ON THE PATHOLOGY OF PSORIASIS.

By GEORGE THIN, M.D.

PSORIASIS is a disease of the skin, in which white masses of epidermic scales are attached, more or less firmly, to a reddish vascular base. When the scales are removed by the finger-nails (as they easily can be), small drops of blood ooze from this vascular surface. These two appearances—the heaps of epidermis and the highly vascular papillary layer of the skin—are the essential features of the affection, and are those for which the pathologist has to account. The other distinguishing characteristics, such as the development of circular and gyrating patches by the spreading of the small round spots which constitute the first visible stage of the malady, are of secondary importance in relation to the special pathology of this particular affection.

But to these two very apparent conditions of accumulated epidermic masses and a hyperæmic papillary layer, a moment's reflection compels us to add a third. The fact that the removal of the scales leaves a bleeding surface, shows unmistakably that between the vascular cutis and the horny layer of the epidermis the healthy rete mucosum is deficient. This membrane, in the normal condition, is a sufficient protection to the blood-vessels of the papillary layer against such slight violence as suffices to produce bleeding from a patch of psoriasis. Further, there is something to be learnt from the epidermic masses themselves. Not only are they abundant as regards quantity, but they are abnormal as regards quality. Instead of being separated as imperceptible *débris*, they are detached as scales of comparatively considerable size; and instead of falling readily from the skin, there always remains on the surface of the psoriatic patch (when in the stage of full development) scales which adhere to the living tissue beneath them. When they are forcibly removed, they carry with them the whole of the epidermic covering of the papillæ. It is thus evident that, over the papillæ, sheets of dead epidermic cells, adherent closely to each other, have taken the place of the living rete mucosum.

Thus, the knowledge acquired by the simple naked-eye inspection of a patch of psoriasis, and the legitimate inferences that follow it, teach us that in psoriasis there are increased vascularity of the papillary layer of the skin, and a morbid formation of epidermis over the papillæ. It does not require the evidence of the microscope to show that the essence of this disease of the epidermis is, that the healthy rete mucosum disappears, and that in its place there is a constant and rapid formation of cells that lose their vitality, but do not undergo the ordinary horny metamorphosis.

But to determine more minutely the nature of the changes in the skin, it is necessary to have recourse to microscopic examination, and it is on the histological data which have resulted from the researches of a few observers, that the prevalent views regarding the pathology of the disease rest. The anatomy of psoriasis is stated in Hebra's work on skin-diseases (*Lehrbuch der Hautkrankheiten*, Band i, p. 348) to consist in an unusual development of the rete mucosum, infiltration of the connective tissue of the papillæ with cells, more especially around the blood-vessels, and serous effusion into the interfascicular spaces (œdema). These views, it is stated, are founded on the investigations of Wertheim and Neumann, and were confirmed by the authors (Hebra and Kaposi). It is assumed that these investigations have shown that the excessive formation of epidermic scales in psoriasis, is the immediate consequence of a chronic hyperæmic, or inflammatory, condition of the papillary layer. Rindfleisch (*Lehrbuch der Pathologischen Gewebelehre*, 2nd edition, p. 258) also describes the disease as a chronic inflammation of circumscribed parts of the skin, but gives no sufficient explanation of the differences which exist between this and other forms of cutaneous inflammation. The results of an examination of psoriasis-tissue in all stages of development have been recently published by Dr. Robinson of New York, in a valuable paper in the *New York Medical Journal* (July 1878). Dr. Robinson was able to dispose of a large amount of material for histological study, and has enriched his memoir with a number of instructive woodcuts. Reference to this paper I believe to be indispensable on the part of pathologists, who wish to make themselves familiar with what has been recently done in working out the histology of the disease.

Dr. Robinson gives the following summaries of the views of Simon and Wertheim. Simon believes, he tells us, that the condition of chronic inflammation of the skin has a share in the excessive formation of scales, "in that the newly formed epidermis is continually separated from the cutis by the accumulating exudation beneath it. That part

of the epidermis still closely united with the cutis is thinner than in the normal condition—that is, it is in a condition of atrophy".

Wertheim "found the papillæ, in both their horizontal and perpendicular diameter, enlarged twelve to fifteen times their normal size. The blood-vessels of the papillæ were enlarged and bent. From the enlargement of the papillæ, and the changes in the blood-vessels, he considered that there arises an obstruction to the circulation, which produces the sharply contoured psoriasis patches. His view of the disease, therefore, was, that it arises from changes which take place in the blood-vessels of a circumscribed region".

Dr. Robinson finds that the earliest change consists in a growth downwards into the cutis of the interpapillary epidermic projections. As a consequence of this downward growth of the epidermic boundary-wall of the papillæ, the papilla proper, consisting of blood-vessels and connective tissue, becomes longer. The primary and essential change is in the growth of the Malpighian layer of the epidermis. In the papillæ and superficial part of the corium, Dr. Robinson has, like his predecessors, found enlarged blood-vessels and cell-infiltration.

In the advanced stages, Dr. Robinson has found the same changes, only more marked. The essential feature of the disease, according to his views, is the increased growth of the rete mucosum, by the formation of new cells. He lays stress on the fact that, so far as his observations went, in no place in the abnormal tissue do the papillæ approach nearer the corneous layer than they do in the normal structure—a point regarding which, as will subsequently be seen, I am unable to bear him out.

In the *Vienna Medizinische Jahrbücher* for 1879, Professor Neumann contributes a paper on the results of a renewed examination of psoriasis-tissue. He has found that, in an early stage, the cells of the rete mucosum assume a horizontal position, the "prickle cells" disappear, the "nuclear cells" increase, and instead of one, there are several rows of cylindrical cells. The rete grows downwards, the papillæ are enlarged, and new papillæ are formed. All these changes are, he believes, due to an inflammatory hyperæmia of the cutis. In this hyperæmia is to be found the essence of the disease.

From this summary of the views of the observers who have hitherto occupied themselves with the pathology of psoriasis, it will be seen that, whilst there is a tolerable unanimity amongst them regarding the anatomical changes, there is considerable diversity of opinion regarding their causes and sequence. It is generally admitted that in psoriasis-tissue there are an excessive production of the superficial layer of the epidermis, a new growth downwards of the rete mucosum, enlargement of the papillæ, hyperæmia of the superficial layer of the cutis, and cell-exudation around the blood-vessels. Why the rete mucosum should grow downwards, and why an inflammatory hyperæmia should occur in the superficial layer of the cutis, is not explained.

For the opportunity of contributing to the histology of this disease, I am indebted to the ready co-operation of a patient, a gentleman aged 21, whose skin has not been free from psoriasis patches for the last seven years. Taking an intelligent interest in the nature of the malady, he very willingly agreed that a portion of skin should be removed for examination. The part excised was from a patch of nummular psoriasis on the back, which had existed for about a year, and included a sufficient margin of healthy skin, the raised border of the patch, and a part of its surface. The excised skin was carefully hardened in bichromate of potash, and numerous sections from it were made and examined.

Fig. 1.—Section including the adjacent healthy epidermis (on the right) and the psoriatic epithelium (on the left), as seen under a low magnifying power.

When a section, which included the whole breadth of the excised portion, was examined with a low power covering a large field, it was seen that, whilst the rete mucosum in the healthy skin was of almost uniform depth (the skin here scarcely showing any signs of papillæ),

the rete on the other side of the section corresponding to the psoriasis tissue was indented with long deep papillæ. Even with this low power, it was evident that the interpapillary projections of the rete mucosum had grown to a considerable depth, giving rise, as Dr. Robinson has shown, to an elongation of the papillæ. The margin of the patch was indicated by an unusually adherent crust of horny scales, and a projection of the epidermis outwards. Between this (the naked eye) margin and the perfectly unchanged epidermis, the rete diminished in depth outwards, the papillæ becoming fewer and shallower until they ceased to exist.

Fig. 2.—Section through the margin of the patch at a part where the edge was marked by a prominent epidermic growth, as seen by a low magnifying power. (Both in this figure and in figure 1, the bent form of the portion of skin is due to the contraction produced by the hardening solutions.)

With higher powers, the following important facts could be ascertained. Although the interpapillary downward projections of the rete mucosum were several times deeper than the healthy rete, the parts which covered the papillæ were much thinner than in the normal epidermis. As this is a different condition from that described by Dr. Robinson, and as it has an important bearing on the nature of the disease, I have put it permanently on record, and more definitely than I could do by any mere description, by making the accurate camera-drawings from which Figs. 3 and 4 are copied. Fig. 4 shows the con-

Fig. 3.—Section through the psoriatic patch; magnified 45 diameters. *h*, the horny layer of the epidermis; *m*, the rete mucosum.

Fig. 4.—Section through the healthy skin next the psoriatic patch; magnified 45 diameters. The beginning of the psoriatic change is seen on the right in the first stage of the formation of new papillæ: *h*, the horny layer of the epidermis; *m*, the rete mucosum.

dition of the skin outside the visible patch of psoriasis, passing towards perfectly healthy skin which has not yet been affected by the disease. On the right of the figure, the extension downwards of interpapillary projections and the formation of papillæ have begun. By comparing this drawing with Fig. 3, it will be at once seen that the rete mucosum, *m*, above the papillæ is there much thinner than in the healthy skin shown in Fig. 4, *m*. It will also be noticed that the horny layer *h* is much thicker in the diseased than in the healthy skin. It will be readily understood, from an examination of Fig. 3, why bleeding

follows the removal of the scales from a psoriatic patch. The dried epithelium of which they are formed being still adherent to the rete mucosum, carries with it, when it is forcibly removed, the thin strip of cells which are the only covering of the papillæ, and leaves the vascular tissue exposed.

In order to understand the changes in the constituent parts of the rete mucosum which lead to this result, it is necessary to glance, for a moment, at the composition of the epidermis in health. This membrane consists of a mass of epithelial cells firmly adherent to each other in unbroken continuity from the undermost to the outermost layer. But, although the physical continuity is unbroken, their chemical, vital, and histological qualities vary at different depths. On account of these varying qualities, histologists have given distinctive names to different so-called layers of the epidermis. But in reality these layers run into each other, and represent the gradual transitions which mark the transformation of the living epithelium into dead horny cells. It is possible to over-refine in this distinction of layers; but, for our present purpose, and generally in pathological investigation, it is convenient to distinguish four. Planted on the cutis, and separated from it by a distinct membrana propria, (Fig. 5, *p*) is the undermost layer of cells, which have an elongated cylindrical form, and are placed side by side, like the stakes of a palisade. To these cells, physiologists have assigned—but hypothetically, and without any serious attempt at proof—the function of reproducing the successive crops of epidermic cells, which are being continually formed. The cells immediately above these are more or less cubical, and from the surface of each cell a number of minute spikes or “prickles” project. The so-called layer of “prickle-cells” is always several cells deep. Its exact relative depth can only be stated arbitrarily, and the estimates given of it depend on the greater or less success of the mode of preparation, and on the definition of the objective employed by the observer. I have occasionally been able to trace “prickles” on cells well into the layer above it. This “prickle-cell” layer, in the limits within which it is usually understood, is composed of living cells; but, towards the surface, they already begin to indicate changes by which the formation of horny substance takes place. These changes are more fully developed in the third layer—the so-called *stratum lucidum*—which separates the prickle-cell layer from the fully formed layer of horny epidermis. The cells of this layer contain a newly formed substance, which stains deeply by certain dyes. This substance disappears in the cells of the next or uppermost layer, which is composed of the dead corneous epidermis.

Bearing in mind this division of the epidermis into layers, let us study the thin epidermis which covers the apices of the papillæ, in preparations such as those from which Fig. 5 has been drawn. This study has satisfied me that the layer of prickle-cells has in this position been much reduced in depth, and that the cells which are present are many of them undergoing degenerative changes—indicated chiefly by disappearance of the whole or part of the nucleus (see Fig. 5). The *stratum lucidum*, although present, has lost its definiteness, and above it a great thickness of corneous layer is found. The lowermost layer of cylindrical epithelium contains a greater number of cells undergoing degenerative changes than is found in normal skin. The disease is thus found to coincide with a diminished and abnormal “prickle-cell layer” immediately above the papillæ, that is, of the layer of living cells which form the part of the rete mucosum which is most directly in correlation with the blood-vessels, and from which, by a series of gradual changes, horny cells are being constantly formed. It is undoubtedly as a consequence of the diseased condition of this layer, that we find an unusually rapid formation of an abundant but imperfectly developed horny layer. The cells do not persist as living elements so long as they do in the normal condition, passing almost at once into the horny metamorphosis, which, however, they accomplish incompletely. To such an extent does this disappearance of the living cells of the rete mucosum immediately above the papillæ sometimes go, that I have occasionally found the so-called “prickle-cell” layer entirely absent, and the palisade-like cells of the undermost layer in immediate contact with a closely packed layer of cells corresponding to the horny layer, with scarcely a trace of the appearance characteristic of the *stratum lucidum*. In such a case, the cells that, in healthy skin, are developed into a living resistant membrane (the rete mucosum), are thrown off in the familiar scales of psoriasis.

Contrasting with the deficiency in the rete mucosum which exists over the apices of the papillæ, is the excessive development in the deep interpapillary projections (see Fig. 3). On this point, my preparations bear out the descriptions of Professor Neumann and Dr. Robinson. The large broad masses of rete mucosum found in the sections on the sides of the papillæ show that there is a luxuriant growth of epithelium, representing a “prickle-cell” layer of great depth. Here there is no premature formation of horny from living epithelium, the latter

retaining its normal qualities (as is shown by its thickness) for a considerable time.

Fig. 5.—Section through the epidermis and the upper part of a papilla in the psoriatic tissue, showing defective "prickle-cell" layer and *stratum lucidum* above the apex of the papilla. Magnified 500 diameters: *p*, membrana propria.

To examine the first stages in the formation of new papillæ, it is necessary to examine the epidermis on the extreme edge of the disease—beyond the edge as visible to the naked eye—in a part such as is shown in Fig. 4. The skin of the back, from which my sections have been taken, is well fitted for the study of this process in psoriasis, as the papillæ in the healthy skin can hardly be said to exist (see Fig. 4, *m*). The first stage in the formation of the papilla is, I find, a breaking down in some of the cells of the rete mucosum at a given point, beginning at the lowermost or cylindrical cells, and extending upwards. This first stage is seen in the drawing Fig. 6. A description of this figure will serve as a detailed description of the process. The contiguous cylindrical cells (probably two in number) have disappeared from the deepest layer of the epidermis at the point where the new papilla will form. At the bottom of the gap, two nuclei are seen with a faintly defined zone, which probably corresponds to the area of the cell-substance which has disappeared. Higher up, two well-marked nuclei are found, each within a cell, of which the cell-wall is now alone visible. Immediately beyond these, towards the surface, are two cells, in both of which the nuclei are degenerating. The sides of the space occupied by these degenerating cells have well-marked contours—indicating, as I understand it, the line taken by the lymph-stream between the epithelial cells. In this space, occupied by the degenerating cells, is to be found the first step in the formation of a papilla. The next stage consists in the disappearance of the broken-down epithelium, and the occupation of the space by connective tissue. But, side by side with this destruction of epithelium, a new formation of epithelium takes place at the side of the gap, which leads to the projection downwards into the connective tissue of the rete mucosum, and contributes greatly to the length of the papilla. This new formation has taken place only on one side of the embryo papilla (if I may use the term), shown in

Fig. 6. On the left side of this new papilla, epithelial cells have been added to those which originally formed the boundary of the rete, the whole of the area of the new cells being an encroachment on the connective tissue.

The mode by which these new cells come to be there, opens up a physiological question which, fortunately, it is not necessary for me to discuss in this paper. The current views imply that the new cells are formed by division of the pre-existing cells of the rete mucosum. I may state here, parenthetically, that I have found nothing in my preparations to support this view. In the appearances which are so readily received as proof of cell-division, I can see nothing more than a breaking up of the nucleus.

Fig. 6.—Section through the epidermis, external to the naked eye border of the psoriatic patch; showing the mode of formation of a new papilla. Magnified 500 diameters.

In all my examinations of healthy and diseased skin, I have not once found evidence that the cells of the rete mucosum multiply by division. The new cells, as is shown in Fig. 6, which form the increase of the interpapillary epithelium in psoriasis, are added to the epidermis from the subjacent connective tissue. I see in this fact, however, no proof that they are formed from the fixed cells of that tissue. On the contrary, I find in them transitions which mark development by an enlargement of the nucleus, and a gradual increase in the protoplasm, from elements which are indistinguishable from lymph-corpuscles.

Not only in the papillæ, but along the blood-vessels in the cutis, a moderate amount of cell-infiltration is present. In the cutis, the extent of this infiltration is so comparatively insignificant, as to show that the injury sustained by the vessels in the disease is slight. An example of the penetration of the rete mucosum by the effused colourless blood-cells is shown in Fig. 5. At the apex of the papilla, interruptions in the membrana propria can be observed; and, in one of them, the nucleus of one of these cells can be seen, partly within and partly without the rete. These white blood-cells can, in all pathological conditions of the rete mucosum, be found between the epithelial cells, and in the cavities formed within obsolete cells by breaking down of the nucleus and of the cell-substance. It is in these cells that I find, with Biesiadecki and Pagenstecher, the source of the continual supply of new epidermis; and it is in the numbers that leave the vessels in the superficial parts of the skin in psoriasis that I find the pabulum for the imperfectly formed horny epithelium that is being continually thrown off.

In determining whether in psoriasis the hyperæmia (or, more correctly speaking, the inflammation) is the cause of the affection of the epidermis, or whether, on the other hand, a primary affection of the epidermis is the cause of the inflammation, we are met by this difficulty—that, whilst a morbid condition of the epidermis may provoke inflammatory changes in the subjacent blood-vessels, an inflammation in the vascular tissue may provoke changes in the epidermis; and when, as in psoriasis, we find both these changes coincident, it is not at once evident which change is the primary one. In considering this question, I do not think that much light is thrown on it by the downward growth of the epidermis. I find that, in all conditions in which pathological changes provoke and sustain a continued increased vascularity

in the papillary layer, there is a thickening of the rete mucosum, caused by an increased growth of epithelial cells. In lupus, for example, a cell-growth in the cutis, with which the epidermis has nothing to do, the rete mucosum becomes much thickened. In the cases of rodent ulcer, in which a marginal "ridge" is present, the rete becomes very thick near the margin of the ulcer, before it yields and breaks down, the thickening being purely physiological, and having nothing to do anatomically with the peculiar epithelial growth in the papillary layer, which is the essential feature of the disease. In *malignant papillary dermatitis of the nipple*,* also, I find, coincident with effusion and infiltration in the superficial layer of the cutis, a considerable thickening of the rete mucosum. A thickening of the rete is one of the changes which are produced when there is moderate and continued pressure from effusion from the superficial blood-vessels, and for the interpapillary thickening in psoriasis it is not necessary to seek any other cause. The very moderate amount of cell-infiltration that accompanies psoriasis, and the considerable changes found in the epidermis, are much in favour of the view that the affection of the blood-vessels is secondary to that of the epithelium. The scaling of the epidermis (desquamation in the various degrees) with which we are familiar as a consequence of inflammatory congestion of the cutis—is different in character from that which distinguishes psoriasis. I regard as direct proof that the epidermis is first in fault, appearances such as that shown in fig. 6. Here we have a breaking-down of the epithelium limited to a given point, and far in excess of any changes in the blood-vessels which, at this distant part, can be found to explain it. The special diseased change is at this point in active operation, although it is beyond the zone in which inflammatory changes are found in the blood-vessels. Beneath the part thus figured, the nuclei of the vascular wall are, in the preparation, distinctly stained, and here and there a lymph-corpusele is seen in the tissues; but there is no appearance which justifies the idea that there is distinct inflammatory congestion. My explanation of the changes found in the vascular tissues is, that they are provoked by the irritation set up by an unhealthy epidermis.

Shortly expressed, histological analysis has carried us thus far. A diseased condition of the epidermis at certain localised points leads to inflammatory changes in the subjacent vessels. The serous effusion which takes place from the injured vessels, breaks down the diseased epithelium, and leads to the formation of a papilla. At the same time, whilst the apex of a papilla is being thus excavated, a new formation of epithelium takes place at the side of the new papilla, and by growing downwards, the papilla becomes longer. The exudation from the vessels favours a rapid formation of cells in the rete mucosum; but these cells, from a defect the nature of which is not understood, do not go through the normal changes by which the horny layer is formed, and are thrown off whilst the transformation is incomplete.

The pressure of the effusion from the blood-vessels on the rete produces certain mechanical changes. Neumann had already observed that, in an early stage of the disease, the cells of the rete assume a horizontal position. This can be sometimes seen affecting the cells immediately above the cylindrical cells.

Neumann further observes that the "prickles" of the prickle-cells disappear, that the so-called "nuclear cells" increase, and that, instead of one, there are several rows of cylindrical cells. This description applies to accidental appearances which do not have any direct bearing on the essence of the disease.

Of the cause of the change in the vitality of the epidermic cells, by which the process is introduced, we are ignorant. That it is associated with some constitutional condition of the cells themselves, appears to me to be shown by two considerations. Psoriasis is a hereditary disease, and an inherited tendency to any form of degeneration on the part of a tissue is properly described as constitutional tendency. But further, in a person in whom the disease is in full development, it is possible to excite psoriatic patches on the integument by scratching. One can write psoriasis on their skin. Injury to the rete mucosum is followed by the development of the changes peculiar to this disease. There can be no stronger proof that the predisposing cause of psoriasis is to be found in a constitutional peculiarity of the epidermic epithelium in certain individuals and families.

But although the psoriatic change may break out independently on different parts of the body—apparently spontaneously, but probably always provoked by some external irritation—its extension follows a law which, although not peculiar to this disease, deserves careful study wherever examples of it are found. The law to which I refer is the multiplication of a special diseased action by the influence of contact. When a circle of psoriasis spreads by continuity, it spreads by infecting the contiguous healthy epithelium, and setting up in it the change by

which the normal formation of the rete mucosum is depraved. I apprehend that this action is different from that of a *trauma*, that by which irritation by scratching, for example, produces the psoriatic change. Other lesions of the skin—as eczema and syphilis—run their course in psoriatic patients, and pass over without leaving psoriasis behind them on the parts of the skin which were affected, without generating or extending the morbid action. The psoriatic epithelium, on the other hand, is a slow but certain agent in extending the specific effect; and it is reasonable, therefore, to infer that it is here the specific cause of the advance of the disease.

Of the unknown *materies morbi* in psoriasis, something may be learned from the effects of treatment. The psoriatic epithelium produces a cause of irritation which acts injuriously on the walls of the blood-vessels (that is, in common language, produces inflammation), but the degree of inflammation which it is capable of exciting is comparatively slight. To the narrow limits within which this inflammation is confined, the disease owes its characteristic features. Some morbid agents produce sufficient inflammation—that is, sufficient exudation from the blood-vessels—to ensure their own destruction, and bring about what is then called a spontaneous cure. This is not the case with psoriasis; but a certain degree of superadded inflammation can be artificially induced, in which the exudation is fatal to the disease, and supplies the material for the formation of a healthy epidermis. This degree of inflammation has limits which, in treating the disease, should not be exceeded. All irritants are not equally well suited to produce it. It is to their capacity of ensuring just sufficient, but not too much, inflammation, that the curative effects of irritants like tar, sulphur, chrysarobin, and pyrogallic acid, when properly applied externally, owe their power to cure psoriasis; and it is by bearing in mind that this is their *modus operandi* that the practitioner will best learn how to use them. This curative action of superinduced inflammation is well illustrated by treatment with pyrogallic acid.*

I introduce this subject only parenthetically here, in order to illustrate my suggestions regarding the incompatibility of the disease in the epidermis with a certain amount of exudation from the blood-vessels. The congestion produced by pyrogallic acid is made apparent by the colour of the skin under treatment; and its power to excite destructive inflammation when used even in moderate strengths is illustrated, sometimes disagreeably, by the superficial abrasions around the *lanugo* hairs.

HINTS FOR TRAVELLING, IN THE CASE OF CHILDREN.

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THE following practical hints may prove of service to many members, who are often appealed to by their patients, at this time of year, as to the management of mere infants and young children on long railway journeys, whether to the seaside or elsewhere.

Severe attacks of sickness and diarrhoea, not unfrequently ending fatally, are often met with in infants, who, for some reason or another, are not being suckled, but are solely dependent upon milk from the bottle, from the fact of the milk turning sour. To prevent this, the milk, before starting, should first be boiled, allowed to cool, and then either a teaspoonful of Dinneford's fluid magnesia, or as much carbonate of soda as will lie upon a threepenny-piece, added to each pint. The milk so treated should be placed in soda-water bottles, first carefully rinsed out with hot water, taking the precaution to fill the bottle completely, so as to avoid its being shaken in transit. It is better to use soda-water bottles than the larger wine-bottles, as there is less risk of the remainder turning sour or being spoiled by the shaking inseparable from rapid travelling. A soft cork from a champagne, soda-water, or seltzer bottle (if a perfectly new cork cannot be obtained), which should be first well scalded, and then slightly tapered off, so as to allow of its being readily replaced, is inserted into the neck of the bottle. As each bottle holds about half a pint, it will be requisite to take as many as are likely to be required on the journey. Care should be taken to wrap them in paper or linen, to prevent any risk of their being broken by knocking against each other in the basket.

In very warm weather, more especially in unsettled states of the atmosphere, when thunderstorms are prevalent, even these precautions may not be sufficient to prevent the milk from turning sour. Under these

* This acid, a powerful poison when applied to too large a surface, or incautiously used, I have found, in confirmation of the statements of continental dermatologists, produce very speedy and satisfactory results. My experience with it has shown me that the strength in which it is used should be varied according to the differences in the skin of individual patients, and I never apply it to a large surface.

* See the BRITISH MEDICAL JOURNAL, May 14th and 21st, 1881.