The daily food ration of the Indian soldier contains over 60 mg. iron, but of this all except 1 mg. is derived from vegetable sources. It is concluded that the greater part of this iron is not assimilated.

These men, who may develop iron-deficiency anaemia due simply to dietary deficiency, are unduly susceptible to hookworm anaemia.

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"ECZEMA AUTOLYTICA"

BY

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The term I use can be defined as a skin reaction culminating in an acute eruption of scattered vesicles breaking out rapidly on forearms, neck, face, and trunk from above downwards, in that order; following in point of time late after a persisting dermatitis or ulceration or patch of varicose eczema in the leg or ankle region, more often than elsewhere; standing as secondary in relation to one or the other, and as the outcome and sequel. Of a sudden, the primary eruption becomes tender, dry, red, and sharply inflamed, from neglect by the patient or from unsuccessful treatment, so exciting at a distance by way of the blood stream a secondary, autolytic eczema of constant character and uninfected kind, usually beginning on exposed parts, then spreading to covered parts.

The Lesion and Distribution

The vesicle characterizes this autolytic eruption, arising as an erythema about the papillary vessel-loop in the skin, speedily becoming a papule, then a vesicle. Some degree of oedema of the adjacent skin accompanies the vesiculation; there is no urticarial reaction.

At first the vesicles break out on the flexor forearms, sparingly on the sides of the neck and face, and on the trunk in its upper parts—not in patches, but, when fully developed, as a uniform eruption of discrete, scattered pink vesicles deep-seated in the skin, in area unmarginated, without figured pattern but with an uneven bilateral symmetry. Coincident with this outbreak, vesicles erupt about the primary patch; which patch, in its behaviour, offers the clue to the pathogenesis, and points the ready remedy for the secondary eczema. Owing to the limb being dependent, the vesicles in leg or ankle are of venous tint; in disease of the right side of the heart the whole secondary eruption is of a constant cyanotic hue, not pink.

The Cause

To what can this eruption be ascribed? The starting-point lies in a long-standing ulceration or circumscribed dermatitis of the leg or arm that has refused to heal in weeks or months, or after a year or two; the actual cause turns upon this delayed healing. The patient usually has for long laboured in vain with treatment by unsuitable applications, loose bandaging, and without necessary rest in bed. The original patch, vitiated by time and mixed infection, is usually found to have been treated by the mistimed use of a dry non-absorbent tissue dressing secured by one or two turns of a bandage; an aniline dye or silver nitrate paint; a too strong impermeable ointment; or a dusting powder. Any one or sequence of these, used prematurely in the course of the primary dermatitis, has the power to excite dermolysis by occlusion. A dermolysin is thereby formed locally: a foreign protein of bacterial origin or, more likely, a tissue-breakdown product—one or other, or both acting in concert—that sensitizes the patient to his own exudate

and is the trigger that fires the systemic outbreak. This, in its changed form, is absorbed into the blood stream, circulating to the terminal capillaries of the skin, there to be slowed down and subjected to quick, sensitive variations in local temperature and exposed to other physical influences. Perhaps this is a sufficient explanation of the nature and distribution of the eruption. At all events, the lysis is of toxic origin, blood-borne; that much seems certain.

It is generally accepted that autolytic eczema has a constitutional cause to dispose to it, with a closer exciting cause in an auto-intoxication, rather than a micro-organismal toxæmia, arising locally in the patch. Clearly, the oedemato-vesicular eruption is a superficial non-microbic affection of the skin in areas guided and determined by the vessel route taken by the causal agent mentioned, always in the direction first of the uncovered parts. The constant aspect and locality of the autolytic eruption gives the denial to any suggestion of contagion carried by fingers or fomites; spread can happen only by absorption of autotoxin and to a chosen site in the circulation. The autotoxic agent cannot be identified by present-day tests; it is certainly not lethal. An analogous case is the trichophytide—a generalized eruption found in the occasional patient with ringworm of the glabrous skin; a truer analogy is with the tuberculides; another perhaps, though less true, with the eczematides. Autolytic eczema has, however, an origin, character, and independence of its own and, taken in time, is readily curable by eliminating the cause.

The Main Features

In any skin disease it is advisable to examine the whole cutaneous surface, never to omit examining legs and feet, for it sometimes happens that the patient thinks the doctor unlikely to be interested in her "old leg." An incomplete or missed diagnosis will miscarry only to increase the patient's suffering, threatening greater severity and a continuance of the disease, with added difficulty in its control.

In autolytic eczema the signs are those of a systemic affection. The patient is usually ambulant, a woman, the average age of occurrence 50 years; and the lesions may erupt to reach their peak even in so short a time as seven days. The course of the eruption is this: a primary dermatosis in a lower limb from an infected confined dermatitis, ulcerative or other, below the knee; this had begun with an overlooked or neglected trauma, however small, such as a scratch, a burn, or the pulling out of a hair, to breach the surface; and, being still unhealed, is followed in an average of nine months by a secondary eczema erupting in the positions described, the earliest crop of vesicles occurring on the forearms. Autolytic eczema is not seen in extensive acute dermatitis in the leg, but only where the area affected is comparatively small and lasting; in so-called "eczema rubrum" and the moist-surfaced "eczema madi-dans," both more or less acute or subacute in their behaviour, the disease is not seen.

Subjective symptoms are much complained of: hot tense skin, almost intolerable itching, loss of appetite with furred tongue, malaise, distress of mind, insomnia. The patient is apyrexial, and shows no characteristic changes in the blood that can be discovered by tests at present known. Perhaps these facts can be taken to mean failure of immunity response as is believed to happen in protracted disease that is localized. In this case the autolytic eczema develops unchecked. The patient feels ill, depressed, and miserable, chiefly from the subjective discomfort and unhappy self-consciousness created by the disfigurement of face and neck by oedema and vesicles, as well as the fear of infecting others. Itching precedes the outbreak of the autolytic eruption to give warning of its coming, usually worst in the areas about to erupt, but by and by becoming widespread and paroxysmal in attack. The visible eruption begins in an acute phase superimposed locally on the primary focus of inflammation; the secondary eczema follows on, passing through an initial stage of pruritus to papulation, then full efflorescence in an oedemato-vesicular eczema, far distant from its source. Clinically, this progression can be observed in its orderly sequence.

The secondary autolytic eruption bears no resemblance to the original leg or ankle inflammation out of which it arises. The primary patch becomes red and painful, pervaded by a mixed pyogenic infection, and has come to a standstill in the healing. Is it this changed excited dry state of the patch which determines the formation and absorption of a transmuted and toxic agent there, which calls out the autolytic eczema in its acuteness, sometimes to come and go afterwards in a subacute way?

Treatment: The Plan

Treatment, preventive and curative, must first be directed towards the primary source of the disease: here is the key to success. Once established, the autolytic eczema will refuse to go until this is healed. To prevent the secondary eczema, the use of moist permeable absorbent dressings, changed often till the surface has skinned over, is advisable, with the avoidance of all imporous dressings that stifle discharge. This, early in the autolytic outbreak, will prevent or abort the eruption.

The patient ought to be confined to bed, with six-inch bed-blocks under the foot of the bed to hasten the return circulation. The leg congestion being gravitational, this elevation will quickly help to control itching and heat and tightness of the skin, and will go a long way to expedite healing, shortening the time taken measurably.

Treatment of the local patch is best done by applying boric fomentations or wet compresses of hypertonic saline solution, replaced every four hours for three days, to be followed by acriflavine emulsion soaks on gauze, renewed twice daily, for a further ten days. In the case of an open ulcer, the cleansing foment is, after the first three days, followed by carefully adjusted "red lotion" moist dressings morning and night; later, iodine-and-starch paste is applied as a thick spread, changed once a day, then, with improvement, once in two or more days. Bandaging should be firm and supporting, extending from the toes to below the knee, to counter the passive venous congestion of the leg, which only delays repair. This paste, with a tight bandage in the daytime and bed-blocks at night, is far and away better than the Unna-paste-impregnated bandage, which only holds up and confines any discharge, to become potentially, sometimes actually, harmful.

With the conversion of the hot dry surface of the primary patch into a moist freely discharging area, the patient is relieved of much discomfort; and the autolytic eczema begins to fade spontaneously, having itself needed little actual attention beyond the direct application of zinc cream morning and night, or the painting on of a weak ichthyol-in-calamine liniment (3i to 3viii), as being protective and antipruritic. As covering for the arms cotton sleeves are cool and more acceptable to the patient than lint and bandages. When the scaling stage is reached, which is the end-process, a simple cold cream at nights is all that is required, any dressings being discarded.

With the appearance of autolytic eczema, to secure relief for the itching, with peace of mind and enough sleep, nothing is so effective as fractional doses of phenobarbitone with bromide at spaced intervals for as long as is necessary. Autohaemotherapy is of some service at the start, so is non-specific protein therapy, though neither need be overdone or its value overestimated.

The diet should be lacto-vegetarian and salt-free, with an ample daily allowance of fluids (five or six pints, all told); condiments and spices are to be forbidden and over-flushing of the skin avoided. Two drugs are of real use, if indicated: acid. hydrochlor. dil. B.P., given in full dosage soon after meals; and a one-drachm morning dose of magnesium sulphate in hot water, for a time, daily.

Summary

Autolytic eczema is not uncommonly met with in medical practice: the malady has increased in incidence during the war years. Much standing at work while suffering from a patch of open dermatitis in the leg is a predisposing cause, if an indirect one; thus far can autolytic eczema be accounted "occupational." The condition could not exist without the stubborn primary focus to begin it, and refuses to fade before this itself has healed. As a rule, the primary disease persists for long before the secondary outbreak appears; there is a wide, though varying and indeterminate, interval between the starting-point of the one and the other. Peculiarly, the trouble most often originates in a patch about the lower half of the leg or about the ankle; but not always; the primary mischief may occur in a localized dermatitis in the arm or elsewhere following injury. The typical expansive eczema autolytica follows a primary source in the leg; a milder, less extensive eruption when the local source is elsewhere. It is the rule that the eruption breaks out unexpectedly. What actually times and detonates the outbreak has yet to be determined.

The secondary eczema does not behave like a septic toxæmia, to show massive invasion by way of venous or lymph channels: the course taken, and the ready control by early treatment before secondary characters add themselves to the vesicular eruption, suggest a non-virulent auto-intoxication with sensitization. Sepsis alone does not afford so satisfying an explanation as does the theory of causation by autotoxic dermolyisin, the constitution of which is not made manifest by present-day test methods.

It is not to be forgotten that "the skin faces both ways," having an internal surface coextensive with the external and of more importance to the bodily economy. Clearly, the mode of conveyance is vascular, the lysin reaching and acting upon the skin itself from within by way of the capillary loops of the papillary body, likely enough erupting at points of slowing in the circulation there subject to the play of physical influences.

The condition is well and truly named "eczema," qualified by the descriptive term "autolytic." Knowledge of the whole subject is fragmentary still; yet we know that all true eczema is in its proper form vesicular and reactional; that the correct treatment should not be symptomatic merely, but, to be radical enough, ought in the absence of a specific cure to aim at removing the cause. If the remedy is suspended or is unsuitable the disease may take a firmer hold, to become aggravated and in its course lengthy and perverse.

SEGMENTAL MOVEMENT OF THE PUPIL

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In 1911 C. H. Sattler described a pathological movement of the pupil which he called "Wurmförmige Contractionen der Sphincter Pupillae." It is a segmental movement, or twitch of the iris margin. It may be seen in a pupil which has no normal reaction to light, or in some cases in which the light response is present but much diminished. At any one moment during the examination of such a pupil only a single twitch may be apparent; or another similar twitch may appear at the same time further along the pupil margin. Loewenstein described some further cases in 1917 and 1919. He called the phenomenon "Harmonica Contractionen der Sphincter Pupillae." The worm contraction, or undulate contraction, of the pupil border is much less commonly seen than a single segmental movement or double synchronous segmental movement of the pupil in the field examined. The synchronous twitch giving the concertina-like movement would seem to be due to feeble impulses arriving at the same time, while the undulate movement occurred when these feeble impulses arrived at contiguous parts of the pupil border at slightly different times. The amplitude of the movement varies in different cases. Sometimes it can be seen with the naked eye, as in the patient quoted below blinded by quinine. The monocular loupe ($\times 10$) or the low power of the corneal microscope (F/55 mm.) suffices for the recognition of most cases. Only the large inactive pupils of blind or near-blind cases of luetic optic atrophy required the higher powers of the corneal microscope for the recognition of the tiny abnormal movements which were present. They were best seen in the beam reflected from the lens surface. We have been able to observe a progress in amplitude of this segmental movement in a man who had paralysis of the pupil as a result of head injury. At first the movement was tiny, gradually increasing in extent with recovery until there was an entire contraction of the pupil.

The phenomenon has not stimulated much curiosity. In order to determine whether or not the sign had any localizing value a series of cases were examined. Of three cases of luetic optic atrophy with no light projection and large inactive pupils, examined under high magnification, two showed segmental contraction of small amplitude and one an undulate or travelling contraction of the pupil border. Two cases of post-traumatic optic atrophy having no light projection in the affected eye showed segmental contractions. Patients with partial field loss after head injury and patients with partial field loss in glaucoma showed no segmental movement. One man with Leber's disease having a visual acuity of 3/60 Snellen, right and left, had a marked segmental movement. A middle-aged healthy man blinded by quinine showed active segmental movement from the outset of the observed period, which extended over two months. The movement gradually acquired greater amplitude, merging into a total pupillary response to light. Even when the total response to light was established the segmental quality was evident in relaxation until it finally