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THE CLINICAL FEATURES OF MUSTARD-GAS POISONING IN MAN

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The occurrence of general systemic symptoms following exposure to heavy dosages of mustard vapour or to massive liquid contamination was well recognized in the 1914–18 war. Since then experimental work on the subject has been largely concerned with the pathological changes observed in animals following the administration of mustard gas by different routes. These investigations have been reviewed by Smith (1943), who gives a comprehensive account of the literature.

In the absence of any large body of human experimental material, investigations on the changes in the haemopoietic system and blood chemistry of animals have tended to relegate to the background the clinical symptomatology produced in man by exposure to mustard gas. Isolated cases of accidental exposure have occurred from time to time, and some have been reported in the medical press (Hobbs, 1944). In these also, however, attention has been focused on the pathology rather than on the symptoms, many of which are nevertheless of the greatest importance, since their occurrence in the case of mustard-gas burns throws light on the mechanism by which similar symptoms arise following thermal burns.

It is difficult to demonstrate the occurrence of systemic manifestations in cases of slight or moderate mustard burns in temperate climates. In the Tropics, however, systemic poisoning by mustard gas is much more readily produced. From 1943 to 1945 I had the opportunity of observing a series of 438 volunteers exposed to mustard gas under tropical conditions, and thus of becoming familiar with the clinical picture of systemic mustard-gas poisoning.

Material and Methods

Of the 438 men observed 320 were exposed to mustard vapour and 118 to contamination by liquid mustard. In both the vapour and liquid groups the resultant burns were of all degrees of severity, ranging from a faint erythema of a localized area of skin to multiple ulcers many hundreds of square centimetres in area. All the men exposed to vapour and 77% of those exposed to liquid wore fully effective respirators throughout exposure.

The occurrence of systemic poisoning in these men was assessed both objectively, by daily clinical examination for a minimum period of 21 days after exposure, and subjectively, by daily interrogation. Each man was examined separately, and was asked only, "How are you feeling to-day in yourself?" No leading questions or suggestions were used.

Not all the symptoms of which the volunteers complained were easy to evaluate. Great care, however, was taken in each case to exclude adventitious causes, such as indulgence in the local mixture of lower alcohols, and to sort out genuine complaints from those which might be regarded as suspicious. In general, it was impossible to assign any accurate degree of severity to the subjective clinical symptoms owing to the variation in the general character and education of the volunteers. It is felt that considerable confidence can be placed in the findings, since they were recorded in men classified as A1 who were in good physical condition and well acclimatized to the Tropics. Further, the men had all volunteered to be exposed to mustard gas, and showed the keenest interest in the work.

Owing to lack of time and facilities it was only possible to follow blood changes in selected groups of men. The clinical pathology of some of the severely affected men was more extensively investigated.

Observations

Threshold and Severity

No symptoms attributable to systemic poisoning occurred in any vapour case in which no lesion more severe than erythema developed. There were 84 such men. Similarly, no man with liquid burns in whom the total raw surface ultimately produced was less than 20 square centimetres in area complained of systemic symptoms. In this category 83 men were included. Of the remaining 271 men, 102 reported one or more symptoms.

The severity of the effects varied within wide limits. In 35 men systemic manifestations were present on one day only, and in most of these cases the symptoms were in no way incapacitating. At the other extreme were cases in which systemic symptoms were a major factor in producing prolonged disability. In 37 men never more than one symptom was present, while the remaining 65 complained of two or more symptoms at various times. In general, the latter group included the more severely affected men.

Time Factors

Of the 102 men affected 66 (65%) showed symptoms within 24 hours of exposure; 83 (81%) were involved within 48 hours and 96 (94%) before the sixth day. It is also of interest that only 3 (16%) of the 19 cases in which the onset of symptoms occurred later than 48 hours after exposure could be described as more than very slightly affected. In contrast the onset of systemic symptoms in some of the worst cases preceded even the appearance of erythema of the skin.

Fig. 1 shows the number of men complaining of one or more systemic symptoms on each day after exposure. The number is largest on the first day, and falls fairly regularly till the end of the second week. No complaints were received from any man after the forty-fifth day. It is interesting that there is no correlation between the number of men complaining of systemic symptoms and the severity of the skin burns. Characteristically, the latter developed slowly, and in the vapour cases did not reach their maximal development until, on the average, 11 days after exposure. This time appeared to be independent of the severity of the lesions (Sinclair, unpublished work,

1944). The time taken by the liquid burns to attain their maximum varied considerably, but in general was somewhat shorter. Considering the group as a whole, there was a steady rise in the extent and severity of the skin lesions

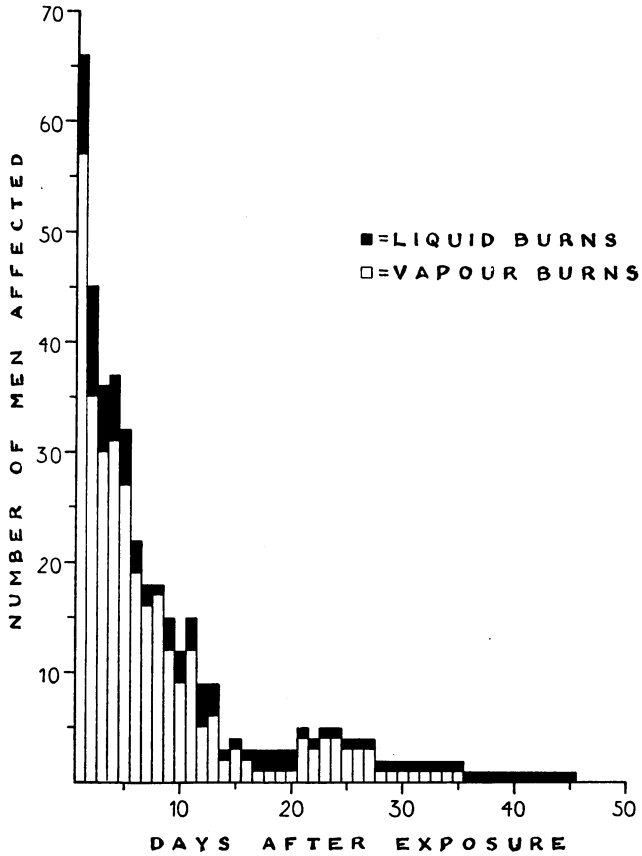

FIG. 1.—Distribution of systemic poisoning in time.

until the eleventh day, whereas during this time there was a steady fall in the number of systemic symptoms recorded.

Symptoms and Signs

Table I sets out the various symptoms observed in this series. It is evident that most of them might be produced by any state of general intoxication, and are not in any way peculiar to mustard-gas poisoning. Two rather more unusual signs—tremor and tachypnoea—were restricted to severely burned cases. Table I also shows, for each symptom, the percentage of the total number exhibiting systemic symptoms who complained of that symptom. It will be seen that there is a striking agreement in these percentage figures between the vapour and the liquid cases, thereby indicating that the overall clinical picture of each group of men was similar in spite of the differences in the type of skin lesion and its mode of production.

TABLE I.—*Relative Incidence of Symptoms*

Symptom	Vapour Cases		Liquid Cases		All Cases	
	No. of Men Complaining	Percentage Complaining (out of 86)	No. of Men Complaining	Percentage Complaining (out of 16)	No. of Men Complaining	Percentage Complaining (out of 102)
Nausea	50	58	11	69	61	60
Headache	36	42	6	38	42	41
Lassitude	33	38	6	38	39	38
Insomnia	26	30	5	31	31	30
Vomiting	16	19	3	19	19	19
Anorexia	14	16	5	31	19	19
Abdominal pain	13	15	1	6	14	14
Diarrhoea	9	10	2	13	11	11
Tremor	5	6	2	13	7	7
Vertigo	6	7	0	0	6	6
Tachypnoea	4	5	1	6	5	5
"Anxiety state"	1	1	2	13	3	3
Total no. complaining of one or more symptoms	86		16		102	

TABLE II.—*Distribution of Individual Symptoms in Time*

Period	No. of Complaints of											Total
	Nausea	Vomiting	Anorexia	Abdominal Pain	Headache	Lassitude	Insomnia	Tachypnoea	Diarrhoea	Vertigo	Tremor	
First day	41	10	3	3	18	12	4	5	1	4	6	107
Remainder of first week	82	7	17	18	37	47	53	—	11	1	9	282
Second week	15	7	17	14	12	11	24	—	3	1	7	111
Third week	2	—	14	2	3	1	—	—	—	—	7	29
Fourth week	5	—	12	15	6	—	—	—	1	—	7	46
Fifth week	—	—	7	7	—	—	—	—	—	—	7	21
Later than fifth week	—	—	—	—	—	—	—	—	—	—	10	10
Total	145	24	70	59	76	71	81	5	16	6	53	606

shows the number of complaints made of each symptom during different periods after exposure. A consideration of this table shows, for example, that on the day after exposure nausea and vomiting between them accounted for 47.7% of the total number of complaints made on that day, whereas complaints of anorexia and abdominal pain taken together accounted for only 5.6% of this total. Thereafter, however, there is a gradual decrease in the proportion of complaints due to nausea and vomiting, while the proportion due to anorexia and abdominal pain steadily rises with time. Other symptoms show proportional changes which are less striking. Thus the percentage of total complaints due to headache remains fairly constant, and complaints of lassitude and insomnia are practically restricted to the first two weeks.

It may therefore be said that in the first week following exposure the most characteristic complaint was of nausea with or without vomiting, whereas after the beginning of the third week anorexia and abdominal pain accounted for the greater part of the complaints made.

To evaluate the relative frequency with which any given symptom "presented," a simple marking system was adopted. One mark was allotted to each man, and if there was only one presenting symptom the whole mark was entered under this symptom. If several symptoms presented simultaneously the mark was split proportionately between them. By this procedure it was found that nausea with or without vomiting was the commonest presenting symptom, receiving 38.3 marks. Headache and lassitude each received 22.8 marks, and all the other symptoms together accounted for the remaining 18.1 marks.

Individual Symptoms and Signs

Nausea and Vomiting.—Of all the symptoms which have been recorded in man after exposure to mustard gas nausea and vomiting are the commonest (Norris, 1918; Moorhead, 1919; Wilson and Mackintosh, 1920; Soltau and Elliott, 1923; Aitken, 1943; and others). Vomiting is also frequent in experimental animals (Lynch *et al.*, 1918; Warthin and Weller, 1918; Smith, 1943). It has already been noted that nausea was the commonest individual symptom of the present series. A characteristic feature of the nausea was the occurrence of intermissions. Thus one man was nauseated on the first day, and then had no symptoms of any kind until the sixth day, when he had an attack of

severe nausea lasting four days. The onset of this attack preceded by two days a generalized outbreak of vesication. The complaints of nausea recorded in Table II in the fourth week after exposure were received from a group of men exhibiting abdominal symptoms in whom nausea accompanied the other complaints.

Vomiting was always preceded and accompanied by nausea. In the worst case there was repeated vomiting and retching over a period of 48 hours. It will be noted that in Table II vomiting is recorded several times during the second week. The circumstances of such delayed vomiting were carefully examined, but it was necessarily difficult to exclude factors other than the effects of mustard. It is possible that in some instances the vomiting may have a partly functional origin (Wilson and Mackintosh, 1920; Soltau and Elliott, 1923).

Headache.—Headache of the type described by Moorhead (1919), Soltau and Elliott (1923), and Aitken (1943) was a feature of many cases. It was characteristically frontal, continuous rather than remittent, resistant to medication, and of a peculiarly unpleasant nature. In some of the worst cases headache was responsible for a considerable amount of disability. For example, one man complained of intense headache from the first to the third day, sufficient to make him cry out and to necessitate his admission to hospital on the fourth day. The headache disappeared on the seventh day, but from then until the fifteenth day intermittent nausea, vomiting, vertigo, insomnia, and anorexia were present. On the twenty-first day the headache reappeared in an even more severe form than before, and lasted until the twenty-seventh day. This man suffered from mustard burns which were only moderate in degree, but it was necessary to retain him in hospital solely because of his systemic manifestations. It is probable that he represents an unusual degree of sensitivity to mustard-gas poisoning.

Lassitude.—Lassitude has been noted as a feature of mustard cases by Moorhead (1919), Wilson and Mackintosh (1920), and Aitken (1943). In the severe cases in this series the men were most unwilling to do anything more in their spare time than lie down and try to sleep. This finding has the more weight since it was recorded on Australian fighting troops in the absence of pain or severe burns. It might be expected that the occurrence of lassitude would run parallel to that of insomnia, but in only 12 of the men were lassitude and insomnia both present, and in only 5 of these were the two symptoms recorded simultaneously. It is evident, therefore, that the lassitude could not be attributed solely to loss of sleep.

Insomnia.—Before attributing insomnia to the specific action of mustard gas it is necessary to exclude the factors of pain, irritation, or itching arising from the skin burns. It is probably for this reason that insomnia has rarely been mentioned as a symptom of mustard poisoning (von den Velden, 1921). Nevertheless, in their initial stages mustard burns are relatively painless (Soltau and Elliott, 1923). In 12 of the 16 more severely affected men the onset of insomnia occurred before the third day, and it is noteworthy that none of these men advanced the reason of discomfort as the cause of his complaint. It is possible, however, that insomnia occurring later was, at least in part, due to pain or irritation.

Anorexia and Abdominal Pain.—Reference to Table II shows that complaints of loss of appetite occurred not only in the first few days, as might perhaps be expected in any series of skin burns, but also as late as the fourth and fifth weeks after exposure. At the beginning of the fourth week a condition supervened in 4 men which was characterized by anorexia, abdominal pain, and occasional nausea. Abdominal pain was also recorded in the early stages (Table II), but was usually not so severe as in the delayed cases. In the literature epigastric pain appears frequently as an early and acute symptom of mustard-gas poisoning (Mandel and Gibson, 1917; Moorhead, 1919; Soltau and Elliott, 1923; Chiesman, 1944), but its occurrence in the later stages does not appear to have been mentioned. Hobbs (1944) has described ulceration of the duodenum in a fatal case of mustard-gas poisoning, and the nature of the delayed pain observed in this series is of interest in this connexion. It occurred from half an hour to an hour and a half after meals, and lasted for a few hours. It was usually a steady ache, situated in the epigastrium and accompanied by a diffuse epigastric tenderness. It was not relieved by food, but could be alleviated by alkali; it did not interfere with sleep. Flatulence and heartburn were not observed, and the pain bore no relation to the type of food consumed.

Diarrhoea.—Diarrhoea was in all cases mild and of short duration. Both Soltau and Elliott (1923) and Wilson and Mackintosh (1920) are emphatic that diarrhoea in gassed cases in the 1914–18 war was due to associated conditions rather than to mustard-gas poisoning. Moorhead (1919) mentions diarrhoea of a dysenteric type, but states that constipation was the rule. Nevertheless, diarrhoea is a prominent feature in experimental animals (Lynch *et al.*, 1918; Smith, 1943), and it has been regarded as a leading symptom of systemic intoxication by mustard gas (Smith, 1943). In this series detailed records were not kept of controls, but it may be said that diarrhoea was certainly not more common in the volunteers than in the camp staff, who were living and working under similar conditions.

Tremor.—The occurrence of tremor has been described in experimental animals following the intravenous injection of mustard gas (Warthin and Weller, 1918), but, except for one case mentioned by Wilson and Mackintosh (1920), does not appear to have been particularly noted in man. In this series it was restricted to severely burned men, in whom it appeared within a few hours of exposure. In all the vapour cases affected it involved the whole body, especially the hands, and at its worst resembled well-marked shivering. The men did not, however, complain of feeling cold, and the body temperature was in all cases normal. The tremor was coarse and showed frequent remissions; it was not under voluntary control. The deep reflexes were invariably exaggerated, but examination of the central nervous system disclosed no other abnormalities. In all cases the tremor disappeared within 48 hours. In one man with liquid burns an exactly similar tremor was noted on the second day after exposure. In another a coarse tremor of the hands and fingers was present throughout his stay in hospital (45 days). This man had severe multiple third-degree burns in 30 distinct areas. These areas were such as to preclude any testing of the usual deep reflexes in either legs or arms. He was, however, extremely unsteady and incoordinated. He became nervous and excitable, and his mental condition during the first few weeks approximated to an anxiety state. Examination of the central nervous system was negative, and by the time his burns had healed adequately the deep reflexes were normal.

Vertigo.—Vertigo has been noted by Aitken (1943) in vapour cases, and was present in six vapour cases in this series. It did not occur apart from nausea, and never lasted longer than one day.

Tachypnoea.—In four of the five vapour cases with tremor tachypnoea began within a few hours, at the same time as erythema appeared. Nine hours after exposure the resting recumbent respiratory rate had risen in all four to 30 per minute or over, and in one case to 40 per minute.

Breathing was in all cases shallow and fairly regular, and there was no evidence of dyspnoea. Twenty-four hours after exposure there was no disturbance of respiration in any man.

One of the men severely burned by liquid also complained of considerable "breathlessness" during the first 24 hours, even while lying in bed. It is interesting to note that an increased respiratory rate has been reported in goats following the contamination of the skin by 40–50 mg./kg. of liquid mustard (Allen, Cameron, Coles, and Rutland, unpublished work, 1944). These authors state that the respiratory rate "may reach high levels between the sixth and twelfth hours." Similar results were reported by Lynch *et al.* (1918) in dogs. It is therefore probable that the tachypnoea seen in human cases is due to a direct action of absorbed mustard derivatives rather than to any non-specific cause connected with skin damage. Both Mandel and Gibson (1917) and Norris (1918) mention rapid breathing as an early symptom in mustard-gas casualties, but in the men they describe it is apparent that protection of the respiratory tract was inadequate, and that the rapid breathing was the first sign of bronchitis. Moorhead (1919) and Aitken (1943) note the occurrence of pain in the chest, probably from the same cause.

Anxiety State.—Reference has already been made to the occurrence of a well-marked anxiety state in one of those severely burned by liquid mustard. Two other men, both badly burned, showed a very similar condition for the first two weeks after exposure. All three were "jumpy," irritable, and restless, and were afraid that they were going to die. In all cases recovery was rapid once the lesions had demonstrably started to heal. These men presented a marked contrast to the tired listlessness characteristic of many others in this series. Restlessness and irritability are described by von den Velden (1921), while Wilson and Mackintosh (1920) mention a case in which "mental excitement" was prominent.

Temperature : Pulse : Blood Pressure.—Cases of marked toxaemia were sometimes accompanied by an irregularly raised temperature, which did not exceed 101° F. (38.3° C.). This was not associated with sepsis, which was very uncommon in this series. The pulse rate in such men was correspondingly increased. Elevation of the pulse rate on the first day usually appeared to be due to the effort of retching.

In Moorhead's series (1919) the blood pressure was often low, while in Aitken's cases it was slightly raised. In four cases of liquid poisoning in this series there were signs of surgical shock, accompanied by evidence of haemoconcentration and a rise in the blood non-protein nitrogen, but in none of these could the blood pressure be satisfactorily taken owing to the position of the burns on the arms and thighs.

Clinical Pathology

One of the most characteristic effects of severe intoxication by mustard gas is a gradual fall in the number of circulating leucocytes, with concomitant changes in the bone marrow (Needham, Cohen, and Barrett, 1947). In fatal cases a condition amounting to aleukaemia may precede death (Hobbs, 1944). In none of the men in this series was there any definite evidence of a leucopenia, but in some of the severely burned cases there was a statistically significant temporary fall in the ratio of neutrophil polymorphs to lymphocytes. A polymorphonuclear leucocytosis, attributable to the presence of raw skin surfaces, occurred in many cases of vapour poisoning about the end of the first week and lasted for about ten days.

Moorhead (1919) observed that in mustard-gas poisoning the coagulation time of the blood was often markedly

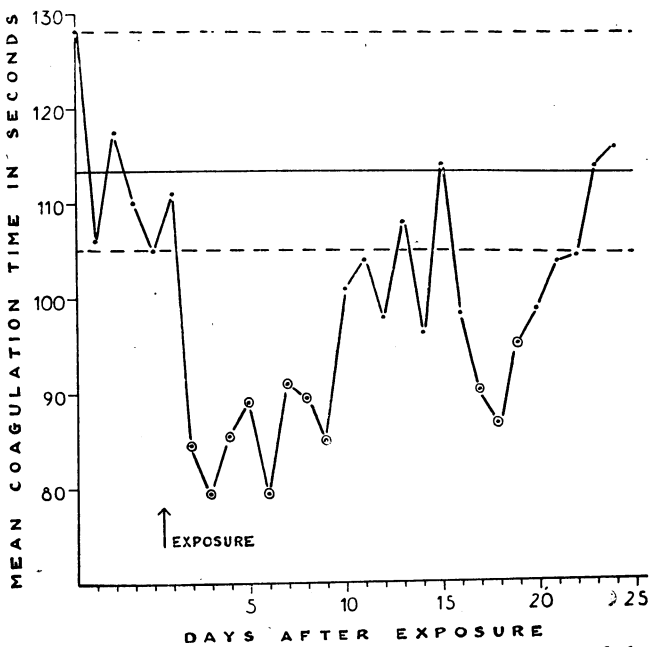

FIG. 2.—Effect on blood-coagulation time. The course of the mean blood-coagulation time in 10 men exposed to mustard vapour. The day of exposure is indicated by an arrow, the limits of the pre-exposure values by interrupted lines, and the mean pre-exposure value by a continuous line. Points on the graph showing statistically significant departures from this mean are circled.

decreased. This observation was confirmed in a number of vapour and liquid cases in this series. Fig. 2 shows the course of the mean coagulation time for a group of ten men exposed to mustard vapour. It was unfortunately not possible to investigate the cause of this rapid coagulation of the blood.

In view of the occurrence of "abdominal" symptoms, and of the known association of peptic ulcers with mustard-gas burns (Hobbs, 1944), an investigation of the total and free acidity of the gastric contents was carried out by Thompson (unpublished work, 1945) on a number of men in this series. He concluded that "under the conditions prevailing . . . exposure to mustard gas gives rise, in a proportion of cases, to a significant increase in the secretion of acid by the gastric mucosa." The rise in acidity was not immediate, and the maximum figures tended to occur about the eleventh day. No investigations on the gastric acidity of the men complaining of abdominal symptoms in the fourth week were carried out.

Even in the most severely burned men in this series no abnormal changes could be demonstrated in the sedimentation rate, the platelet count, or the composition of the urine.

Discussion

Lynch *et al.* (1918) were among the first to suggest that many of the symptoms observed in men gassed by mustard were due to the action of mustard gas as a specific systemic poison. At the time it was believed by many that the effects were caused solely by the local tissue damage. Thus Soltau and Elliott (1923) state that "vomiting . . . is due to the swallowing of saliva or nasal secretion which has been infected by mustard gas"—an explanation which was also accepted by Aitken (1943). Warthin and Weller in 1918 found "no evidence of any systemic poisoning by the absorption of dichlorethyl sulphide from the skin, eyes, or mucous membranes of the respiratory or gastro-intestinal tracts." Subsequent work, however, has left no doubt that mustard gas has a specific toxicity, manifested particularly by its effect on the haemopoietic tissues, irrespective of the route by which it is administered (Smith, 1943).

In 1943 work on the exposure of human volunteers in Australia showed that severe systemic symptoms could be produced despite the fact that fully effective respirators were worn throughout exposure. This condition also applies to all the cases reported in this paper, with the exception of five of the men exposed to liquid contamination, who wore eye-shields instead of respirators. The only possible channel of entry for mustard derivatives into the body was thus the intact skin. It follows that the symptoms and pathological changes observed can only be due either to the action of derivatives of mustard circulating in the blood after having been absorbed through the skin or to the absorption of non-specific metabolites from the damaged skin.

The latter explanation cannot be wholly neglected. Gibson and Brown (1944), reporting on shock following thermal burns, state that vomiting was frequent and often severe. Further, many of the symptoms occurring in the cases exposed to mustard are, by reason of their general character, difficult to attribute solely to the action of mustard derivatives. Nevertheless it is probable that the observed picture is for the most part due to this cause, for several reasons:

1. It has been shown that the systemic symptoms in experimental animals exposed to mustard vapour or to liquid skin contamination can be almost exactly reproduced by the intravenous injection of pure mustard or derivatives of mustard (Smith, 1943).

2. In this series the symptomatology of vapour and liquid cases was exactly similar in spite of the marked differences in the type of skin burn produced.

3. In the present series 65% of the men affected became so within 24 hours, and in very few of these was the degree of skin damage greater than an erythema by the end of this time. Indeed, in some cases the onset of nausea preceded the appearance of erythema. It is thus difficult to explain the symptoms solely on the basis of the liberation of toxic metabolites by skin damage. Further, in the group as a whole the severity of the skin lesions showed a progressive increase until the eleventh day, whereas during this time the number of systemic symptoms steadily fell.

4. The leucopenia characteristic of severe poisoning by mustard gas, however administered, is not produced by other, non-specific, substances injurious to tissues.

It is reasonable, therefore, to regard most of the findings in these men as being due to the specific action of mustard. There is one group of symptoms, however, which may be in part due to a non-specific mechanism. Peptic ulcer is a well-recognized complication of thermal burns (Harkins, 1938), and it seems possible that the association of abdominal pain, anorexia, and nausea which occurred in a few of the men during the fourth week after exposure was in some way related to the skin damage rather than to mustard poisoning. The number of cases examined by Thompson (1945) is too small to show any definite relation between symptoms referable to the gastro-intestinal tract and the appearance of hyperacidity, but the findings merit further investigation.

The mechanism by which the mustard derivatives which have been found in the blood stream of experimental animals act to produce clinical manifestations is obscure, except that there is a specific toxic effect on the bone marrow (Needham, Cohen, and Barrett, 1947). It is possible that many of the apparently diverse symptoms produced in man have a common pathological origin. It must be pointed out that the results in this series show that systemic intoxication in both liquid and vapour cases occurs much more readily in the Tropics, probably owing to a generally enhanced power of the skin to absorb mustard gas (Cullumbine, 1948).

Not all the symptoms and signs observed had the same practical importance with reference to disability. In this respect the incapacitating potentialities of nausea, vomiting, and headache may be emphasized. The necessarily limited clinical pathological investigations in this series showed that, although changes in the leucocyte count and in the blood coagulation time were noted, these changes were in no instance such as to cause concern, and did not affect the clinical course of the cases.

Summary

An account is given of the various clinical symptoms and signs encountered in 102 cases of mustard-gas poisoning observed in the Tropics.

These symptoms followed the absorption of mustard gas through the skin, and were in great part the result of a specific toxic action of mustard derivatives.

Severe systemic manifestations were produced in some instances, although the blood changes found were in all cases clinically unimportant.

The incidence of systemic symptoms was greatest on the day after exposure, and fell progressively thereafter.

The commonest individual symptom was nausea, in the more severe cases accompanied by vomiting. The possible relation of abdominal symptoms to a disturbance of gastric secretion is discussed.

Potentially incapacitating symptoms occur in the Tropics very much more readily than in temperate climates.

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The Board of Trade reports that the President of Finland has submitted a Bill to the Diet proposing the establishment of a Government Company for the importation and distribution of medicinal preparations on a wholesale basis. The share capital of the company is to be 100 million Finmarks, but can be raised to 300 million Finmarks. The Government will hold all shares with the exception of two, which could tentatively be transferred to the Public Pension Board and the Helsinki University. In the preamble to the Bill the Government states that as long as the importation and distribution of medicinal preparations are solely in the hands of profit-seeking private companies there is always the danger that prices will be too high. It is therefore essential that the trade should also be conducted by a company whose objects are entirely confined to promoting the public welfare. As the Government company will sell the products at the lowest possible prices it will force the competing firms to make corresponding reductions in prices. The new company will also handle the distribution of medicinal products to the State and municipal hospitals.