In recording this case it must be remembered that the actual herpetic eruption was not seen by either Dr. Riseley or myself. But from the very evident scarring (*vide* figure) over the distribution of the upper division of the fifth nerve, and the history of the intense pain preceding and accompanying the eruption, there can be no doubt as to its nature. An associated paralysis of the third and fourth nerves on the same side has been several times recorded in these cases of Herpes ophthalmicus. When we come to the intercurrent attack of paralysis of the *right* third nerve, together with severe pain and iritis, all of which passed off in a few days without any herpetic eruption, there is introduced an element of considerable interest. Cases of bilateral Herpes zoster are not unknown, but I am not aware of any record of a Herpes ophthalmicus with associated paralysis on one side being followed by paralysis without herpes on the opposite side in the same area. The explanation seems to be that the patient is suffering from diabetes. As is well known, in this disease it is not uncommon for paralysis of ocular nerves and iritis to occur, probably as the result of a toxic neuritis. Such was probably the cause of the right-sided intercurrent paralysis, and possibly also of the herpes and paralysis of the left side. It is curious, however, that whilst neuritis in various parts is not uncommon in diabetes, yet herpes in any form apparently is.

SOME OBSERVATIONS ON SMALLPOX.

By LESLIE ROBERTS, M.D.,

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Early in the winter of 1902 I was consulted by a man for an eruption which had appeared on his face three days previous to his visit to the Royal Infirmary. The lesions, of which there were hardly more than a dozen, had the aspect of solid growths. They were nodular in shape, had smooth contours, and were not umbilicated. They looked not unlike the lesions of Adenoma sebaceum. When pricked nothing issued but a little blood; there was certainly no pus, and no hyperæmic reaction. The face was pale. Three days before

the eruption appeared the man felt ill, and for a day took to his bed. I made no diagnosis on his first visit, but the difficulty was soon cleared up, and the patient was admitted to the smallpox hospital. It was a case of variola highly modified by remote vaccinia.

This remarkable, but by no means singular case, occurred at the beginning of a very extensive epidemic of smallpox which passed over certain parts of Liverpool last winter. Through my failure to diagnose this form of variola I became conscious of a defect in my dermatological training. In none of the clinics abroad or at home had I seen an example of variola. And present-day authors of dermatological treatises, trusting to the final extinction of this dread pest, have either omitted it altogether from the list of cutaneous diseases, or, if they referred to it at all, a few perfunctory paragraphs sufficed for its description. But variola is not extinct, and, fostered by foolish legislation, it will once more become an active disease. Since the beginning of the winter of 1902 nearly 1800 cases of smallpox have passed through the hands of Dr. N. E. Roberts, the physician to the city fever hospitals; and thanks to his obliging courtesy, I was able to avail myself of the rare opportunity of studying the disease in its epidemic form.

I shall not essay in this brief article any formal description of variola. It has been admirably done by such masters of dermatology as Hebra, Anthony Todd Thompson, Erasmus Wilson, and Tilbury Fox, not to speak of the great medical writers of the early decades of the nineteenth century. My present purpose shall be confined to the simpler task of noting one or two points which a physician not specially familiar with cutaneous pathology might overlook, and which struck me forcibly, looking at the cases from the standpoint of a dermatologist.

Post-vaccinal Variola.

The older observers recognised these modified cases as forming a distinct class, which Dr. A. T. Thompson called post-vaccinal variola. They were referred to in the old text-books as Variola verrucosa, Variola cornea, wart-pock, horn-pock, stone-pock. I saw many of these acneiform cases of variola, which the old German authors would have called *hornpocken*, in which the lesions were confined to the face,

and which had so little of the variolous aspect that, regarded by themselves apart from the history and the fact that they came from a variola-infected house, their true nature would never have been suspected even by an experienced dermatologist.

In one of the wards I saw a man about fifty years of age. He appeared to be in good health, and answered our questions with a degree of energy which did not suggest any serious constitutional disturbance. The scalp and face were seborrhoic, the follicles dilated, and the capillaries were in the condition seen in mild rosacea. Scattered over the face were a few hard papules, having the aspect and dense, firm consistence of small nodular tumours. I noted specially the absence of pus, and had it not been for the fact that he came from an infected street it would hardly have been possible to make a certain diagnosis. The medical superintendent assured me that there was no doubt as regards the variolous nature of the case, and his opinion was corroborated by the negative result of vaccination.

I observed this facial modification of the variolous lesions in a young man aged 21, who had not been vaccinated since infancy. His body and limbs were universally covered with the typical smallpox pustules. The lesions on the face were so numerous as to completely occupy the whole of the facial surface, and had an entirely different aspect from the lesions elsewhere. They were broad, flattened, slightly elevated tumours of a dull red colour. Each of these tumours had arisen from its own centre, and was separated from the contiguous tumours by shallow grooves. There was no pus in the face lesions. Such cases as these, in which the lesions of the face differed notably from the variolous lesions in other parts of the body, occurred from time to time. Out of a batch of, say, 100 cases of smallpox, the acneiform variety would occur two or three times. There is certainly some tissue influence which modifies the evolution of the variolous lesion, and I believe it is co-existing seborrhœa. Those individuals in whom the horn-pock was best marked were seborrhoic at the time when they became infected with variola.

I was struck by the resemblance presented by the variola pustules to Herpes zoster vesicles. The tendency for the lesions to run in curved lines and to develop in clusters is common to both these diseases. And again, their mode of fusion with adjacent lesions

forming a corymbiform efflorescence is not less conspicuous in variola than it is in Herpes zoster. I think the likeness may be carried further than this; for by observing the extension of variolous lesions over the cutaneous surface in a large number of consecutive cases, I cannot resist the conclusion that one of the major influences which governs the extension of the disease issues from some central nervous mechanism. The evolution of smallpox is so deliberate; its descent from the head, its steady flow outwards to the remote parts of the body so steady, so regular, often so swift; the repetition of its lesions so mathematically uniform (unless where modified by special causes), that I cannot but see in these phenomena the evidence of some force, probably of central nervous origin, which controls the distribution of the disease.

But while it is probable that the general development of cutaneous lesions in smallpox is controlled by a central nervous mechanism, it is quite certain that variations in the intensity of the development of the eruption are produced by external causes, and also by the degree of irritability of the skin itself. Thus Hebra had often observed that the irritation provoked by garters or corsets would determine an extra development of lesions in the irritated site. Indeed, Hebra went so far, if I remember rightly, as to say that one could sometimes guess the occupation of the patient from special variations in the development of the variolous lesions.

I saw two cases which illustrated this principle. The first was that of a young woman who had been subject to repeated attacks of Herpes simplex on the left cheek. The variolous pustules developed in semi-confluent masses in this herpetic area, while only a few pustules were to be found in other parts of the body. The second case was that of an adult woman who had been vaccinated while incubating smallpox; the vaccine pustules formed perfectly, and in the near neighbourhood of the vaccine pustules I noted an unusually rich development of variola pustules, elsewhere the eruption being sparse and discrete.

This leads me to another point of great theoretical as well as practical interest. Can two morbiferous viruses incubate simultaneously in the same body? The answer to this is in the affirmative. I saw cases in which the whole body was covered with variolous pustules from head to foot, in which typical vaccine pustules had

formed on the arm. Clinically the lesions of these two morbid processes can be distinguished by the free superficial suppuration of vaccinia and the early neoplasmoid character of the true variola lesions. The dualism which is so striking a feature in the life-history of these two processes is rendered possible by the fact that the individual inoculated with variolous poison becomes immunised more slowly than the same, or any other, individual inoculated with vaccine matter. Thus, to quote from my friend Dr. N. E. Roberts, the immunising period of vaccinia is nine days, while the immunising point of smallpox is probably not attained until about the eleventh day of the disease—a good three weeks after the disease was contracted, allowing twelve days for incubation. Hence, if an individual contracts the two diseases, variola and vaccinia, on the same day, he will manifest in due time vaccinia, but not variola. If he be vaccinated on the second day after variolous infection, he will develop vaccinia, but not variola. If he be vaccinated on the third day he may still be protected by vaccinia; but if the vaccination be delayed till the fifth or sixth day after variolous infection, the variola will develop *pari passu* with the vaccinia. It is proved now to the point of demonstration that an individual who has incubated variola, and has actually developed the smallpox eruption, is not protected against vaccinia *until a certain time* has lapsed. Dr. N. E. Roberts cites the case of a nurse from one of the general hospitals who was vaccinated forty hours after the variolous eruption had broken out, and yet in this case vaccine pustules developed. This is certainly remarkable, but I have reason to believe that even forty hours is not the extreme limit. The lesson we can learn from this is that the obscure cellular processes by which immunity is brought about require many days to be safely realised, and that the immunising process is certainly longer for variola than it is for vaccinia.*

Another point of no small importance is that the child of a mother affected with smallpox may be inoculated *in utero*. A married woman was admitted to hospital on December the 13th, and three days later (the 16th) was delivered of a child, which was vaccinated on the 17th. In this case vaccination appeared to be successful, but on the eighth day of vaccinia—that is, on the 25th December—the onset of variola was noticed in the child, and on the 27th the eruption

* *N.B.*—Dr. N. E. Roberts's observations on these points will shortly be published.

appeared. The child therefore contracted smallpox twelve days before the 25th December *in utero, i. e.* three days before birth.

The following case presents a contrast to that which I have just cited. A child was born just one day before the mother was admitted to the smallpox hospital. The child was vaccinated immediately. In this case the mother was covered with smallpox pustules from head to foot, but her infant remained well in spite of contact with the mother. The vaccination had just been in time.

My last note relates to the general character of the variolous eruption. I have already mentioned the fact that the eruption is a descending one. This I believe is invariable, and the descent is, in the vast majority of cases, from the scalp or the upper part of the head. The eruption may appear almost simultaneously on the face and hands and forearms, but then we know how close is the nervous relationship between the hands and face. But apart from the apparent skipping, the spread of the disease over the body is so regular as to suggest comparison with a tidal wave proceeding from a focal centre of disturbance, and travelling outwards to the calm beyond the boundary of disturbance. If the whole blood of the patient is equally infected with the variola organisms, how is it that the development of lesions in the skin progresses from one end of the body to the other, and always in the same direction? Before we can answer this question we must know something of the laws which govern the distribution of rashes and eruptions in general, and of those we know very little at present.

There is a striking uniformity in the method of formation and retrogression of the smallpox lesion. If we suppose the body to be divided into transverse planes, we may say as a general truth that all the lesions in any one plane are in the same stage of development. In point of time the lesions on or near the head are always in advance of those on the trunk, while those on the trunk are more advanced than those below the pelvis, and still more in advance of those near the feet. This, I should say, is one of the most notable of all the diagnostic features of smallpox. There appears to be no exception to this law, and so striking is it that it conveys to the mind the impression that nature is working on a single uniform plan. The lesions appear to be moulded on one common pattern. If morphological variations do appear, they are due to influences

which involve whole regions, as, for example, seborrhœa of the face, and gravity and remoteness from the centre of the circulation, the effects of which are seen in the hæmorrhage into the lesions on the soles of the feet.

The smallpox lesion appears to pass through the macular, papular, vesicular, and pustular stages, but even in the evanescent macular stage the lesions are composed of dense infiltration. In one sense the terms *vesicular* and *pustular* are misnomers as applied to the lesions of variola, for the vesicles are never simple cavities in the epithelium filled up with fluid, and the pustules are never simple cavities filled up with pus. Indeed, the formation of pus is an accident which befalls the lesion, and, as Finsen has demonstrated, the accident may be avoided by the total exclusion of blue and violet and ultra-violet rays from the room in which the patient lives. Under ordinary circumstances, however, the lesions terminate in suppuration. But it is not impetiginoid suppuration. It does not consist in a loose collection of leucocytes around a microbic focus. The maturation of the lesions of smallpox which correspond with the advent of the "secondary fever" on the seventh day, is in reality due to the sudden breaking down of the dense cell-infiltration masses impacted within the epithelium. It is during the period of breaking down of the lesions, when so many toxins and irritating agents are liberated, that so much local as well as constitutional disturbance is liable to arise. This is the time when the lesions are surrounded by a red areola, when the eyes become closed and the lips swollen by œdema, and when most pain and distress are experienced by the patient. If by adopting Finsen's method of excluding blue light we can prevent this sudden dissolution of the original dry lesion, we should certainly rob smallpox of its greatest terror.

LABIOMYCOSIS.

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There are doubtless many different conditions included under the general term of patchy eczema of the face, and some of them have