MUSTARD GAS LESIONS OF THE EYES

BY

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Interest in the lesions caused by dichlorethyl sulphide (mustard gas) is again aroused by the possibility of its use in the future. The first "gas attack" was made by the Germans at Ypres in 1917, and a number of ophthalmic surgeons became conversant with the immediate results of doses of varying severity. Owing, however, to the difficulty of accurate observation under Service conditions in the last war, and also to the fact that in most cases spontaneous recovery occurred after a variable interval, few detailed data of the clinical pathology and total course of the lesion were available. Interest in the subject dropped, though occasional case histories and speculations as to after-effects appeared between 1920 and 1935. After that period, however, it became clear that cases of recurrent ulceration were occurring among men subjected to relatively heavy doses of mustard gas ten to twenty years previously. Thus the condition of "delayed mustard gas keratitis" came to be recognized as a clinical entity (Phillips, 1940). It is now possible, by correlation of the appearance of these delayed cases with lesions induced in animals in a series of experiments carried out at the Imperial Cancer Research Fund Laboratories and with other human material available at the present time (e.g., in laboratories), to piece together the story of the effects of mustard gas on the human eye (Mann and Pullinger, 1942).

In the first place one can say that these effects depend on the peculiar physical and chemical properties of the gas, on the anatomical situation at which it acts, and on the size of the dose. There is no essential difference other than that of degree between the effects of the vapour and of a splash of the liquid, since very prolonged exposure to vapour will cause damage exactly similar to that produced by liquid. We can therefore recognize grades of the lesion of varying severity, dependent on the size of the dose and characterized by the involvement of certain tissues in a definite order. Broadly, it can be said that the lids and conjunctiva are involved first, that with a slightly larger dose the cornea will show changes—first in its epithelium only and; with a greater dose, in the substantia propria as well—and that with severe doses the limbus is damaged and vascularization of the cornea occurs to a varying degree. It should be of use, in view of the possibility of gas attacks in the present war, to summarize these clinical varieties of lesion, since if they are recognized the prognosis in any given case is rendered much easier, and the sorting out and disposing of cases is facilitated in consequence.

Varieties of Lesion

1. *Cases in which the Conjunctiva and Lids alone are involved, the Cornea remaining Clear Throughout.*—These cases run a mild course, as the total gas received by the eye is very small. There is no complaint of discomfort at the time of exposure, but about six hours afterwards there is burning and smarting of the lids, with some lacrimation. The lids are swollen and reddened and the conjunctiva is hyperaemic. The eyes can be opened voluntarily and there is no complaint of photophobia. The cornea is bright and sight is not affected, so that, except for the discomfort, there is no disability. Such cases clear up spontaneously in four to seven days and do not relapse. It is likely that a number of cases would be of this mild type. In the absence of any known specific antidote it would seem that treatment of these mild cases should be expectant.

2. *Cases in which the Lids, Conjunctiva, and Surface Layers of the Cornea are involved.*—There is no discomfort at the time of exposure and, as in the first group, the complaint is of redness, swelling, and burning of the lids about six hours after. In addition there are lacrimation and photophobia. The presence of photophobia six to nine hours after exposure can, even in the absence of facilities for detailed examination, be taken as a sign of corneal involvement (either superficial or deep), and therefore calls for greater care in treatment and prognosis. If the eye be examined in detail it will be found that, although much swollen, the lids can usually just be opened, when the cornea in the palpebral aperture will be seen to be very faintly hazy, with superficial oedema and epithelial bedewing. There may be a fine punctate staining with fluorescein. The conjunctiva is very hyperaemic. The patient tends to keep the eyes closed for four or five days and then begins to improve. The swelling, hyperaemia, and discomfort may continue for as long as six weeks, but recovery is complete, with no relapses. The patient at the beginning is a casualty, since, though not totally incapacitated, he is unfit for any activity. As the condition here is essentially a superficial keratitis it would seem logical to treat with a mydriatic. Excessive treatment, such as repeated irrigation, is to be avoided. The superficial tissues being somewhat devitalized, any irritant treatment will tend to retard healing, and measures should be restricted to the relief of ciliary spasm and the removal of discharge, if any. Discharge is seldom a marked feature, and secondary infection in these cases is rare. Bandaging, if used at all, should be given up after three days at most. Reassurance is also required, as the patient is apt to think that his sight is severely involved.

3. *Cases in which the Whole Thickness of the Cornea is involved Together with the Limbal Blood Supply.*—These cases are severe and will be total casualties for from two months to a year or more, and in the worst cases for much longer. Even here there is no complaint of discomfort at the time of exposure. Indeed, I have examined one man who in the last war received a splash of liquid mustard and, beyond the sensation of fluid entering the eye, experienced no immediate disability and was able to see for nearly ten hours afterwards. It is important to recognize this in assessing prognosis.

The usual history is of severe exposure, followed in a few hours by intense oedema of the lids with burning and smarting and complete inability to open the eyes. If an examination can be made it will be seen that the conjunctiva is hyperaemic and there are haemorrhages in it. At the limbus on each side in the palpebral aperture there are avascular white necrotic patches in which the conjunctival and limbal vessels are destroyed. There is a band of dense oedema across the cornea in the exposed part. This has a "peau d'orange" surface and stains with fluorescein. The destruction of limbal blood vessels at once places the case in the severe category, but only continued observation will show how severe it is likely to be. The subsequent possible stages are:

(a) The lids may remain closed for five days and then the primary oedema of the cornea may subside. Apart from some activity of the limbal loops above and below the triangular white patches at the limbus, vascularization may not occur. The condition then subsides in about two months and does not relapse, though the triangular areas of destruction of limbal capillaries are permanent. These cases show that destruction of limbal capillaries is not enough to lead to vascularization and to prevent recovery of the cornea.

(b) If the dose received is larger the eyes may remain closed up to seven weeks, with profuse discharge and intense discomfort. There will be an accompanying iritis, and synechiae

may form if a mydriatic is not used. After the subsidence of the primary oedema of the cornea in three to five weeks, a secondary oedema makes its appearance accompanied by ingrowth of vessels into the cornea from the undamaged region of the limbus contiguous to the triangular white patches of conjunctival destruction. These vessels assume characteristic and fantastic shapes; they show pointed ends, right-angled branches, intracorneal haemorrhages, sudden alterations of calibre, and constrictions cutting off the tips of the vessels, which may form blood islands just under the epithelium, also often of bizarre shapes. These vessels extend into the cornea, the secondary oedema spreading ahead of them. This process may continue for eight weeks or more and may then begin to subside, the vessels narrowing and often in course of time disappearing. A faint nebula is left in the substantia propria, the texture of which appears to be altered, but no further change occurs.

(c) In more severe cases, after the initial vascularization has cleared up, there is a relapse within a few weeks; the secondary oedema reappears, more vessels enter the cornea, and the same process of intracorneal haemorrhage, constriction of vessels, and regression occurs. These repeated relapses may continue for a year or more and then cease. Such an eye is left with a fair amount of sight but is in an unstable condition. As the intracorneal haemorrhages subside and the vessels retrogress they leave behind them a scarred area containing intracorneal deposits of fat and cholesterol. This stage of haemorrhages, retrogression of vessels, and degenerative deposits is symptomless and may continue for years. As the sight is fair (6/9 usually) these patients make no complaint, and numbers of them escaped notice between 1920 and 1935. After about ten years, however, such eyes are liable to a degenerative ulceration (delayed mustard gas keratitis). The ulcers are superficial and occur in the deposits of cholesterol where these reach the surface layers. The cholesterol is cast off and the individual ulcer heals with loss of substance after treatment. Hyoscine would appear to give greater relief than atropine in these cases. A number of faceted scars remain which seriously interfere with visual acuity, but as there are no very dense opacities this can be strikingly improved with contact lenses.

This delayed keratitis, which is now seen as a degenerative ulceration, can continue at intervals for twenty-four years or longer after the original exposure. Such relapsing eyes present the characteristic and now well-recognized picture of a transverse band of scarring with cholesterol and fatty deposits, facets, and abnormally shaped blood vessels and blood islands, together with destruction of limbal capillaries in the horizontal meridian and irregular and varicose conjunctival vessels.

(d) The most severe cases of all may go on to perforation of the cornea. These are extremely rare, and are probably due to an actual splash of liquid mustard. The initial course is the same as in the other types (a, b, and c), but the initial partial recovery is retarded and the sight poor all the time. The actual perforation, however, may not occur for ten years or more after the injury.

Conclusions

In the present state of our knowledge the effects of mustard gas on the eye are seen to depend on the size of the dose and the structures involved. Prognosis can be based on this. The series of changes set up by the gas tends towards self-limitation and spontaneous recovery, the results being better than the initial condition would suggest, even in severe cases incapacitated for months. It would appear that treatment at present should be symptomatic, since the condition is a chemical injury with possible secondary degenerative changes. The patient should be reassured as to the ultimate outcome.

REFERENCES

- Mann, Ida, and Pullinger, B. D. (1942). *Proc. roy. Soc. Med.*, **35**, 229.
Phillips, T. J. (1940). *Ibid.*, **33**, 229.

Recent gifts by the late Harvey Cushing and Dr. John F. Fulton have increased the medical library of Yale University to 85,000 volumes, making it one of the largest in the United States.

STRANGULATED HERNIA

A CONSIDERATION OF SOME FACTORS AFFECTING THE MORTALITY

BY

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In many surgical fields it is a difficult matter to assess results. It is only too often necessary to apply arbitrary standards of success such as "Good," "Fair," or "Unsatisfactory," and it is evident that results classified in this way are governed to an appreciable extent by subjective factors. It may be for this reason that analytical studies of case series have been criticized as at best inaccurate and at worst misleading.

Whatever the justification for this view, it is difficult to see how such a criticism can be made in a field whose sole aim is to save life and whose standard is nothing more complicated than survival. It will not be disputed that most cases of strangulated hernia will eventually have a fatal outcome if not treated surgically. By the same token, the primary aim in submitting them to operation is to save life. Hence it must be conceded that it is reasonable to use the fact of survival as the criterion of success in this sphere, a criterion which even the most sceptical can hardly criticize as subjective.

The object of this study was to analyse all cases of strangulated external abdominal hernia treated by operation in the surgical unit of the British Postgraduate Medical School in the five-year period 1936-40, with special reference to the fatalities. By so doing, it was hoped to find where the surgical treatment had failed and how such failures could be avoided in the future. The hernias were divided into three types—inguinal, femoral, and ventral—the last comprising para-umbilical and incisional hernias.

Material

During the period referred to, 551 cases of external abdominal hernia were treated surgically. Of these, 448 were simple and 103 strangulated. The types found in the two groups differed considerably in numerical distribution (Fig. 1). Thus in the simple group 77% were inguinal,

FIG. 1.—Types of hernia met with in a series of 551 consecutive cases (expressed in percentages). It will be noted that the femoral type forms no less than 49.5% of the strangulated group and only 10% of the simple group.

10% femoral, and 13% ventral, while in the strangulated group 33% were inguinal, 49.5% femoral, and 17.5% ventral. This leads to the interesting observation that whereas only 10% of inguinal hernias treated were strangulated, no less than 54% of femoral hernias—more than half—developed this serious complication.

It is true that an uncomplicated femoral hernia in a fat patient may easily escape detection, and that many patients, knowing they have a lump in the groin, seek advice only