**I. On the lymphatics in leprosy. II. On the changes in the sweat-glands in cancer and leprosy. III. On the growth of the fungi in ringworm, favus and trichorexis nodosa / by George Hoggan.**

I.

# ON THE LYMPHATICS IN LEPROSY.

II.

# ON THE CHANGES IN THE SWEAT-GLANDS IN CANCER AND LEPROSY.

III.

# ON THE GROWTH OF THE FUNGI IN RINGWORM, FAVUS AND TRICHOREXIS NODOSA.

BY

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## *On the condition of the lymphatics in Eastern leprosy.*

By George Hoggan, M.B.

In the twenty-seventh volume of the 'Pathological Transactions' Dr. Vandyke Carter contributes a paper intended to show that the lymphatics of the skin are specially involved in elephantiasis græcorum, and he emphasizes this opinion in the twenty-eighth volume to the extent of attributing similar importance to the lymphatic sheaths of the nerves. If I understand him rightly, his general contention is that leprosy is zymotic in its nature, and that masses of micrococci or zoogloœa are specially to be found in the neighbourhood of the lymphatic vessels and lymphatic sheaths of the nerves, appearing there as clusters of pigmented granules, varying from light brown to black in colour.

In this communication I am in a manner forced to refer specially to those observations of Dr. Carter from the fact (as stated by himself) that they are noteworthy by their "standing alone;" and if I subsequently differ from him in all essential particulars, we differ only in our interpretation of facts admirably pourtrayed by Dr. Carter, but which improved modes of preparation enable me to read and understand in a sense opposed to that arrived at by him.

On last Good Friday a leper died, whose case is probably familiar to most of the skin specialists in the United Kingdom, to all of whom he had applied for relief at some period or other of his long illness of fifteen years, his case having been published at different times by different observers; and I present a photograph of him as he appeared ten years ago, when he entered St. John's Hospital under my colleague, Mr. Milton.

T. S. C— was born in India; his parents were Irish, and his mother suckled him herself. He had been vaccinated in India,

but was unable to trace from whom the lymph had been derived, and at the age of ten years he had been sent to be educated in this country.

Four years later he had an eruption which was treated as scarlatina, and six months afterwards he began to experience tingling sensation in the tips of the ring and middle fingers of the right hand, which soon ceased, but left the skin of those parts anæsthetic. It will be unnecessary for me to go further into his case than merely to state that for fifteen years the disease made steady progress, and when he died sight and smell had entirely gone, voice and cutaneous sensibility had almost completely disappeared, while taste and hearing were already much affected. His nose had entirely disappeared, sight had failed through opacity of the cornea, not apparently from retinal derangement, and his countenance presented the horrible disfigurements characteristic of the last stages of the disease.

For several months before his death he had been under my continual personal supervision, and before he died he gave orders that I should be called at once and his body placed at my disposal, so that I might have an opportunity of applying the silver, gold and osmic-acid methods of preparation upon the still living tissues.

Thanks to his kindness and forethought, I am now able to bring before you some of the results I have obtained, as far as they concern the lymphatic system in general and Dr. Carter's conclusions in particular.

Under the microscope I have arranged specimens, and alongside of them drawings of the appearances they present illustrating the conclusions I have arrived at. In the silver and gold preparations of the plexus of lymphatic vessels as seen from the outer surface of the skin, it will be noticed that the silver lines marking the boundaries of the crenated endothelium, which alone forms the wall of the lymphatic vessels in the skin (fig. 2, pl. XXVI), are everywhere well defined, showing that, as far as the component cells are concerned, the lymphatic walls are absolutely healthy.

Under a low power, embracing a large portion of the plexus of lymphatics in one field (fig. 1), certain notable deviations from the normal conditions in the general appearance and arrangement of the lymphatic vessels are to be detected. In the first place the lymphatics are considerably more dilated than usual, especially at the valvular pouches (fig. 2), some of which appear enormously dis-

tended as they lie immediately under the epidermis, the cause of which I shall subsequently endeavour to explain. This appearance will be clearly recognised in one specimen under the microscope, where a slice was made horizontally from the surface of the skin, that was intended to pass immediately below the plane of the lower cells of the epidermis. At one point, however, owing to a slight depression of the surface, the knife has left a thin layer of epidermic cells still attached to the dermis; these cells have sufficed to protect the underlying dermic tissue, as well as the red colour of the injection filling the blood-vessels, from the action of the silver and gold solutions.

At the very edge of that patch of epidermic cells some largely dilated lymphatics are to be seen, looking like great bags or reservoirs on the course of the lymphatic vessels which pass horizontally from them, and seem to be of the ordinary calibre where they join the dilated portion.

As the crenated endothelium characteristic of the lymphatics is very well marked, no one, I think, can doubt that these are lymphatics, and that this is a condition in which they are seldom seen. Another preparation by a different method, where the tissues were first fixed by osmic acid and then stained by picrocarmine (fig. 4), shows one of those sinuses collapsed; the nuclei of the endothelium lining it appear to be absolutely healthy. It will also be noticed that the lymphatic plexus is much denser than normal, the vessels being closer together (fig. 1), so as to form smaller meshes.

This is probably due to the fact that the skin has become extremely thin, and consequently the lymphatics which run horizontally through the whole thickness of the skin have become, so to speak, compressed into one plane, presenting the appearance of a greater number of lymphatic vessels than usual to a given extent of surface, while in reality the number of vessels is normal, but appearing in a thinner stratum.

This is just what we should expect from their manner of development, for I have shown elsewhere [1] that in the fœtus the lymphatics are all developed on one plane on the hypodermic surface of the skin, but afterwards become separated into different planes in the whole thickness of the skin by the interposition of gelatine, or, as it is called, white fibrous tissue. It is, therefore, only reasonable to suppose that when, as in leprosy, the gelatinous tissue becomes

[1] 'Journal de l'Anatomie,' 1879, page 1.

absorbed, the lymphatics will naturally return nearly into the one plane.

It is probably this same thinning of the skin which causes the great dilatation of the lymphatics at the valvular pouches. If we can suppose a lymphatic vessel passing obliquely through a certain thickness of skin, we may reasonably hold that if that thickness becomes diminished to one half its extent by absorption of the skin, the lymphatic vessel occupying that thickness would also require to accommodate itself to the diminished extent, and this would naturally take place by the walls of the lymphatic settling down, so to speak, to the level of the valvular attachments, where the vessel is strongest and most rigid, and the regurgitating pressure of the lymph also greatest.

Such a movement would give an appearance exactly coinciding with the appearance seen under the microscope.

Owing to the destruction of the individual nerve-fibres in some parts in leprosy, the blood-vessels become greatly dilated. The same cause may also lead to general dilatation of the lymphatics, although such special cases as I have noticed are evidently due to the cause I have assigned to them. I may further mention here that, while that skin was only of half the usual thickness, it was highly elastic, and when cut horizontally with a sharp knife it gave a sensation like that of slicing cheese, being in this entirely different from the tough, and generally with difficulty sliced normal skin.

While I believe that this is the first time that the lymphatics of the skin have been shown in Eastern leprosy, I must also give my opinion that what Dr. Carter has described as lymphatics are only veins, for the lymphatics of the skin have no such course as that assigned to them by Dr. Carter in his drawing, which, on the other hand, corresponds with the course of the cutaneous veins. In fact, I believe that it is impossible with the process he used to show the course of the lymphatics.

Again, in one of the silver preparations and drawings I exhibit, it will be observed (fig. 2) that, while the irregular blotches which mark the spaces containing the cells pathognomonic of leprosy, generally cover the whole surface, at certain points close to the walls of the lymphatics they are entirely absent (fig. 2, *b*).

This is a point of special interest as opposed to Dr. Carter's conclusions, for it will be shown afterwards that these cells are

identical with the leprous elements—the micrococci or zoogloea which he describes. If we examine carefully we shall find that these cells are generally found close to the walls of the blood-vessels, or, properly speaking, the veins, and when they appear to surround the lymphatics, it will be found that this is due to the presence of the blood-vessels in the same locality. But to see this clearly we must have recourse to other methods of preparation. If we make a section through a lately invaded portion of the skin, either in the early or last stages of leprosy, we find that immediately external to the veins (fig. 3), but only there, a great number of cells become deposited in the position usually occupied by the wandering cells.

I submit a preparation and drawing under a high power of such a deposit of cells around a vein (fig. 3), and this, I believe, is the true interpretation of the cells shown in Dr. Carter's drawing. It must, however, be clearly understood that it is only in an early stage of invasion that this can be well seen; for afterwards the various layers join together by absorption of the intervening gelatinous tissue, forming the great masses of leprous tissue shown by Neumann and others in their drawings of skin affected in leprosy. These cells seem to be first deposited in thin laminæ concentric with the vessel, so that if the razor cuts parallel on either side of the vessel, the laminæ become easily disassociated, and it is from such a lamina that the cells seen in the preparation are shown under a high power. In an early stage these cells have a yellowish tint in the white races, but probably they would be darker in the dark races. Osmic acid does not blacken them readily as it blackens fat-cells, as you may have remarked under the microscope. In one preparation, where they are shown in the mass, and which has been stained by indigo (fig. 3), it will be seen that, while the gelatinous tissue of the skin is but slightly tinted of a light lavender colour, the leprous cells lying around the vein appear of a dark green colour, caused by the combination of their own yellow tint with the blue colour of the indigo-staining fluid. This peculiarity cannot be seen if the section has been stained by picro-carmine, as you may satisfy yourselves in the preparation under the microscope.

But what are those cells which become deposited outside the veins? I have little hesitation in pronouncing them to be altered wandering cells, resembling those which, when about to develop into fat cells, first grow a larger amount of protoplasma round their

nuclei than they possess in the active wandering condition. It is in this protoplasm that the first globules of oil make their appearance; and, indeed, this is exactly the condition of several of the cells in the group I have drawn. Some of these cells show oil globules developing within them, and a large globule in one of them has been blackened by the osmic acid injected to fix the tissues.

Now why do these wandering cells congregate and remain outside the walls of the veins? Have they passed out of these vessels, and are they unable to proceed further, or have they returned to them, but are unable to pierce the wall, and thus remain outside and close to the nutrition supply? These are questions I am unable to answer, but I am strongly of opinion that these cells are destitute of the ordinary amount of vitality which enables them to move about, and that this want of vitality is increased by the destruction of the nerves of the part.

It is known that section of the sciatic nerve, in studying nerve degeneration in the living animal, results always in ulceration of the limb and generally death of the animal; and although I have never examined such a limb microscopically, I venture to predict that, when examined, it will be found that a local traumatic leprosy has been unwittingly produced, the ulceration in which is due to the same cause.[1] As those cells continue to accumulate in the immediate neighbourhood of the blood-vessels, they finally extend as dense masses up to the lower surface of the epidermis, from which they appear to cut off the supply of nutrition, causing the epidermis at such spots to break down as if by a localised gangrene, and thus it is that the ulcers characteristic of leprosy appear to be produced.

As regards Dr. Carter's supposition that the lymphatic sheaths of the nerves are similarly affected to his supposed lymphatics, I

[1] In the 'Archives de Physiologie' for 1875 Jean Tarchanoff publishes an account of certain experiments on frogs which are very appropriate to this question. He found that when frogs were curarised (p. 44) "there was a well-pronounced emigration of white cells through the walls of the veins and capillaries into the tissues." He further proved that this was due to paralysis of the vaso-motor system by curare, by means of the following experiment:—"If we destroy (he says, p. 45) the cerebro-spinal axis of a frog we find identically the same picture under the microscope in the mesentery, tongue," &c. Hence it appears that both after experimental destruction of the nerve-centres and during the temporary paralysis produced by curare a condition is brought about similar to what I have shown in leprosy.

can only state that I have given long and minute study to this question of lymphatic sheaths of nerves where the results appeared to me to be satisfactory, and I have come to the conclusion that such lymphatic sheaths of the nerves are purely hypothetical, and have no existence in fact, and that, therefore, it is unnecessary for me to enter further into the question of their importance in eastern leprosy.

Finally, as the result of my investigations into the lymphatics and other systems in leprosy, I feel bound to state that, although leprosy causes changes in the appearance of the lymphatics, such as I have described, these changes are merely secondary, and the lymphatics themselves have no share in the causation of the disease, which must be sought for in other tissues.

*November 5th*, 1878.

*Report of the Committee on Morbid Growths on Dr. Hoggan's microscopical specimens of the skin in leprosy.*—We have carefully examined Dr. Hoggan's specimens of leprous skin, which exhibit well most of the changes described by him. The lymphatics appear to be abundant and to be of larger calibre than normal, especially at the valvular pouches. They do not contain masses of micrococci, nor do we see any canals plugged with micrococcus masses. The chief feature in the disease appears to consist in abundance of cell elements in the cutis and in the subcutaneous tissue. These cells lie not only around the veins but in the interstices of the fibrous tissue around the sweat glands in the vicinity of veins and occasionally of small arteries.

JAMES F. GOODHART,
HENRY T. BUTLIN.

*December 16th*, 1878.

*On the changes in the sweat-glands in cancer and leprosy as illustrating the extremes of atrophic and hypertrophic pathology.*

By George Hoggan, M.B.

It will be within the recollection of some now present that, last session, when I brought before the Society the investigations I had made into the condition of the lymphatics in cancer, and the manner in which secondary tumours develop from cancerous lymphatics,[1] I promised that, as the discussion then going on forced me to confine myself to the effect of the disease on the lymphatics, I would, if permitted, take a future opportunity of bringing before you the effect of cancer on other elements or organs.

To-night I propose, with your permission, to consider the manner in which cancer affects the sweat-glands, and especially the epithelial cells lining the tubes, they being, of all the *fixed* cells, the ones I have found most liable to take upon themselves the cancerous condition.

In my former communication, the only two kinds of cells I treated of as becoming cancerous were, first, the wandering cells, which having become cancerous, formed a plug occluding the lymphatics, and also by far the greater part of the secondary tumours developing from cancerous lymphatics; these cells, from their embryonic character being, as might have been expected, by far the readiest of all cells to assume the cancerous condition. The other cells I spoke of were the endothelial cells, which alone form the walls of the lymphatics and the greater part of the so-called stroma of the lymphatic glands. These fixed cells, as I then showed in several preparations, resisted for a considerable time the cancerous action, and only became cancerous after having been exposed for a certain time to direct contact with the cancer cells which formed the tenacious

[1] 'Pathological Transactions' for 1878, p. 384.

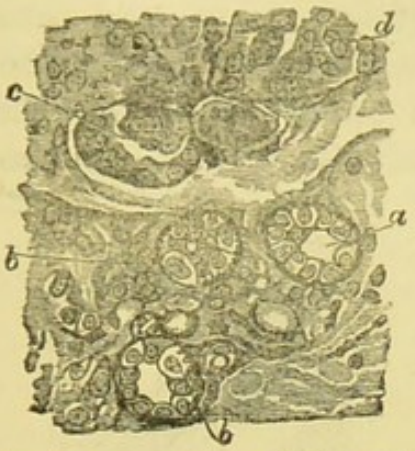
plug occluding the lymphatics; and, indeed, in some specimens, where they were in direct contact with the cancerous plug, both the nucleus and cell-substance of the endothelial wall of the lymphatic still remained intact, as if these, by a special design of nature, were intended to hinder as much as possible the spread of the disease.

With the epithelial cells lining the sweat-glands the case is different, and the above recapitulation has been rendered necessary in order to show, by comparison, the difference between the different kinds of cells in their relation to cancer. In the sweat-glands the epithelial cells are seen to become cancerous, not by direct contact with other cancer cells, but apparently merely as a consequence of the development of cancerous tumours in their vicinity, as if only a certain amount of specific influence, and not necessarily contagion, was sufficient to throw them into the cancerous condition. Indeed, it is in consequence of that susceptibility to take on morbid growth when exposed to excessive stimulation, that I am led to consider the epithelium of the sweat-gland as the element (in this case) in which cancer first developed through the tying of the nævoid wart on the chest. When we make sections of the skin, across the margin or line of infection which separates the healthy from the cancerous tissue, we find numerous instances in which portions of the sweat-glands can be observed, forming part of the lower border of large (microscopically speaking) cancer tumours, as if the tumours were being developed from the upper portions of the glomeruli. In these partially destroyed glands, every step can be traced in the course of infection of their individual cells, and in these it will be clearly seen that there is no continuity of diseased cells in the earlier stages, a solitary cell being seen here and there becoming cancerous, while lying amongst other cells apparently healthy; and it is this absence of continuity or contiguity which has led me to suppose, as already mentioned, that only a pervading influence, and not a direct contagion, was sufficient to throw those epithelial cells into the cancerous condition.

In one preparation under the microscope (Woodcut 1), we have a view of such a section through a sweat-gland becoming cancerous, in which one tube cut transversely contains cells, all of which appear as yet to be healthy (comparatively ?). Next to it are two transverse sections of gland tubes, in each of which only one cell appears to have taken on the cancerous condition, and is now twice as large as the contiguous cells among which it lies, some of which it has

begun to elbow out of their position, so to speak, in order to give itself room to grow, thus leading to disorganisation of the epithelial lining of the gland. It will also be observed that the solitary cancer cell in each appears singularly transparent, as if both nucleus and

Woodcut 1.




Section through the lower portion of the glomerulus of a sweat-gland, showing different stages of cancerous infection of the lining epithelium. *a*, tubule whose lining cells are still comparatively healthy; *b* and *c*, tubules, in each of which only one cell has become cancerous; *e*, tubule in which all the cells have become cancerous; at *d* they have become disorganised and incorporated in the cancer tumour. $\frac{1}{220}$.

cell-substance had become swollen. In a fourth portion of the tube, cut transversely, all the cells have become cancerous and disorganised, so that, but for intervening forms, it is difficult to recognise its glandular character or structure.

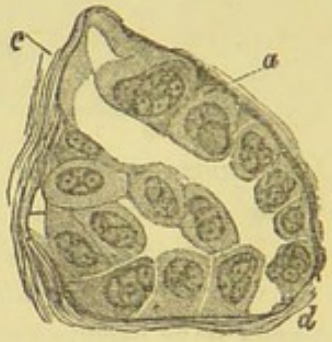
These changes can be seen even more clearly in a neighbouring glomerulus in the same field of the microscope (Woodcut 2), where, although the whole of the epithelial cells lining the tube have become clearly cancerous, yet, owing to bursting of the basement membrane or tube, the cancerous epithelium has been allowed to develope *in sitû*, and thus the appearance of the gland structure has been preserved. In that gland tube the growth of the cells in the cancerous condition seems to have proceeded gradually from one side of the gland towards the opposite point in its circumference, as shown by the regular gradation in the size of the cells passing from right to left (or from below upwards in the figure).

In one respect, this gland tube affords an interesting comparison with the two tubes in Woodcut 1, for while in each of the latter, only one cell had become distinctly cancerous, in this one, only one cell may be said to remain comparatively unchanged.

The mechanical effect of the more advanced cancer cells in bursting, by their swelling, the basement membrane at that point is well shown. This is, however, a rare occurrence, the general rule being that the cells themselves get thrown into an irregular mass.

Even where the regular position of the epithelium does not exist to enable us to recognise a sweat-gland tube in a cancer tumour, we have a valuable index in the cells of the basement membrane to show what was a gland tube, even after its epithelium has become cancerous and disorganised. Normally, those cells of the

Woodcut 2.




Shows under a high power a section across the tubule of a sweat-gland, in which all the lining cells have become cancerous, increasing in intensity from *d* towards *c;* the basement at *c* having ruptured, allows the cells to swell *in sitû*. *a*, triangular end of basement-membrane cell. $\frac{1}{420}$.

basement membrane appear as endothelial cells of the shape of an elongated rectangle, lying longitudinally along the gland tube, and joined at their edges. On transverse section, as will be recognised in the preparation and drawings, they, with their nuclei, appear triangular in shape (probably owing to contraction), and deeply stained black by the pyrogallate, so that the epithelial lining appears encircled by a very distinct serrated girdle. The peculiar shape and staining of these cells are easily recognised in cancerous and disorganised gland tubes, where, indeed, they form the only certain index of its true character.

As far as I have been able to make out, these endothelial cells of the basement membrane only become cancerous some time after the epithelial cells which they enclose have become cancerous, showing, in this apparent resistance to the cancerous influence, a certain

analogy with the endothelial cells forming the lymphatic wall. The cancerous change in the individual epithelial cells can easily be traced throughout its course.

In the first place, both nucleus and cell substance appear to become swollen, and the latter especially grows more transparent, and, if originally polygonal in shape, it becomes globular, as in Woodcut 1. By-and-bye, growth of the cell-substance ceases, although the nucleus continues to grow larger, and finally separates, or rather differentiates into several portions of more or less globular form, as shown in Woodcut 2. I may mention, by the way, that this appearance, which I endeavoured to describe, without pretending to understand it, in my former communication, and which was also drawn in Woodcut 2 under like circumstances, has now been clearly explained by Pouchet's investigations into cell nuclei, published in the 'Journal de l'Anatomie' for January of this year, as being merely an exaggerated condition of the differentiation which normally exists in the healthy nucleus, although it is only recognised with difficulty in the healthy condition.

There is no propagation of other cells from the cell which has become cancerous, and it appears to undergo no other change than what I have described, but goes, as one of the few fixed cells become cancerous, to assist in forming cancer tumours, of which, however, by far the greater part is formed by wandering cells becoming cancerous, which may be seen streaming towards the tumour from the nearest blood-vessels. Finally, as the tumours increase in size, there is no corresponding increase of blood-vessels within them, so that the cells thus deprived of nutrition gradually degenerate into amorphous, clay-like masses, in which no structure can be traced, and amongst which the originally existing blood-vessels, having become cancerous and destroyed, pour out blood into the dead tissue, thus acquiring for such cases the characteristic name of "fungus hæmatodes."

With regard to the gland epithelium seen in Woodcut 1, which I have spoken of as comparatively healthy, I ought to mention that this only appears so when examined by itself. In another preparation which I have placed under the microscope, and which represents a very common condition, it may be seen, by comparing one half of a gland with the other half, or with another gland in the same field, that the slightly swollen and transparent condition of these apparently healthy cells shows that they have indeed entered upon the morbid condition, a condition, however, which appears to be

only passive or intermediate compared with the active cancerous condition into which one cell in each tube has entered.

After the earlier stages of the disease in the glands, when the basement membrane bursts and sets free the cells within it, or the cells themselves get disorganised, it becomes impossible to trace the exact amount they contribute to the whole tumour amongst the shapeless masses of cells which form it; and it is for this reason that I offer Woodcut 2 as an example of hypertrophic pathology for comparison with Woodcut 4, an example of atrophic pathology in the same glands in leprosy, under the same magnifying power. Not that that tubule has reached its maximum hypertrophy by any means, but it is difficult to trace it as a sweat-gland beyond that condition, a point which is equally true of Woodcut 4. As a rule, the cancer tumour or cancerous change in a sweat-gland seems to begin at the summit or upper part of the glomerulus, and in some hundreds of specimens I have never noticed the cancer developing from, or at the lower border of, the glomerulus. This, however, may be due to the position of the cancer-filled lymphatics, which, as I have shown elsewhere,[1] are abundant, and almost always found in close relation to the upper portion of the glomerulus in the more superficial glands, and seldom, if ever, in contact with the lower border. I ought also to mention here, what indeed will be gathered from my previous communication, that the tumours growing near or from the sweat-glands are few, in comparison with the secondary tumours developing from the cancerous lymphatics immediately underneath the epidermis.

To prevent misconception, I ought also to state that the cancer of the skin, in this case, has no relation whatever to epithelioma. In epithelioma the malignant influence begins in, and extends down from, the cells of the epidermis, whereas in this case I have never found an epidermic cell affected by the cancer, owing, apparently, to the fact that the cancer tumour developing beneath the epidermis generally cuts the supply of nutrition off from the latter, and the moist, sodden condition of the underlying diseased tissues allowed or caused the superjacent epidermis to perish by a process of moist gangrene, so that it fell away, or was rubbed off, before it had time to become cancerous—a fate which, I may add, was shared by the

[1] 'Journal de l'Anatomie,' January, 1879.

hair, hair-follicles, and sebaceous glands, although the hair-muscles became incorporated and cancerous, as shown in Plate XXVI, fig. 7.

In my former communication I mentioned that the cancer tumours developing at the glands were of the medullary type, due, of course, to the fact that they were wholly composed of cellular elements. As, however, growth proceeds, the wandering cells, which seem to be attracted by their influence, and are seen flocking to them from every direction, become cancerous while they lie close to them between the bundles of gelatine, or, as it is generally called, white fibrous tissue of the dermis.

These communicate, in turn, the cancerous influence to the wandering cells beyond them, and thus we have layers of cancer cells lying between gelatinous bundles, whose nature also is so changed by the influence as to form a hard, tough material, and in this way what is known as scirrhous cancer is formed, a character given to the whole of the skin in the locality by the union of the various tumours, by the means I have just described, and the incorporation of the altered gelatinous tissue within the one mass.

Sometimes a secondary tumour, developing from one of the superficial lymphatics, compresses the tube of a sweat-gland near its orifice, and thus causes some part of the gland in the glomerulus to become cystic; this cystic condition is, however, a healthy one, the cells being healthy, and placed regularly in one layer, and is quite different from the unhealthy, dilated, or cystic condition of the sweat-glands in leprosy, as shown in Plate XXVI, fig. 6.

The changes which take place during degeneration of the sweat-glands in leprosy, leading to atrophy and complete disappearance of these glands, are much more difficult to trace, and require a longer description than in the case of cancer. Degeneration may be described as consisting of two phases, the first characterised by actual hypertrophy throughout, but leading to destruction of the cellular elements lining the tubes, after which come the atrophic changes of the second stage, which lead to complete disappearance of the gland.

Attention has already been directed to the disappearance of these glands in leprosy, but, as far as I can find out, not one of the various phenomena which characterise their disappearance has ever been described. The contrary has, indeed, been the case, and appearances due to them alone have been described as specific tissue or elements, as, for example, when Neumann gives a per-

fectly accurate drawing of a section of the skin in this disease, in which is seen the duct of a sweat-gland and several transverse sections of the glomerulus portion of the tube.

Following the vicious habit of giving specific names to elements or structures whose true nature the observer fails to understand, Neumann[1] calls the latter "colloid globules," and the former he calls "sinuous connective-tissue strand, formerly hair-follicle." I may also mention here that the destruction of sweat-glands is by no means general in this disease; and although, in the case I have been studying, the patient died in an advanced stage of the disease, it is only at certain parts, such as the hands and feet, that the sweat-glands were found in the last stages of atrophy, while in other parts of the skin of the body, the sweat-glands were either unaffected or only in the first stage of degeneration, and function was still performed. Indeed, during the last hours of his life, the intense perspiration to which he was subject was a constant cause of complaint with this patient. It has also been stated that the gland first becomes destroyed at its orifice, an error which is the more serious because it is the very opposite of its actual condition, and may lead others, as it led me, into a long search in an entirely wrong direction. As a matter of fact, degeneration begins, and goes on fastest, at the lower portion of the glomerulus, and subsequently passes up towards the duct and its termination.

To facilitate description, I have prepared a drawing (Pl. XXVI, fig. 5), under a low power, of a section of skin which is placed under one of the microscopes, and which resembles very much the woodcut given by Neumann. The left gland of the two I have figured is much further advanced in degeneration than the right one, and is fairly typical of the first changes. That gland to the left also appears as if its duct had become occluded before reaching the epidermis, but it only requires a little careful examination to recognise that at that spot the duct has merely made a spiral turn, and the spiral been cut across in making the section; we have thus an appearance as if the gland had actually terminated. This can be best recognised by using the binocular microscope, which shows that, in the present case, the spiral turn passes downwards (towards the glass slide); and, indeed, without going further into the question, I may state

[1] I quote the English translation of his work.

that in no case whatever have I found that the gland-duct dis appeared at its peripheral end in the early part of the degeneration.

The first point of interest we have to observe in the gland just referred to is the peculiar distortion that it has begun to undergo, and which is evidently due principally to the great deposition of leprous cells around it. There is nothing specially unusual in the fact that a gland duct may become spiral near its termination, even before it enters the epidermis, where, of course, the spiral is the rule for very obvious causes. In the present case, the spiral is probably increased by the manner in which the deposit of leprous cells take place. In a former paper, I showed that this deposit takes place specially in the plane or zone of the vascular plexus, thus leaving a certain amount of gelatinous or white fibrous tissue between that zone and the lower surface of the epidermis. This layer of gelatine finally disappears, or is absorbed, and, consequently, that portion of the sweat-duct which formerly stretched across it, is forced to accommodate itself, in its absence, by assuming the spiral form, upon the same principle that it assumes the spiral in the epidermis.

The preceding change is, however, unimportant compared with what takes place at the glomerulus. There it will be observed that the globular form of this, the secreting portion, of the gland, is being gradually destroyed; partly, apparently, by lateral pressure from the neighbouring tissues upon it, but principally and primarily through pressure from above downwards, owing to which the glomerulus is becoming elongated, and its tubular coil is becoming unwound or unravelled. This, I think, is evidently due to the deposition of the thick layer of leprous cells above the glomerulus, which, reacting upon the gelatinous tissue surrounding the glomerulus, pushes it downwards, forcing it to uncoil at the same time, a change which is very manifest when compared with the globular form of the glomerulus of the gland to the right, which is as yet but little affected. This elongating or uncoiling of the glomerulus is, I may state, the general rule throughout the degenerating glands.

Contemporaneously with the distortion of the glomerulus, but whether caused by it or not I cannot say, we have much more important changes going on in the structure of, and in the cells lining, the gland tubules. Even by the low power at which that gland is represented, it is easy to observe that the lumen of the glomerulus portion of the tubule is becoming much more dilated than that of the excretory duct. We may also observe that the lower portion

of the dilated tubule is filled up with *débris*, which does not exist in the upper portion of the dilated tubule, and, further, that where the first coil of the glomerulus joins at right angles the lower end of the gland-duct, there is not only no dilatation, but rather contraction or occlusion of the lumen of the tubule, in consequence of the inner layer of cells becoming vacuolated, and by this swelling occluding the tube. The whole of these changes, however, are better represented in another preparation and drawing of a gland (Pl. XXVI, fig. 6), almost identical with the one we have been studying, and drawn under a high magnifying power, in order to show all the stages which characterise the hypertrophic part of the degeneration, passing from above downwards.

The first thing we observe is that, instead of only having a single layer of cells, as in the normal gland, we have several layers of cells lying within the tubule; but, while the size of the lumen is not materially encroached upon, accommodation has had to be found by distending the tube or basement membrane, and thus we have hypertrophy of the gland by numerical increase of the solid cellular elements.

It will also be observed that the whole of the inner or central lining cells have become vacuolated, and that vacuolation is likewise taking place to a considerable extent among the more external cells, or, in other words, the cells nearest the nutritive centres or regions; and where this vacuolation is taking place, the swelling of the cells next to the lumen leads to a more or less complete, even if temporary, occlusion of that lumen, and dilatation of the lower portions naturally ensues to a small extent. Unlike, however, the cystic condition caused in a healthy gland by pressure upon its excretory duct, as we saw in the case of cancer, we have not here a single layer of healthy cells, but several layers of cells undergoing further changes of vacuolation, as seen in the lower left hand tube.

Before proceeding further, let us now inquire into the character of this condition of vacuolation. In this case, there appears to be nothing specific about it, but it is only the ordinary form of vacuolation that has often been observed even in physiological degeneration of glands, as for example, by Creighton, in degeneration of the mammary gland after the period of lactation is over, although he only describes this one phase as seen in the upper tubule, and gives no clear interpretation of its cause or course.

In the upper tubule every stage of this vacuolation may be

observed, from its first appearance as a minute globule of fluid lying between the nucleus and the cell-substance, up to the formation of a vacuole the size of the original cell, which in most cases compresses the nucleus against or within the protoplasmic wall which surrounds the vacuole. When such a nucleus is observed edgeways, it appears like the signet of a signet ring, being itself somewhat crescent shaped from the distortion it has undergone. When, however, it is viewed from the front, the nucleus possesses its normal shape and appearance, except, perhaps, that it is a little thinner. From this point of view it is more difficult to detect the vacuolation, as the nucleus lies in the centre of the circular vacuole so as to obscure to a certain extent its existence. A commonly accepted view of the cause of vacuolation is that it is caused by the swelling of the nucleolus within the nucleus, but to me that view is altogether untenable, from the very fact that in many cases the nucleus is seen floating free within the fluid of the vacuole, like a blood disc seen edgeways, and sometimes bent in addition. That the nucleus could float within the cavity of something within itself is simply impossible, and I therefore entirely reject the nucleolar theory of origin.

To me vacuolation is merely a process by which the connection of a cell nucleus with its protoplasma is severed, leading to the destruction of the individual cell, a condition which is much more common than is generally suspected. In this vacuolation I do not include such forms of vacuolation as are seen in developing blood-vessels, in the development of bone from embryonic cartilage, &c. I merely refer to the vacuolation which leads to death of the cell.

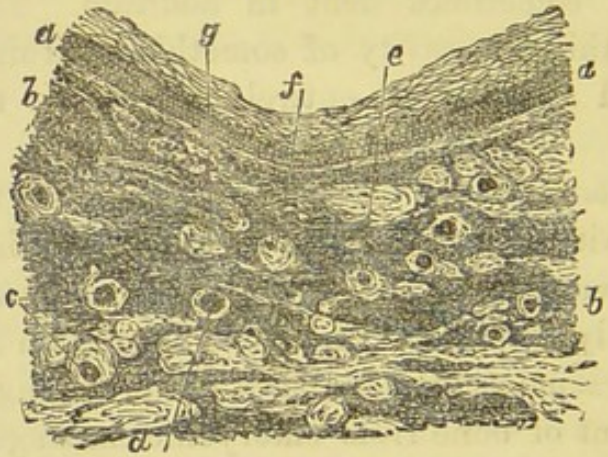
Lately I have found stated in a German article that this vacuolation was simply the formation of fat within a fixed cell. To this I demur, and I have placed under the microscope a sweat-gland whose cells are undergoing vacuolation, and which have been treated with an excess even of osmic acid, and there it will be observed that, although the neighbouring fat-cells are rendered intensely black by the osmic acid, the liquid within the vacuole is entirely unaffected and colourless, showing that it is not fatty, but probably albuminous in character.

Let us, however, trace the process of vacuolation further, as it is seen in the lower tubule on the left hand, in which the vacuolating cells have increased greatly in size, and have become distorted by the pressure of the one upon the other. They are still, however, attached

to the wall, for the cavity or lumen of the tubule is still clear; but in the tubule to the right we see that they are not only falling away from the wall into the lumen, but they appear also to have burst there, freeing the nucleus as well as their contained fluid, and forming an amorphous granular *débris*, that soon fills up the cavity of the tubule. This falling away of the vacuolated lining cells from off the basement membrane of the tubule constitutes the end of the first or hypertrophic stage of degeneration.

From this point the downward or atrophic changes succeed each other rapidly. The *débris* within the tubules becomes absorbed or excreted, and the formerly distended basement membrane contracts laterally through contraction of its cells, which also become vacuolated, and the whole tubule may be now described as attenuating, a layer or deposit of gelatine taking the place externally of the contracting tubule. I present a drawing (Woodcut 3) under a low

Woodcut 3.




From an oblique section of the skin of the finger, shows the different stages of attenuation and absorption of the sweat-glands in the atrophic stage of degeneration. *a*, epidermis; *bb*, leprous cells; *c*, sweat-ducts only slightly attenuated; *e* and *f*, further advanced stages; *g*, spot from which the gland has entirely disappeared, its place being now occupied by gelatinous tissue or tendon. $\frac{1}{350}$.

power, of a section of the skin of the finger, which is also placed under the microscope, and in it will be observed the various changes of attenuation which are being undergone by several glands which, from the distortion they have experienced, appear to have their tubules cut transversely or obliquely across, the gland looking like a dark circlet or dot within a transparent circle formed of gelatine; such a circle of gelatine can also be observed from which the atten-

uated gland has entirely disappeared, thus reaching the lowest possible point of atrophy. I have also drawn and placed under the microscope, under a high power, four stages of degeneration in these glands (or colloid globules of Neumann), which explain themselves.[1] The last of these (Woodcut 4) represents the attenuated gland

Woodcut 4.


Shows a gland-duct about to disappear, similar to *f* in Woodcut 3. *a*, attenuated gland; *c*, tendon-like gelatinous tissue; *b*, leprous cells surrounding the whole. $\frac{1}{420}$.

about to disappear, and as it is drawn under the same power as the hypertrophied gland (Woodcut 2) in cancer, the comparison shows well why they have been chosen as extreme types of hypertrophic and atrophic pathology of sweat glands. Of course it will be understood that neither of the two represents the actual extreme reached in either disease, for, as we have seen in leprosy, the gland disappears altogether from its gelatinous matrix; and in the cancers the gland tubules soon become unrecognisable within the general tumour. I have ventured to describe the whole of the changes in the sweat-glands in leprosy at so great length because, while I am unaware that a careful investigation has ever been made by any one into the changes which take place in an atrophying gland, I also believe that what is true of the glands in leprosy is likewise true of all other glands undergoing chronic atrophy.

It, however, remains still to be shown how the *débris* of the cells becomes absorbed from the cavity of the tubule, or how the cells forming its disappearing basement membrane are absorbed. I have

[1] In a subsequent case of leprosy, I noticed that the attenuating sweat-ducts, when seen longitudinally, presented a moniliform appearance, through irregular contractions and dilatations of their lumen.

every reason to believe that the solid particles move off by their own power, as the particles of an absorbing fat-cell may be seen to move off from the neighbourhood of the cell-nucleus within the clear membrane of the mesentery, a question, however, which can be better studied in a joint article which has been published in the 'Journal of the Royal Microscopical Society' for June, 1879, where it is entered into at greater length than I could possibly do in this paper. *April* 1*st*, 1879.

## *The growth of the fungi in ringworm and favus comparatively studied.*

By George Hoggan, M.B.

Contributions to the history of the fungus of ringworm have of late been so numerous, and apparently so exhaustive, that I almost feel called upon to apologise for bringing this before you; and I can only justify the step by submitting that our knowledge of the course and progress of ringworm and its allied diseases is still incomplete, and having noticed some points which, I think, are not generally known, and which may prove interesting to many here, I have taken the liberty of bringing them before you.

I have chosen the fungi of ringworm and favus, because to me they present extreme types of two different conditions or directions of growth, between or under both of which most of the diseases of the skin due to vegetable parasites may be classed, and I have arranged specimens under the microscope which I think will be found fully to bear out the views I am about to lay before you.

Let me, however, begin by stating that I am not about to argue whether ringworm be due to an oïdium, an ustilago, a penicillium, or an aspergillus, or that favus may be due to any of these classes, or whether the two fungi be clinically the same, or different, or no fungi at all, each of these views being supported at the present day by some authors. What I am about to point out is that each one of the two fungi in its seat and direction of growth, in the appearance of its individual spores and in its clusters, in the element it destroys and its mode of destroying it, is entirely different from the other. I shall ask leave to direct your attention to the condition of the ringworm fungus upon the hair, rather than to its condition upon the surface of the skin, because in the hair we have a definite structure and arrangement of elements which we have not in the skin, and because its limitation to one general mode of disintegration renders the hair specially suitable for my purpose.

It may appear strange that, after the thousands of examinations that have been made of hairs affected by ringworm by eminent dermatologists, any point in its life-history should remain unnoticed; but I believe the reason of this to be very simple. In examining for ringworm fungi, only the hairs which appear to the naked eye to be affected are chosen for examination, and the more they are affected the more readily are they chosen; and thus hairs in the initial stages, come to be overlooked, and the key to the whole thing unnoticed.

Now, in searching for the key, I pursue a different course. Having procured a clear case of ringworm, I search among what appear to be the healthy hairs at its margin for one in which only the first stage of invasion has been reached, and if among a hundred healthy hairs examined under the microscope I find one slightly affected, the find is well worth the trouble taken, and such an one is now under the microscope.

To understand the pathological effect of the fungus upon the hair, allow me to draw attention to the three or four points in its normal structure which make the point intelligible.

In the first place, we have the cuticle of the hair formed of the well-known imbricated cells, lying in one layer like slates upon a roof, and it is because the cells form only one layer that the hair is more suitable for our investigation.

As long as that one layer of cells remains intact, the hair, as a rule, remains also intact, but that layer once broken the hair perishes. It protects the soft, moist, and probably living cells lying underneath it, not only from external influences, but also from becoming hard and dry from evaporation, as they are seen to do at the so-called point of the hair, which becomes tapered and hard through evaporation from the unprotected extremity. We shall afterwards see that the first step taken by the ringworm fungus is to get underneath this cuticle, and to throw it off like the bark of a tree, and this much effected, complete destruction ensues.

Upon the surface of the skin the cuticular layers of cells are too numerous, irregular, and constantly being rubbed off, to allow of its being a suitable tissue in which to fix and study the exact seat of the ringworm fungus. As a matter of fact, the fungus is seen growing several cells deep in the skin, leaving the question vague, but we have only to keep in mind that the many-layered cuticle of the skin becomes continuous with the one-layered cuticle of

the hair to understand how in the first instance the fungus gets underneath the latter. So much for the cuticle. Now for the shaft of the hair, which consists of a comparatively solid tube, formed of elongated cells, and surrounding a more or less patent cavity containing the loose cells of the pith. The way in which the shaft is formed is easily explained by the manner in which a score of cells growing from the side of the hair papilla are forced, during growth, to compress themselves into the same length of the hair which accommodates the one layer of cells growing from the apex; but it is not necessary for this inquiry to go further into the point at present than to note that, owing to the locality where that shaft is formed, the ringworm fungus is not likely to penetrate into the shaft from below, as we shall afterwards see to be the case with the fungus of favus. Let us now follow the action of the fungus, as seen in the drawing and specimens under the microscope. That action may be divided into three stages, passing from the point of the hair towards its root.

In the first place, the fungus propagates itself as a thin layer, only one spore deep, immediately under the cuticle. The spores grow only laterally, as if from buds, the growth, however, being greater, or budding more numerous, in the direction of the length of the hair than round its periphery. For some time after it shows itself, the spores seem too small to work mischief, lying in regular rows under an undisturbed cuticle, but they represent the thin end of the wedge, which has been firmly inserted, and soon brings about the second stage, when the spores growing larger in their bed, make room for themselves by bursting the cuticle of the shaft, as the bark is stripped from a tree, and throwing it off altogether.

The position is now entirely won by the fungus, which, from inability to attack the compact, elongated cells of the shaft, or the necessity of having convenient access to oxygen, had hitherto confined itself to the lower surface of the hair cuticle; but that cuticle removed, the fungus attacks the now defenceless cells of the shaft underneath, grows between them in rows or sheets, and separates them laterally like the bristles in a brush. The hair is now destroyed and breaks off. These three stages appear much plainer in the specimens than any description I can give of them, and they teach a lesson that would never have been learned upon the shapeless and structureless trunks found in an advanced stage of the disease. I may add that the specimen has not been treated by

any caustic alkali, a process which makes more fungi than it shows, both in my hands and the hands of others, judging from one half of the woodcuts that are published.

In the fungus of favus, on the other hand, we have an element whose special seat is immediately underneath the epidermis, and between it and the dermis, its special action being to separate and raise the whole layer of epidermis up bodily from the dermis, and thus to form the circular cups on the surface of the skin, which are characteristic of the disease.

That this point of primary interest should not have been noticed before, is not so surprising as in the case of ringworm, for in favus it can only be clearly seen in such perpendicular sections of the scalp as I have placed under the microscope; and as patients do not die of favus, and the risks of cutting a piece of skin from the scalp are too great, it is not surprising that the point should have remained so long unnoticed.

But just as the ringworm fungus, which had its special seat immediately underneath the cuticular layer of the hair, ends by bursting it off and then appearing everywhere upon and within the hair, so does the favus fungus, after raising off the epidermis, end by piercing and destroying the dermis underneath and the epidermis above, and appearing in the shaft of hairs affected in the disease.

Its action in raising the epidermis as a sheet will be clearly seen in the specimens, the hair follicles and ducts of the sweat-glands remaining attached to the epidermis while it is being raised, and being drawn out of their pits in the corium, like fingers from a glove. This operation has been facilitated by the growth of the fungi downwards between the cellular and gelatinous (white fibrous tissue it is called) sheaths, separating these in the first place, so that elevation of the dermis draws them also out.

The plants and spores of the fungi may still be seen attached to the outer surface of the sweat ducts in the preparations. In these preparations, and also in the drawing, may be observed the first stages of growth of the plant (if I may use the term) where three or four cells or spores appear growing from what is now the free surface of the dermis, the globular spores diminishing in size and tapering towards the point, until the plant appears like one of the little turned wooden pinnacles that one sees taking the place of a chimney on the ends of schools and meeting houses. We have no longer here, as in the cases of ringworm, fungus growth going on

in lines or sheets, but the large favus fungi form plants like oak trees when seen separately, and looking like cactus hedges when seen in the mass cut perpendicularly, that is to say, in the direction of their growth. This, however, is a description which in no way corresponds to existing ideas, which conceive and represent favus plants as long lines of spores. Those conceptions are formed through examining the *débris* of the plants, which has become mixed up with the yellow inspissated serum or albumen exuding from the unprotected dermis, and forming the favus crust generally examined under the microscope. It would be quite as reasonable to expect that one could form a correct conception of an oak tree from an examination of the *débris* and shed leaves lying below it, as to describe a favus plant from the *débris* it sheds. It is quite true that favus fungi have been described and figured in the shaft of a hair as long lines of spores or mycelia; such appearances are, I have no doubt, quite correct, although I have not met with any in my specimens.

It is quite reasonable to expect that, as the favus fungus grows underneath the epidermis, any spores or plants growing within or from the papilla of the hair, would be carried along in the direction and process of growth of that structure, and would take the elongated appearance it there represents, just as the prickle cells of the epidermis assume the elongated form of cells in the hair shaft (or fibres as they are sometimes called), but in neither of the two elements thus modified would the true and general type be recognised. Here and there in the section, but especially in the angle formed, at the periphery of the favus cup by the dermis and epidermis, a layer of coagulated albumen has been cut through in which may be seen imbedded hundreds of the spores and pieces of the favus plants of all sizes and shapes, and looking as natural and well preserved as flies in amber.

The manner in which the fungus propagates by budding can be very satisfactorily observed on some of the large spores forming, so to speak, the trunk of the trees. They all appear, however, to fall off when they have reached a certain size, and do not attempt to grow *in sitû*, which would destroy the tree-like appearance of the plants; they probably go to form the great mass of the *débris* composing the crusts, and also to form the nuclei of new plants.

It will be observed how beautifully the fungus cell or spore changes its shape when, from forming part of a small shoot or branch, it is called upon to act as the trunk of the tree. The round spore

first becomes oblong in shape, but as greater strength and power of adhesion to contiguous cells is required, it becomes constricted somewhat in the middle, like the bones of the fingers, while its end surfaces flatten, so as to form a large surface attachment for the next trunk spore on its distal and proximal ends.

Had this communication been made, as intended, at the last meeting, I should have only referred shortly to the question of the dynamic effect of the fungi in trichorexis nodosa in forming a third type of parasitic growth and mode of destruction, as I had already given my opinion upon it in the 'Lancet.' The discussion, however, which arose on Dr. Pye-Smith's communication, and in which I took part, may make it advisable that I should put on record here the opinion I expressed, and which I justified the same evening by exhibiting specimens showing clearly the masses of spores which were the special character of the disease. At the same time, I ventured to remark that, if the drawing and specimens of Dr. Wilks and Dr. Pye-Smith were typical of the disease they referred to, it seemed probable to me that two different diseases were being confounded, as they were in no way characteristic of the disease to which I referred, and of which I again show specimens.

There was no brittleness of the hairs in my case; on the contrary, when the exploding force of the growing granular masses within the hair tube had burst the tube at what is called the node, it only split the tube open at one side, forming a boat-shaped cavity, which seemed filled with spores, and from which masses of the spores were shed all around. Of course the injury thus done exposed the cells of the shaft to subsequent destruction, but to the last they showed a non-brittle condition, and even when the hair was broken, it showed rather the longitudinal shiver of hard wood than the transverse, cane-like brush of the specimens shown by Dr. Pye-Smith.

I then insisted strongly on the point that the nodular swellings, and subsequent bursting and breaking, were due solely to the growth of the roe-like masses occupying the tube of the hair. I, however, admitted the possibility of those roe-like masses being a granular degeneration of the cells of the pith, but whether fungi or granular degeneration the dynamic effect was the same in bursting the hair tube. Histologically speaking, they might be either, but their exceedingly minute size makes it difficult to decide. Chemically, all the evidence was in favour of their being parasitic. In the first place, the disease began at the tips of the hair and passed down the

hair tube, as if infection had been from without, while a granular condition of the cells would almost certainly have passed from the nutritive cells to the periphery.

Secondly, in similar cases, cutting off the diseased tips of the hair had cured the disease, showing again that the disease was due to external influence, for had it been due to internal influences, cutting off the diseased tips could scarcely have interfered with further development. In the present case, the beard and moustache were quite white, and the disease made its appearance in definite spots of a bright red colour, causing considerable disfigurement and consequent annoyance to the patient.

For the above reasons, then, coupled with the very distinct microscopic appearances, I incline to the opinion that the roe-like masses were parasitic and not granular, and, at all events, choice was limited to the two causes.

As I am not aware that any drawings exist showing the relation of the spawn-like masses to the injured spots, I have prepared drawings of two stages of dynamic action, namely, a newly exploded node and an exploded node being followed by disintegration of the cells of the hair shaft. They have all been drawn from a hair which had undergone no change from reagents, being preserved in glycerine as it was taken from the beard. I submitted other specimens to various reagents, but the appearances still remained, although the mechanical action of the washing and boiling had been to get rid of most of the free spores. As a rule, I condemn the use made of most of the reagents recommended for the examination of hairs. It is difficult to say whether they do most harm by destroying natural appearances or by introducing artificial ones.

After what I have said of the comparative seats and results of growth of the fungi of ringworm and favus, it will readily be understood why the one should be less amenable to treatment and less easily eradicated than the other, and it is because I can nowhere find that the reason has been shown pathologically, that I have ventured to lay before you those views upon what I fear you may be tempted to consider a very paltry subject.

*December* 17*th*, 1878.

## DESCRIPTION OF PLATE XXVI.

Figs. 1—4 illustrate Dr. Hoggan's paper on the condition of the Lymphatics in Eastern Leprosy. (Page 1.) From camera-lucida drawings of preparations by himself.

FIG. 1 shows, under a low power, the changes in the lymphatics (*a*, *a*) of the skin of the neck, and their relation to the hairs (*b*, *b*), sweat-glands (*c*, *c*), and main blood-vessels (*d*, *d*). Owing to thinning of the skin by absorption of its gelatinous tissue, the whole of the lymphatics lie in one plane, so that the plexus appears to be abnormally dense, and the lymphatics themselves dilated. Silver and gold preparation. $\frac{1}{40}$.

FIG. 2 shows a small portion of fig. 1 under a high power. The dilated lymphatic represented has its wall formed of healthy endothelium. The leprous cells (*a*, *a*), which lie everywhere near the blood-vessels (*c*, *c*), are absent from the lymphatic wall at *b*, where no blood-vessel exists. $\frac{1}{200}$.

FIG. 3 shows the early stage of deposit of leprous cells (*a*, *a*) around a vein (*b*). Epidermis (*c*); gelatinous tissue (*d*). Carmine and indigo preparation. $\frac{1}{120}$.

FIG. 4 shows a section through cutaneous lymphatic (*a*), the nuclei of whose wall are perfectly healthy. No leprous cells lie close to the lymphatic wall, although they are becoming deposited at a vein to the left. Gelatinous tissue (*c*); epidermis (*d*). Osmic acid and carmine preparation. $\frac{1}{120}$.

Figs. 5—7 illustrate Dr. Hoggan's paper on the changes in the Sweat-glands in Cancer and Leprosy. (Page 9.) From camera-lucida drawings of preparations by himself.

FIG. 5 shows two sweat-glands imbedded amongst leprous cells in the skin of the nose. The gland to the right is but little affected, but the glomerulus to the left is becoming distorted and elongated by pressure upon it. Its lining cells are vacuolating, causing obstruction of the duct and consequent dilatation of the glomerulus portion of the tubule. These changes are better seen under a high power in fig. 6. Epidermis (*a*); leprous cells (*b*, *b*); gelatinous tissue (*c*, *c*); muscular fibres (*d*). $\frac{1}{30}$.

FIG. 6 shows a portion of glomerulus similar to that in fig. 5. The cells in upper portions of tubule are undergoing the first stages of vacuolation. In the left lower portion the process is further advanced, and the lining cells are greatly distended. In the right lower portion is seen the last stage of vacuolation; the cells are bursting, and falling with their contents as *débris*, filling up the tubule, and thus ending the first or hypertrophic stage of degeneration. Preparations stained with carmine and pyrogallate of iron. $\frac{1}{120}$.

FIG. 7. Two smooth muscle cells from erector pili become cancerous.

Fig. 1.

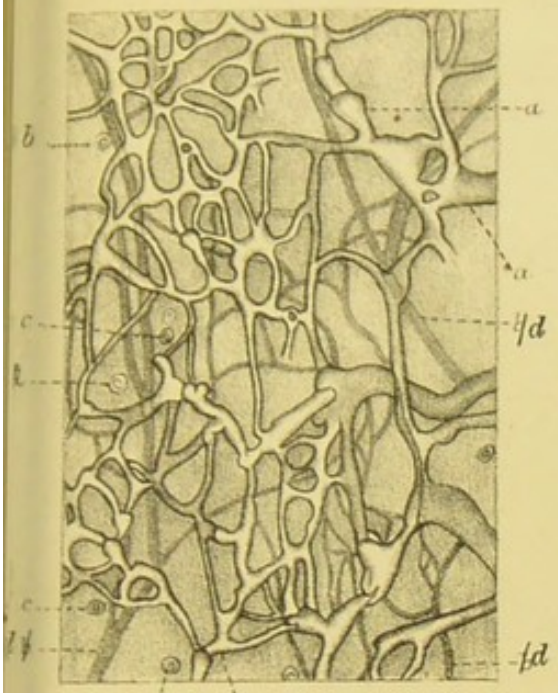



Fig. 3

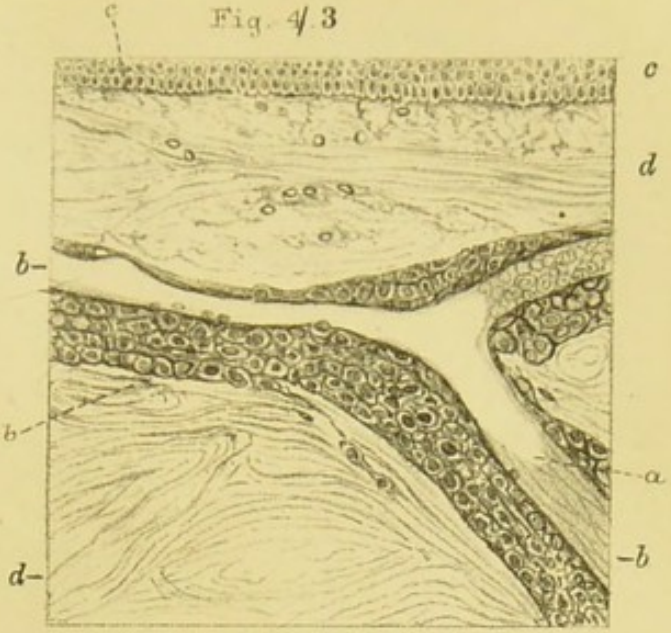



Fig. 2.

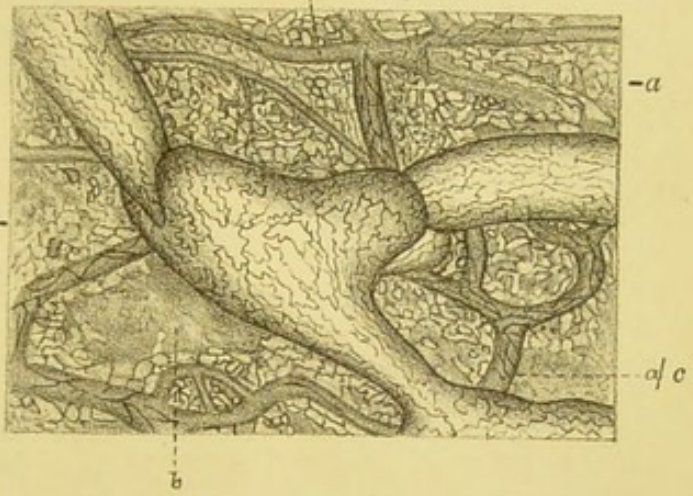



Fig. 4

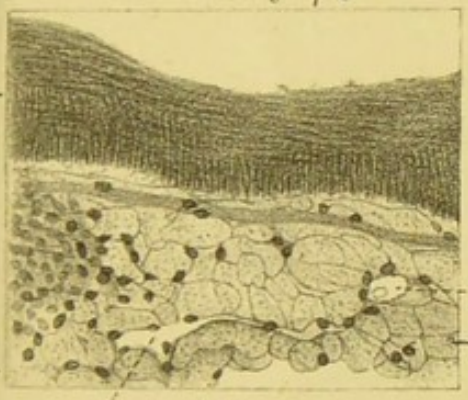



Fig. 5

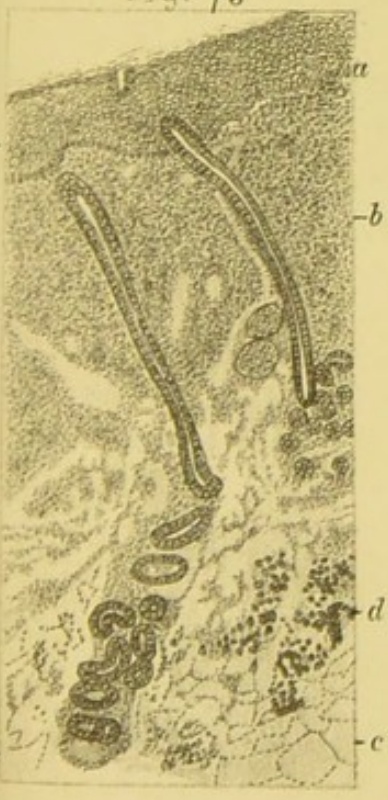



Fig. 6

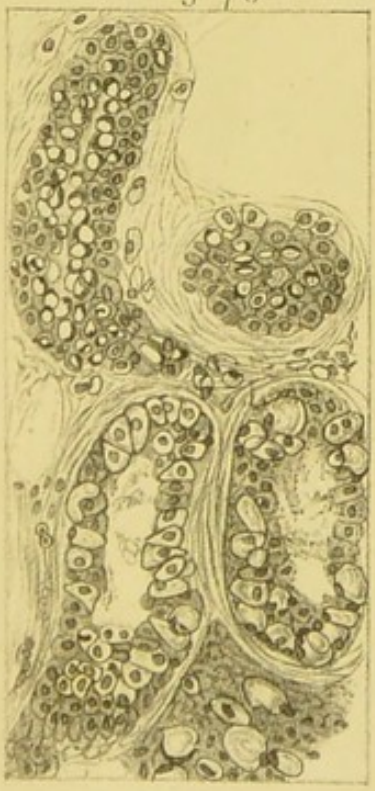


Fig. 7