillery shelling of Ypres, but it was not unusual, and by noon time it had stopped. During the early part of the afternoon everything was quiet. Suddenly at five o'clock (British Time) the calm of the spring afternoon was shattered as a vicious bombard-

ment of Ypres was started by the heavy guns. For the first ten minutes the German light batteries were silent and no shell fell in the forward trenches, which were about four miles north of Ypres. Nothing must disturb the quiet air here. As the bombardment began, the men in the forward positions heard a hissing sound coming from the German trenches. At first two greenish-yellow clouds appeared on each side of Langenmarck, which was in possession of the French. Then these merged and a heavy cloud almost like a wall, nearly five miles in length, stretched itself along the German trenches from Steenstraat to a point a half a mile west of Poelcappelle. This wall-like cloud was perhaps as high as a man's head at first, but gradually it became higher as it moved slowly to the Allied trenches. Observers who saw it from a distance said it had the appearance of a mist 'such as is seen over water meadows on a frosty night.' The wind was light that afternoon, only four or five miles per hour. Gradually the cloud borne by the wind swept over the Allied lines, bringing horror, confusion and death into the ranks of some fifteen thousand men. The time consumed by emission of the gas was only fifteen minutes, but the poisonous clouds of chlorine hung over the Allied trenches for a much longer time and swept back into the rear areas for several miles, gradually becoming weaker.

"The French Colonials had the worst of it, for the cylinders were all emplaced in front of them. Only a part of the third brigade on the extreme left of the Canadian Division was in the main path of the gas, but the cloud swung in to the rear of the Canadians and moved back with the wind.

"The Allies were entirely unprepared for such an attack. They had information through military intelligence that a gas attack was coming, but they didn't believe it. A few days earlier a deserter captured near Langenmarck had told the French that the Germans intended to use 'tubes of asphyxiating gas placed in batteries of twenty tubes for every forty meters along the front of the XXVI Corps (French).' The prisoner even had in his possession a cotton pad which he said was to be dipped in chemicals to counteract the effect of the gas. The Royal Flying Corps was ordered to observe the German lines, to see if any special apparatus could be discovered, but they found nothing suspicious, and no further steps were taken. It was not until French Colored troops without their officers began drifting upon the roads through the British area that it was realized that the greenish-yellow cloud was the gas against which they had been warned. It is sometimes difficult to convince a military man of the possibilities of new and untried methods." I am still quoting Colonel Waitt!.

"The Casualties were about 5,000; a large number of deaths occurred on the ground, and besides these there were many deaths in the hospitals."

I now turn from the picture of the gas attack to an eye witness account of the consequences. This is taken from an article by Dr. G. W. Norris, "Some Medical Impressions of the War."

"A field hospital full of freshly and badly gassed men is, in the estimation of all who have had an opportunity of seeing it, the most horrible and ghastly sight of the war. Even the man who has received multiple and severe wounds, when he has been splinted, put to bed and given his morphine, is relatively comfortable; but to see a hundred or more men, hale and hearty a few hours before, slowly strangling to death from pulmonary edema with gradually increasing dyspnoea, cyanosis and pallor, making futile efforts to expectorate and to assist their breathing by voluntary effort and muscular contortions, until exhausted they pass from semi-delirium into stupor, collapse and death, is a never-to-be-forgotten sight, a sight which makes one clench one's teeth and curse the Hun who started this dastardly infamy. This is phosgene! But can nothing be done? Yes! The cyanotic cases are promptly bled, one pint, sometimes two. The ward looks like a shambles because in hurrying from bed to bed, twenty to thirty in a row, the spurting blood has left its trace upon bed and floor and linen. Meanwhile oxygen is being administered to greedy mouths while hands are loath to loose the bag when their five minutes of respite are over. For never are there enough bags for all, and the precious gas we must not waste, for it has been no small task to bring these great iron tanks up to the front. Opium we dare not use, for it checks an oft life-saving cough, But the gray cases, what of them? Lying about with a clammy skin, too weak to move or even care. Some venturous spirits say that one should bleed and then transfuse, but most that we should not meedle."

That is the last war. Such effects are not likely to be seen, in the civilian areas certainly, nor are the same treatments recommended; but, as Dr. Senior has already emphasized, there are certain very obvious morals to be drawn from these experiences by physicians today, and I should like to reiterate some of them. He spoke of the great importance of supporting morale. I agree that unquestionably the most important service physicians can render in a gas raid or a supposititious gas raid will be in supporting morale. Perhaps the best way to start on this problem is to counteract these ugly pictures I have just recalled by some cold figures.

Slide 1 is a summary of the American Expeditionary Force casualties and deaths in World War I. As you see, about two-thirds of all casualties were due to gunshot or like mechanical injury and about one-third were due to gas. Now, of those casualties, most deaths were hospital deaths. The actual numbers do not interest us particularly, but note that although one-third of all the casualties were gas casualties, only one-tenth of the deaths were gas deaths. Gas certainly did not produce anything like its proportional amount of deaths. The actual percentage of gassed individuals dying, from all sorts of

gases and under all conditions, was somewhat under two percent. Slide 2 gives the material in somewhat greater detail, for each of the gases. Note that the average hospitalization is about six weeks after gassing with either mustard or phosgene.

Thus gas in itself is not so terribly bad, medically considered. It may disable a large number of individuals and it may have extremely valuable military harassing or other purposes; but, in terms of the clear facts, as we will see, gas is not so horrible. There is no basis for the almost morbid fear of gas and of the whole subject, which most lay people and even some medically trained people have.

Actually, even the deadly war gases are not very deadly agents as compared with many of the substances used routinely by the medical profession. They are not extremely toxic chemicals. In the case of phosgene, for example, the exact amount of gas which is necessary to kill is not precisely known, but it may be assumed it is of the order of one milligram per kilo body weight. You continuously use drugs in practice that would kill in far smaller doses.

Slide 3 gives some data on toxicities and concentrations. Some of this material has already been referred to. It is based largely on Prentiss. Concentration is expressed in ounces per thousand cubic feet or milligrams per liter; and minimal doses are stated for a detectable odor, for irritation after ten minutes exposure, or for lethal action after the same exposure. I call your attention to the law that toxic effect is proportional to the time of exposure multiplied by the concentration of the gas. In these gases, of course, the amount that actually gets into the body increases with the time during which the gas is being breathed; so that with a thirty minute exposure, approximately one-third the indicated concentration for ten minutes would have a like effect.

The slide shows: the vesicant group -- mustard, Lewisite, and ethyldichlorarsine; the lung irritant group -- chlorine, phosgene, and chlorpicrin; the stenutant group -- diphenylaminechlorarsine and diphenylchlorarsine; and the lacrimant group -- chloracetophenone and brombenzylcyanide. You will notice that certain of these gases are particularly mischevious. The concentration which will produce irritation is actually less, in the case of the vesicants, than the amount which can be detected by odor. In other words, one can be seriously exposed to mustard or Lewisite or ethyldichlorarsine and not be warned by smell of the exposure.

The lethal concentrations are rather interesting. Lewisite and mustard are about the same, lethal in lowest concentrations; phosgene is almost the same order; the others are definitely less toxic. Chlorine is relatively a non-toxic gas; and you will note

that the irritant gases, because of their extremely low concentrations which suffice to produce irritation, are less dangerous. The indicated concentrations for irritation can, in fact, probably be tolerated for less than ten minutes.

The duty, then, of the physician, which you will wish to impress especially upon the physicians whom you train, is mainly to support morale, to avoid panic in case of a gas attack or of an attack which is misjudged by the populace to be a gas attack. The physician will have little opportunity to exercise any prophylactic maneuvers. He will ordinarily not be called upon, as you already know, to be responsible for decontamination or even cleansing. He will, however, be responsible for the appropriate treatment of gas casualties, and that involves, of course, a correct differential diagnosis. You have already seen lists of references and guides that you may consult and recommend. I have a few references in slides 4, 5, and 6; and some comments on these may help you to decide on their usefulness. Jacobs' book is essentially chemical. Prentiss' is a little out of date, but is a comprehensive treatment of the whole subject of gas, mainly from the chemical point of view, but including also a discussion of tactics and a little on the medical side. Sartori treats gas almost entirely from the chemical side. Vedder's book, "Medical Aspects of Chemical Warfare," is unfortunately rather out of date, but still has a great deal of valuable information in it and is a full sized treatment of just what the title indicates. Wachtel gives some account of the history and results of research in Germany during the last war. The next two references I most emphatically commend to your attention. "The treatment of Gas Casualties," available from the Superintendent of Documents, Washington, D. C., at ten cents. (I believe the latest edition of that is November 27, 1942, which you have), is very useful as a concise source of information on this subject. Even more readable and informative is the British, "Manual on Chemical Warfare," published in this country by Chemical Publishing Company. For a concise, lucid and highly interesting account of the medical aspects of gas warfare I think you can hardly do better. Finally, the volume by Waitt is a good treatment for the layman and easy reading -- as you can tell from the quotation I read from it.

So much then for such general introductory comments. We must pass on to a more particular consideration of the medical aspects. The speakers to follow will consider with you, in order, the lung irritant group, with emphasis on phosgene; then the vesicant group, covering mustard, the new nitrogen mustards, and Lewisite and the other arsenicals. Later speakers will take up the sternutators and lacrimators, the systemic poisons, the treatment of burns, and so on.

The systemic poison gases will probably not come into much practical use; but you are familiar with them. Cyanide, monoxide, perhaps hydrogen sulphide, and arsine are the ones most likely to need attention. And harassing agents would hardly be dropped on a

civilian population. The lung irritant group constitute a real danger, and the persistant vesicants are likely to need most attention. To varying degrees -- based on present knowledge, time, and personal methods -- I believe the speakers will take up these gases from the medical point of view under the heads of: essential mechanism of action, physiological pathology, signs and symptoms, prognosis, treatment (probably in some detail), and late effects.

The action of all these gases, to sum them up collectively (and they are called gases and are breathed, although some are solids), centers more or less upon the lungs. All of them except the systemic poisons produce lung damage, whatever else they do. All of them, except the vesicants (which reach the skin even with the head protected by a mask) enter the body through the lungs. It will perhaps be specially useful to compare the respiratory system actions of these different gases.

The systemic poisons exert little such action. Hydrogen sulphide does produce some irritation of the respiratory tree; arsine a negligible amount; the others none. The mouth is essentially a mere portal of entry, and the effects are produced after absorption, mostly on the respiratory and other centers of the central nervous system. Dr. Farmer will take them up in a body tomorrow. The other groups do act on the respiratory system. Slide 7 is a simple diagram indicating the positions of action in the respiratory system, of these different types of gases. The position of the label shows whether the gas acts high up or low down in the respiratory tree. Ammonia and the irritant gases act mostly right in the nose and mouth; mustard on the trachea and bronchial system particularly; chlorine acts there and also in the alveoli; phosgene and its related compounds act hardly at all on the upper respiratory passages, but produce violent damage in the terminal tubes and alveoli of the lungs. There is a progressive descent of action from the harassing, through the vesicant, to the lung irritant group.

Turning now more particularly to the vesicants, you are well aware that these gases are called vesicants not because they act only upon the body surface, to produce vesication, but because they are the only ones of the gases which do act on the skin. All the vesicants also do other things. Besides the skin, the eyes are injured and even smaller exposure may do more serious damage to them. They are also respiratory irritants, acting on the pulmonary system, and in addition they are systemic poisons. I shall not go into the systemic action, but mention that mustard, the N mustards, and Lewisite produce marked changes in the bone marrow and the lymphatic system; and, of course, the arsenic group, Lewisite and so on, produce all sequelae or concommitants of arsenic poisoning as well.

A characteristic of this group is latency of onset; from exposure to symptoms there is always an interval. In the case of liquid splashes of Lewisite or mustard, that interval may be rather

short; in fact splashes of liquid Lewisite in the eye will produce serious symptoms within perhaps two to five minutes. But there is no immediate effect even here and, in the case of vapors, the latency of eye symptoms may be a couple of hours and of skin symptoms six to twelve hours. Then the pathology and symptoms progressively increase until a maximum is reached usually within one or two days. Because of the delayed action, one common error is made by the uninitiated person who has not had the mask drill that Dr. Senior emphasized. He is exposed to Lewisite, let us say, and puts on his gas mask. Everything goes nicely for a few minutes, but then he begins to have burning of the nose and eyes and decides, very naturally, that his gas mask has stopped working; so he takes it off. Of course, the gas mask is working, but the effect of the initial brief exposure was delayed.

The further discussion of this group must be restricted to mustard, itself, as the type vesicant. One of the striking early symptoms after gassing, appearing in an hour or two, is hoarseness. It may be noticed even before conjunctival symptoms in those exposed. The reason for the hoarseness is obvious from the pathology. Slide 8 shows the larynx of a dog exposed to mustard vapor; and I think you can see that the tissues are puffy and inflammed. Slide 9 is a photomicrograph of a cross section of the trachea of a mouse one day after exposure to mustard. It shows strikingly the severe damage done to the respiratory passages by the gas. The epithelium is gone, except in patches, and a tremendous exudate and inflammation extends through the mucosa even into the cartilagenous rings of the trachea.

Slide 10 illustrates an exceptional aspect of mustard action but brings out the fact that the damage to the tissues is deep, penetrating and severe; and can lead to scarring. This is the trachea of a dog a month after gassing with mustard. The dog had recovered, but with a stenotic scar in the upper trachea following actual necrosis of the tracheal wall. Such a scar is rare except on the skin under certain conditions or in the eye; but its occurance in the regions of action of the vesicants in the respiratory tract indicates that the gas eats deeply and may do permanent damage there.

Slide 11 shows that mustard is essentially inactive on the lung parenchyma. It is a section of a mouse lung one day after exposure to mustard. You can see slight bronchial irritation, and a very slight injection of the alveolar vessels, a pneumonitis; but this clears up without edema or destruction. There are no immediate lung symptoms with mustard, rather more with Lewisite and with the nitrogen mustards, as you will hear later on.

Slide 12 shows, however, that there are important respiratory aspects of the vesicants. Although the lungs themselves are not directly damaged by the gas, gas injury to the upper passages often leads to aspiration of food and the like, as seen here, and so to

aspiration pneumonia. Slide 13 shows grossly an abscessed pneumonia in the lungs of a dog five days after exposure to mustard; and slide 14 shows the severe histopathology. Broncho-pneumonia is an important sequel of mustard gassing; in fact, it is the main cause of death after mustard. The other mustard actions produce casualties in troops or civilians for weeks or even months and they may, uncommonly, have permanent consequences in blindness or skin scars; but the mortality and long invalidism result from action on the respiratory system.

The vesicant action of these gases is to be considered later in detail,, but a few slides now will serve to illustrate the skin lesions. Slide 15 shows a typical mustard burn on the abdomen of a man, resulting from contact, through clothing, with contaminated rags. The clear fluid bleb and marginal erythema are characteristic. Slide 16 shows mustard blisters on the fingers, and slide 17 Lewisite blisters for comparison. In these the blister fluid is cloudy and the edges sharp with little red flare. Slide 18 shows a mild mustard burn of the face, an erythema outlining the position of a gas mask. This officer was sprayed with mustard from a plane, in a field test with mask and protective clothing. But in the hot weather perspiration carried some mustard under the mask edges - with the result you see.

The treatment of mustard burns, after cleansing, is that of other burns or open skin wounds; as the treatment of mustard pneumonia is that of any broncho-pneumonia. Because of danger of pneumonia, care against cross infection is important. The British recommend, under conditions where it is possible, as in civilian hospitals, that mustard casualties be treated as contagious disease cases and kept in isolation. Further consideration of treatment I leave to later speakers.

May I say one last word, however, about late effects. According to careful studies on casualties after the last war, there seems no justification for the prevalent belief in permanent effects of gas. Mustard cases may retain an injurious scar, such as you saw in the dog's trachea; perhaps an increased tendency to bronchitis; if pneumonia occurred in may leave the usual damage; and, of course, typical skin and eye lesions may be left, although most heal. There is no indication of an increased tendency to tuberculosis, although it is possible that tuberculous lesions may be activated by gassing. Many soldiers after the last war seemed to suffer permanent effects from mustard, such as photophobia and night cough. Most of these cases could rather regularly be cleared up without medical interference by "eye exercises" or deep breathing routines, and so seem to have been essentially psychiatric, with conversion symptoms. The handling is a psychiatric rather than a clinical problem.

Passing next to the respiratory irritants, I shall consider

primarily phosgene and diphosgene, which has an identical action although its different physical properties have important tactical consequences. (For example, there is an impossible discrepancy between American figures for deaths from phosgene, 2% of the casualties, and the German figures, 80%. Perhaps the Germans used more diphosgene which was less effective in building up high vapor concentrations.) By comparison, chlorpicrin and chlorine are more irritating and less toxic. Phosgene, if I may guess, would be as apt to appear over the Chicago area as any gas. It would come on a cold, still, clear winter night, when it might build up to an effective vapor concentration over a considerable area.

Phosgene breaks up over a matter of seconds or minutes on contact with aqueous media. It must, then, act promptly in the lungs after being inhaled, or the damage must be done by the acid liberated by its decomposition. Although we are told that hydrochloric acid, perhaps liberated intracellularly, causes injury, I doubt whether acid is very important. What the exact chemical mechanism of action is, although of profound academic interest and perhaps of importance in developing better means of handling cases, is of no consequence to the physician faced with a case of phosgene poisoning. The pathology and physiology of its action is important. As a result of chemical damage produced in the lung endothelium by phosgene there is an increase in permeability to blood plasma. Whole plasma leaks out through these now permeable membranes under the hydrostatic pressure in the pulmonary vessels, and edema sets in promptly.

That is a particularly unfortunate combination of local conditions. Not only is fluid being lost from the blood, resulting in hemoconcentration to a degree of increased viscosity and decreased blood volume which may seriously interfere with circulation through the body, but also the fluid that leaves the blood remains in the lungs where, like a pneumonia or any other type of pulmonary transudation, it interferes with the gas exchange through the alveolar membranes. Consequently, stasis anoxia and anoxic anoxia sum their effects on the body tissues -- which receive less blood carrying less oxygen per unit of blood.

Slide 19 shows the lungs of a goat exposed some ten or twelve hours earlier to phosgene. You can perhaps identify the foamy exudate, or transudate really, filling the trachea. Dr. Lushbaugh expects to autopsy some phosgene animals tomorrow and you will see the water-logged state of the lungs. Slide 20 represents a very typical phosgene lung, from a rabbit, with irregular patches of edematous consolidation and of emphysema.

Slide 21 shows, in contrast with the mustard series, that the upper respiratory passages are little, if at all, injured by phos-

gene. This is the larynx of a cat after a severe phosgene dose. All the epithelium is intact, a very slight sub-mucous edema is seen, and nothing else.

Slide 22 shows a section of a monkey's lung after phosgene. The tremendous damage -- with emphysematous patches, necrosis and congestion, and the whole full of edema fluid spreading out from the vessels -- is obvious.

Slide 23 is of a case of diphosgene poisoning, with the lungs still in situ in the dog. It shows that diphosgene gives the identical effects of phosgene itself; emphysematous blebs appear all over a congested and edematous lung.

The signs following are rather important because the early symptoms are not. An individual may be well along the course of developing a pulmonary edema, even a fatal one, without the physician being able to hear much on physical examination of the chest. There is perhaps a small increase of breath sounds in the axilla or back, and some rales may be heard, but even these are not at all commensurate with the pathology which has developed. Perhaps the most convenient laboratory test to use in following the course of the developing disturbance is the hematocrit. It may fall a little in the first four hours but subsequently, in all serious cases, it rises and may rise very much indeed -- over 50 per cent. Of course, by that time there is adequate clinical evidence of what is happening to the patient. A decreased blood oxygen, particularly venous oxygen, is obviously indicative of pathology; and a falling arterial pressure is a concommitant finding.

The phases of phosgene poisoning can perhaps be divided into four. First is a phase of acute reflex effects immediately after exposure. These are often mild or absent unless gas is inhaled in high concentration, and even then they soon pass off. Some shallow rapid respiration, slow heart, constriction of the chest, nausea and diarrhoea, are of importance mainly as evidence as to the actual exposure. Then follows the phase of so-called latent period, which may last for five, ten or fifteen hours. The patient may seem, during this interval, quite normal and over his trouble. Yet during this period obviously lung edema is developing. When sufficient edema is present so that the gas exchange is interfered with and so that the circulation is embarrassed in one way or another, cyanosis begins to set in. The patient then enters the blue stage (slide 24). His face is blue, neck veins are engorged, blood pressure is lowered, respiration is labored, frothy and perhaps bloody sputum may be raised, and the individual begins definitely to struggle for breath. It is then that oxygen brings such dramatic relief. If relief is not obtained, if the pathology does not begin to resolve, circulatory failure eventually supervenes. This is the gray stage (slide 25). It is a typical shock, except that the patient's face is a

greenish gray due to the concomitant asphyxia. The blood pressure drops away and the patient dies. Unhappily, in this typical shock condition, there isn't much use in treating the shock, because the plasma leak remains. Fluid introduced into the body simply comes out again into the lungs and exaggerates the pulmonary problem of gas exchange.

In the blue stage, bleeding is widely recommended. It was emphasized in the memorandum I read you from Norris, and is also advised in the current war manuals. I doubt that it is as useful as is generally believed.

While I am indulging in personal medical heresies let me also express one other bit of skepticism, which may be of very great practical import. All present instructions about handling phosgene casualties emphsize the need of immediate and absolute rest. This does not seem entirely logical to a physiologist. It is perfectly true that when fluid has accumulated in the lungs so that gas exchange is interfered with, the oxygen need of the body must be minimized and exercise avoided. But during this early period, of a few minutes to a few hours after exposure, if no considerable lung edema is present, I can't see that the amount of exercise involved in walking from the point of exposure to a proper treatment center will do the patient any damage. And it makes an enormous difference in the organization of the medical services whether every gas case or suspected gas case is to be treated as a stretcher problem or is allowed to ambulate for further attention.

The late effects of phosgene are rarely serious. If the patient doesn't die in a day or so he is likely to recover completely, unless he gets a secondary pneumonia with its consequences. Slide 26 shows the lungs of a dog sacrificed five days after a severe phosgene gassing. As you see, they appear quite normal. Asthma at night and D.A.H. (disordered action of the heart) have been seen with some frequency, but the medical evidence again seems to place these problems in the psychiatric field.

Finally, may I say a few words on the problem of diagnosis. It is well to realize that the doctor faced with a case at a gas casualty station has to make three diagnoses; or has to answer three questions. The first one is: Was the individual gassed? The answer is of importance both medically and tactically. When certain that the patient was gassed, the second question is: Which of the known gases was it? But don't forget that there is a third question, which the physician particularly should keep in mind: Is this a new gas? When symptoms and history are atypical, the alert doctor seeing immediate casualties is about as likely to first get evidence of a new gas as are the special gas detecting agents or the control office.

There are certain points to keep in mind in making the usual

differential diagnosis. The history is of especial importance. Obvious points are: just when was the exposure; how long was the exposure; what were the conditions -- was exposure in a closed room or outdoors, was there a big explosion or a little one, were the eyes open or closed while sleeping, (the eyes being closed during exposure may confuse the symptoms), was a gas mask worn, and so on. But one of the most important things for the physician to ascertain is character of the immediate symptoms. He must note the positive ones, which are reported and, by careful inquiry, make sure those not reported did not occur. Was there an immediate irritation of the eye or wasn't there? It may be a crucial diagnostic point -- severe immediate irritation is probably due to one of the harassing irritant gases and not likely to be serious in the long run.

The British Medical pamphlet to which I referred you has a definite system of differential diagnosis outlined, depending on the parts of the body involved. If the skin is attacked you are dealing with a vesicant, whatever else there may be. These are then differentiated: reddening without blisters probably excludes mustard or Lewisite, it may be one of the irritant drugs. Mustard gives a blister with a red flare and clear fluid, Lewisite a blister with little flare and turbid fluid. If the skin is not involved the next question is: "Are the eyes involved?" If they are, you are dealing again either with one of the vesicants or with a lacrimator. If the eye effect was immediate and acute, one of the harassing irritant gases was responsible, and there is little to worry about. If the effect was delayed, a dangerous vesicant action must be guarded against. If, whether or not the eyes are involved, there are no respiratory symptoms all the lung irritant gases can be eliminated. (Some lung irritant gases, especially chlorpicrin, do produce some eye irritation.) If there is no eye irritation and no respiratory symptoms have appeared but the patient is sick, the case is one of systemic poisoning. With no eye or skin symptoms but with respiratory effects, you are obviously dealing with one of the lung irritants. And let me remind you that harassing gases act highest in the respiratory tract, vesicants lower, and pulmonary irritants most deeply in the lungs. This general scheme of differential diagnosis is a handy one to have in mind.

One last comment: I think you can be pretty sure that when a supposedly gassed individual comes to your attention, the seriousness of the case is likely to be inversely related to the intensity of the initial symptoms. The most acute onsets have to do with the second order nuisance gases, or to be sheer panic. When irritation is intermediate and slowly developing, the vesicant group is indicated; and when there was little or no initial effect but late symptoms begin to appear, suspect the respiratory irritants. The whole situation is a bit like that of the ham actor who had to climax the great scene in a play, when the villian shot him, by clutching his hand over his heart and crying, "My God, I'm shot."

Since he didn't manage to get expression into his cry during rehearsals, a fellow actor put some red ink in the squirt pistol used in the actual performance. The play went on, the climax reached, the gun shot, and the uninspired "My God, I'm shot." As the ham drew his hand away, he saw the red and exploded vehemently, "Jesus Christ, I am shot." Patients who feel that way about their gassing are likely to be as easily cured.