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DR. BALY
ON THE
PATHOLOGY AND TREATMENT
OF
DYSENTERY.



ON THE
PATHOLOGY AND TREATMENT
OF
DYSENTERY;

BEING THE
GULSTONIAN LECTURES DELIVERED AT THE COLLEGE OF PHYSICIANS,
IN FEBRUARY 1847.

By WILLIAM BALY, M.D.
PHYSICIAN TO THE MILLBANK PRISON, AND LECTURER ON FORENSIC MEDICINE AT
ST. BARTHOLOMEW'S HOSPITAL.

[*From the London Medical Gazette.*]

THE HISTORY OF THE
CITY OF BOSTON

IN TWO VOLUMES

BY
NATHANIEL BENTLEY
OF THE BOSTON BAR
AND
OF THE BOSTON COUNCIL
IN 1790
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ON THE
PATHOLOGY AND TREATMENT OF DYSENTERY,

&c. &c.

LECTURE I.

INTRODUCTION—*Reasons for choosing Dysentery as the subject of these Lectures—Unsettled state of Medical Science with respect to the Morbid Anatomy of Dysentery—Structure of the mucous membrane of the large intestine in the healthy state—The solitary glands—Morbid changes in the large intestines in Dysentery, as observed in the Millbank Prison—Three degrees of morbid change—Acute and chronic stage of each described—Nature of the process by which the morbid changes are effected.*

SIR, — When I received from you, as President of this College, the appointment of Gulstonian Lecturer for the present year, I was deeply sensible of the honour conferred on me; but I was conscious also of the weighty responsibility that honour brought with it. I felt, that to address the audience which generally assembles to hear the annual lectures in this theatre, was no trivial task; and that, undertaking this task, I must strive, if possible, so to perform it, as not to bring discredit on the high character of the Royal College of Physicians, into the fellowship of which I had recently been admitted.

This feeling made me anxious that the subject of my lectures should be well chosen. My first hope was, that I might find a suitable topic amongst the more recent discoveries or theories in physiological and pathological science. But, before my choice was made, I was led to consider whether my own observation of disease had not made me acquainted with some new fact, or at least taught me some new mode of regarding facts long known, which I might

fitly communicate to the fellows and members of this College, and to an account of which they might listen with interest. I knew that my experience, compared with that of many who would be my hearers, was very limited; but, on the other hand, I believed that the field in which I had chiefly acquired it, produced some forms of disease rarely seen elsewhere, and presented others under unusual conditions. On further consideration, other reasons suggested themselves to my mind, which finally determined me to devote these lectures principally to a history of *Dysentery*, as it has shown itself at the Millbank Penitentiary. The reasons to which I allude were the following.

Two hundred years ago, dysentery was one of the most prevalent and fatal diseases of London. It now ranks amongst those which are most rare. The great improvement of the physical condition of the metropolis and its neighbourhood during the last two centuries, has doubtless produced this remarkable change. But I had learned from the observation of the diseases prevailing in the great Government Prison of Millbank, that the endemic cause or causes of the dysentery which prevailed in the time of Sydenham,* Willis,† and Morton,‡ have not been wholly eradicated,—that they still lurk in some of the least healthy districts, and, although their power is weakened, are still capable of producing their worst effects when they act on persons of enfeebled nervous and nutritive energies. And I thought some interest might be felt in seeing how the

* Opera Universa, Lugd. Bat., 1726, p. 178.

† Pharmaceutice Rationalis, Genève, 1680, p. 77.

‡ Pyretologia, Genève, 1727, p. 160.

descriptions of the epidemic dysentery of London, given by the three great physicians whose names I just now mentioned, are verified by the occasional reappearance of the same disease, with identical characters, in at least one spot in this city at the present time. I knew, too, that Dr. Latham's elegant and graphic description of the epidemic which prevailed at the Penitentiary in the year 1823,—a work as remarkable for the many profound views of disease it suggests as for the charms of its style,—was not forgotten, and I hoped that a medical account of the health of the establishment, since that period, might be received with interest.

But the foregoing were not the only reasons that led me to the choice of Dysentery for the subject of these lectures. From the perusal of many of the almost innumerable treatises on the disease, I had learned, that, although, like other epidemic maladies, it had been the subject of discussions as frequent as its ravages have been general, several questions, respecting its morbid anatomy, its causes and its relations to other diseases, still remained in an unsettled state; and there seemed to be grounds for believing, that a measure of aid towards reaching the truth as to some of these questions might be derived from observations made in a field where the external conditions affecting the health are, for the most part, known and unvarying, and where few disturbing influences come into play. I conceived, too, that a comparison of the dysentery of temperate climates, with its more formidable representative in tropical countries, would not be devoid of interest. And, lastly, I felt convinced that the fact of the prevalence of dysentery at certain seasons amongst the general population of this city, though in a mild form, and the importance of arresting it, even in this form, at its earliest stage, could not be urged too frequently.

This statement of the reasons which determined my choice of a subject, will serve to indicate the principal topics it is my purpose to discuss.

I shall first call the attention of the College to the still unsettled state of medical science, with respect to the morbid anatomy of dysentery.

When we compare the opinions held by the earliest authors who have treated of this disease with the doctrines of even recent pathologists respecting it, we cannot but feel surprised at the slow advance which appears to have been made towards a certain and accurate knowledge of the internal lesion on which its most characteristic symptoms depend. Hippocrates said, that in dysentery there was "an acrimonious purging with corrosion and ulceration of the intestines and bloody stools." This view of the nature of the disease was adopted by

Galen, and by nearly all the writers who followed him, down to the middle of the 18th century. About that time, when morbid anatomy began to be cultivated more generally, though still imperfectly, the state of the larger intestines in those who died of dysenteric affections became the subject of numerous and varying descriptions. At length, the belief in the existence of ulceration, as the lesion characteristic of the disease, was wholly rejected by many systematic writers; and in opposition to that theory two others were set up. According to one, the local disease consists in congestion and tumefaction of the mucous membrane, especially in patches of some extent, so as to form dark red or purple prominences, called by the French writers "*Boursoufflures*," by the Germans "*Quaddeln*," or "*Protuberanzen*," from the surface of which the epithelium becomes detached by desquamation. This seems to be the idea entertained by Fournier and Vaidy*, by Chomel†, and by two very recent writers Guéretin‡, and Siebert§. The other theory was, that dysentery consisted in an erythematous inflammation of the large intestine which quickly terminated in sphacelus. This is the account Cruveilhier|| gives of the morbid anatomy of dysentery. Rokitansky's description includes these two forms of lesions. The "*dysenteric process*," according to this very accurate German observer, consists in inflammation and swelling of portions of the mucous membrane and sub-mucous tissue,—generally, and in the first instance always, of those portions which project into the cavity of the intestine between the sacculi. This inflammation and this swelling are at first attended with an exfoliation of the epithelium, and at length terminate in sphacelus of the affected part of the mucous membrane¶. Such are the main features of the process as described by M. Rokitansky. All these modern writers, but especially MM. Fournier and Vaidy, Chomel, Siebert, and Cruveilhier, regard *ulceration as having no essential part in the diseased process which constitutes dysentery*, and as being of *very rare occurrence*. It is remarkable, too, that M. Cruveilhier especially insists on the follicles or solitary glands of the large intestine having no share in the disease. "It is not," he says, "a follicular inflammation."

Rokitansky, also, who described an enlarged and ulcerated state of the solitary glands of the colon as the characteristic

* Dict. des Sc. Med. art. Dysent, 1814.

† Dict. de Med., 1823, art. Colite.

‡ Zur Genesis und Therapeutik der rothen Ruhr, p. 98-114, 1839.

§ Archives Génér. de Med. t. vii. p. 52, 1836.

|| Anatomie Pathologique, xl. livraison.

¶ Handbuch des Pathologischen Anatomie, 3te. Band, p. 258-265.

lesion in "chronic diarrhoea," and notices the occasional appearance of irritation and softening of the follicles in cases resembling dysentery, evidently inclines to the belief that these latter cases are not instances of true dysentery.* Yet, during the last year, another author, who has manifestly observed with great diligence and intelligence, Dr. Parkes, has published a minute description of the morbid anatomy of the disease, and has drawn from it the following conclusions: "Admitting the inflammatory nature of the dysentery, the peculiarity about it," he says, "seems to be, that ulceration of the large intestines occurs with great rapidity, and, except in one rare form, a case never presents true dysenteric symptoms without ulceration being present. It is evidently not from the severity of the inflammation that ulceration is so rapidly and so constantly produced, for it occurs in the comparatively slight cases." * * *

"It is owing to the glands of the mucous membrane being particularly implicated in the inflammatory action."† Here, then, we have the statements of an able observer directly opposed to those of two of the most eminent morbid anatomists of our time. How are these contradictions to be reconciled? Does difference of climate cause a difference in the structural changes attendant on the disease? Are distinct epidemics characterized by distinct local lesions? Have two or more distinct diseases been confounded under the one term of dysentery? Or are the various local changes described by different writers only so many forms of the same diseased process, this process being modified in particular instances by constitutional peculiarities of the patients or other circumstances?

Some aid towards solving these questions may be derived from an examination of the morbid anatomy of dysentery, as it has shown itself at Millbank. We shall at least gain data for an inquiry concerning the influence of climate on the disease—an inquiry, the result of which I will anticipate by stating that all the well marked-varieties of structural change in the large intestines observed in tropical dysentery, are found likewise in fatal cases of the disease occurring in our own climate.

From a study of the various anatomical lesions met with in cases of dysentery at Millbank, and their relations to one another, we shall also find grounds, I think, for giving an affirmative answer to the question, whether these various lesions are modifications of the same diseased process.

But to afford solutions to these questions

is not the only object with which I enter upon a description of the structural changes which have offered themselves to my observation in the bodies of those who have died of dysentery in the prison at Millbank. I shall endeavour to show that the opinion which is now, I believe, generally entertained respecting the mode of formation of ulcers in the internal coat of the intestines is, to a certain extent, erroneous,—that these solutions of continuity in the mucous membrane, whatever their size and shape, are produced, at all events at their commencement, *by a process of mortification and sloughing, and not by simple ulceration.*

With a view to facilitate the description of the alterations which the mucous membrane of the large intestine undergoes in disease, it will be useful to glance for a moment at its structure in the healthy state. To enter upon a minute account of the anatomy of the mucous membrane of the intestinal canal generally, is unnecessary, since this subject was treated of most fully by Dr. Todd, in the Croonian lectures for 1842.

The different component strata which are found at every point of the mucous membrane of the large intestine are, the *epithelium*, the *membrana propria* on which the epithelium rests, and the *vascular tissue* which is subjacent to both these structures.

There are besides seen, here and there, small glandular bodies scattered through the substance of the mucous membrane, and known as the *solitary glands* of the large intestine.

The disposition of the *membrana propria*, a transparent homogeneous structure, which has also been named the "basement membrane," and "intermediate membrane," determines the character of the surface of the mucous membrane. In the large intestines it dips inward at innumerable points very close to each other, forming so many blind tubes like the fingers of a glove. These are the *simple tubular follicles*, the openings of which are seen when the surface of the mucous membrane is examined with a lens.

The *epithelium* invests every part of what would otherwise be the free surface of the *membrana propria*, those parts which dip inwards to form the follicle, as well as those which look towards the cavity of the intestine. This epithelium is of the kind called by Henle, "cylinder epithelium," but more appropriately, by Dr. Todd, "columnar epithelium." That is to say, it is formed of little pillar or column-shaped particles placed side by side.

The *vascular tissue* subjacent to the *membrana propria* is composed of a close network of capillaries, and of a mass of amorphous matter, in which a large number of the small bodies called "*nuclei*" or

* Op. citat. p. 260.

† Remarks on the Dysentery and Hepatitis of India, by E. A. Parkes, M.B. London, 1846, p. 3-4.

"cytoblasts" are embedded. This amorphous substance with *nuclei* is capable of being split into fibres, and passes by a gradual transition into the fibrous connecting tissue, which, together with blood vessels, forms the great bulk of the cellular coat of the intestine. The vascular layer of the mucous membrane is a part of great importance. It supplies the material not only for the nutrition of the *membrana propria* and the epithelium, but also for the formation of all the secretions poured out on the inner surface of the intestine: for the *membrana propria* has no vessels of its own, and allows none to pass through it. Like the epithelium, it is a non-vascular tissue, although, as it lies in close contact with the highly vascular stratum I have just been speaking of, it is freely supplied with nourishment.

Such, then, are the structures composing the mucous membrane of the large intestine, except at those points where the solitary glands exist. The condition of these structures is not, however, always the same. The epithelium is from time to time thrown off, and mingling with the liquid exudation of the membrane, forms the healthy mucus which lubricates the canal. At times, too, apparently when the mucous membrane has been the seat of some vascular excitement, the tubular follicles are seen, no longer lined with a regular layer of columnar epithelium, but filled at their deepest part with granular matter containing bodies like the nuclei of cells, and towards their mouths with cells or globules identical in appearance with those which chiefly compose the secretion of all mucous membranes when they are the seat of irritation.

The structure and functions of the *membrana propria* and epithelium, and of the follicles they form, seem to be pretty well understood; but respecting the *solitary glands* our knowledge is at present very imperfect. They have been described as flask-shaped sacs, opening by a small orifice on the surface of the mucous membrane; and they have been supposed to differ from the glands of the small intestine in having constantly open mouths. But after repeated examination of these bodies with the simple microscope, I have satisfied myself that the glands of the large intestine are sometimes open, and at other times closed, and that in this respect, therefore, they resemble exactly the glands of the small intestines, both those known as the solitary glands, and those which are collected into groups, and are termed the glands of Peyer, or *glandulae agminatae*.

When they are closed their existence cannot be recognized on the surface of the intestine by the naked eye, but with the simple microscope a spot can be perceived where the orifices of the tubular follicles are wanting, this spot being slightly raised or de-

pressed according as the gland is completely or only partially filled with its secretion. When the gland has recently opened, the orifice is generally visible to the naked eye, it appearing as a dark depressed point sometimes surrounded by a raised margin. It is in this state that the glands are most easily seen, and they are generally described as they appear in this condition.

When the sacs are quite empty no raised margin exists, but a peculiar appearance presents itself, if the surface of the mucous membrane is viewed with the aid of the simple microscope, and with transmitted light. In the situation of each gland, a number of the tubular follicles are seen radiating from a centre which is depressed below the level of the surrounding membrane. The orifice of the gland is sometimes seen in the midst of these radiating follicles. At other times it cannot be distinguished, having, I suppose, become closed in preparation for the production of the contents of the gland. This radiating disposition of the tubular follicles, which was described and figured in the year 1737*, by an old Italian anatomist named Galeati, seems to be owing to the follicles immediately surrounding the gland being drawn inwards and downwards in proportion as the contents of the gland escape.

The exact structure of these glands of the large intestine I have not been able to ascertain. In the foetus, Dr. Allen Thompson discovered them to be simple closed vesicles,† and he found them occasionally presenting the same appearance as late as the middle of the third year. At a later period of life he believed that they lost this condition. The glands in this simple vesicular state were kindly shown to me by Mr. Quekett, of the Royal College of Surgeons, in the caecal appendix of the colon of a foetus. But they certainly have a different structure in the adult; their walls are thick, not formed of a simple membrane. The matter contained in them is granular, and the granules, instead of being nucleated cells, like the globules found in the tubular follicles, are, according to my observation, solid homogeneous bodies, not much, if at all, larger than the nuclei of the mucous globules, and having a dark well-defined outline. The walls of the gland appear to me to be formed of similar bodies mixed with fibres.

The function of these glands of the large intestine is not known. From their being numerous in the caecum, it might be in-

* Dominici Gusmani Galeatii, De Cribiformi intestinorum tunica in the *Commentarii Institut. Bonon.* t. i. Opuscula, p. 359. I met with the reference to the paper of Galeati in the "*Etudes Hydrotomiques et Micrographiques*" of M. Lacauchie, who has given copies of some of Galeati's figures.

† Report of the British Association for 1840: Transaction of the sections, p. 150.

ferred that they form the acid secretion of that part of the intestines; yet their abounding equally in the rectum seems to be opposed to this view of their office. But whatever their function in the state of health, we shall see that their relation to disease is a very important one.

I proceed now to describe the *morbid changes found in the large intestines in fatal cases of dysentery*.

The diseased changes which I have observed arrange themselves in three different degrees of departure from the healthy structure, corresponding to three degrees of severity in the symptoms during life; and each of these degrees of morbid change has an acute and a chronic stage.

It is not often that the opportunity occurs for observing the state of the large intestines in the *least severe cases of dysentery* at an early stage of the disease. Occasionally, however, the life of the patient is cut short by some other malady; and then the morbid changes discovered in the colon or rectum are these:—the solitary glands in some part or other, generally in the rectum and sigmoid flexure of the colon, or in the cæcum, are swollen, forming round prominences on the surface of the mucous membrane, varying in size from a millet-seed to a full-sized shot. These prominences are sometimes of an uniform pale red colour; at other times they have a ring of bright redness around their base, or present this evidence of vascular turgescence only at their summit. Such were the morbid appearances found in the colon of a female patient in the Millbank prison, who was carried off by inflammation of the lungs eighteen days after the commencement of a mild attack of dysentery. And the same appearances were met with in a male prisoner, who died suddenly with symptoms of cerebral congestion, on the twentieth day, of a similar dysenteric attack.

In two other cases, in which the symptoms of dysentery were rather more severe, life was cut short in the one case on the eleventh day by the supervention of a spasmodic affection closely resembling tetanus, and in the other case on the twenty-second day by pneumonia. Here the swollen solitary glands were surrounded by deeper and more extensive redness, and there was besides an evident disorganization of the summits of many of the prominences; as is represented in this drawing. These disorganized portions, forming minute yellowish sloughs, could be detached with more or less ease from the surrounding tissue. And in a few instances it was clear that these small sloughs had become detached before death, and had left small ulcer-like cavities in the spots previously occupied by the enlarged glands.

The mucous membrane around the glands

which exhibit these effects of inflammation, besides being red and tumid, is usually rough, or covered with a thin aphthous layer, which, by microscopic examination, I have found to be composed of particles of epithelium mixed with amorphous matter, probably fibrine. The mere roughness is, I believe, generally due to an altered condition of the epithelial investment previous to its being detached from the *membrana propria*. This roughened state sometimes exists over irregular spaces of a square inch or more in extent; each such patch having upon it two or three enlarged and altered glands.

Such, according to my observation, are the changes from the healthy state which the mucous membrane of the large intestine presents in the acute stage of the least severe cases of dysentery. And I would call your attention especially to the fact that the small ulcers which, as we shall presently see, are so commonly met with in other stages and degrees of the disease, are here shown to be produced, at all events in their commencement, by a process of mortification and sloughing, and not by ulceration.

The solitary glands of the large intestines in the acute stage of mild dysentery cannot easily be shown to have cavities. Like the agminated and solitary glands of the small intestines found enlarged and inflamed at an early period of continued fever, they seem to be solid masses. But if death does not occur at an early period of the disease, and the inflammatory process, owing to neglect of treatment or other causes, assumes the chronic form, then the cavity of each little gland is restored, and, becoming much larger than in the healthy condition, secretes an increased quantity of fluid of an abnormal character. This change is exemplified by the drawing I now show. It represents a portion of the large intestines of a female patient in the Millbank prison, who died of pleurisy with crural phlebitis, but had suffered for several weeks before her death from dysentery of so mild a character that she did not apply for medical aid until it had existed for more than a fortnight. Here the solitary glands were found enlarged, chiefly in the ascending and the transverse colon. The majority of them had no distinct cavities; they were quite devoid of redness, but were surrounded each by a ring of the ash grey colour so commonly seen in mucous membranes at those parts where inflammation has previously existed. There were some glands, however, situated, for the most part, along the prominences of the rugæ and the longitudinal bands, which were as large as peas, and open at their summits. From these a muciform fluid, in some instances transparent, in others opaque and yellowish, could be squeezed. Several of the largest,

when laid open, would have received a horse-bean into their cavity.

In this case there was no thickening of the coats of the intestine. If inflammation of the submucous tissue had ever existed to considerable extent, it had subsided quickly. But in other cases that pass into the chronic stage, the disease perhaps has originally been more severe; at all events inflammation has been kept up longer in a subacute form, and the result is a generally thickened state of the mucous and submucous coats. The enlarged glands are then not seen as prominences on the mucous surface, but in their places round or oval orifices exist, leading into round sacs or cavities in the submucous tissue. This is a variety of morbid change very often found after death from "chronic diarrhoea;" for this complaint, when not due to tubercular disease, is almost always the chronic stage of dysentery.

Before I pass to the description of the anatomical changes belonging to dysentery of the second degree of severity, let me remark, that in the lesions we have already considered, although the chief characters are derived from the altered state of the solitary glands, the effects of the inflammatory process are, neither in the acute nor in the chronic stage, confined to these glands. In the acute form there is redness, tumefaction, and roughening of the mucous membrane over a considerable extent, with sometimes desquamation of the epithelium and effusion of fibrine; and in the chronic form there frequently is general thickening both of the mucous and of the submucous coat.

In cases of *dysentery of the second degree of severity*, the effects of the inflammation are, as in the slighter cases, generally most marked in the solitary glands, of which a large number are affected. The mucous membrane between them, however, is likewise the seat of active inflammation. The glands lose their vitality, and are converted into small sloughs, but the mucous membrane in which they are imbedded is inflamed and thickened, and is likewise altered on its surface. In the only case I have seen which could be referred to the first fortnight's progress of this second degree of dysenteric inflammation, the mucous membrane was rough, and was, at its surface, evidently converted into a thin lamina of dead tissue connecting together the deeper sloughs that involved the solitary glands. The dead lamina here spoken of certainly implicated the *membrana propria* in the intervals of the mouths of the tubular follicles, but from its thinness seemed not to involve any much deeper portion of the mucous membrane. These changes in the solitary glands and the intervening mucous membrane occupied principally the prominences of the

transverse rugæ of the colon. The surface of the membrane itself did not present much redness, but the submucous tissue of the rugæ appeared very full of blood when compared with the exsanguine condition of most parts of the body of the patient who was the subject of phthisis. At a later stage, but while the inflammatory and destructive process still persists, the small sloughs enclosing the solitary glands are found to have been thrown off; the thin superficial layer of the surrounding mucous membrane which had lost its vitality is also gone, leaving erosions of the surface; and the mucous membrane with the submucous tissue are in most cases much thickened and turgid with blood and serum. The cavities which contained the sphacelated solitary glands appear as small round ulcers with sharply cut edges, resembling holes made with a punch, and give to the inner surface of the intestine somewhat of the aspect of a worm-eaten piece of wood. The ulcers communicate with each other here and there at their deepest part. In other places they have coalesced in their whole depth; and sometimes large ulcers have been formed apparently by the destruction of the septa of mucous membrane which had previously separated many small ulcers. When the disease has affected the intestine only partially, the rugæ are the portions especially occupied by the small ulcers; and the large ulcers, when they exist, are almost always seated on the same prominent parts. The small round ulcers, for the most part, do not pass through the cellular coat; but the large ones commonly reach the muscular coat, and occasionally perforate both this and the serous coat. The drawing I now show, and the corresponding preparation, represent most of the appearances I have enumerated. The man from whose body this specimen of disease was taken suffered from an attack of dysentery, in April 1842; the disease was checked, but diarrhoea, of which he made little complaint at the time, lingered with him during the early summer months, and on the 15th of July he was seized with dysentery of a more severe character, which proved fatal to him in a month. The less acute disease, which had existed three months, may have caused some of the thickening of the coats of the intestine and some of the round ulcers, but the greater part of the changes represented in the drawing, were, I believe, due to the last acute attack. The upper part of the piece of intestine affords proof that the disease was still making progress at the time of death, and at the same time exemplifies one mode of extension of this form of ulceration. Small patches and larger strips of a rough substance are seen which at first sight might be taken for portions of false membrane or

fibrinous exudation. The small patches, however, were found, on close inspection, to line and partially conceal small round ulcers; the larger strips were attached to the margins of large ulcers. They were really small sloughs. When they were removed and examined with the aid of a simple or a compound microscope, the openings of the tubular follicles of the mucous membrane were distinctly visible, and their cavities could also be traced in the mass of slough; though the epithelial lining was gone, and their membranous wall and the tissue on its exterior presented no longer their natural appearance. The diseased changes in this case extended from the ileocolic valve to the commencement of the sigmoid flexure of the colon.

Here, in another drawing and preparation, we have presented to us a second example of the form of ulceration I have been describing; the small round excavations coalescing into larger ulcers. But here the thickened state of the coats of the intestine is absent. The case which afforded this specimen, like the other to which I have referred, occurred in the Millbank Penitentiary. In these two instances the predominant character of the structural changes indicated that the solitary glands of the large intestine were the parts primarily and chiefly affected.

In another set of cases of the same degree of severity the morbid changes found after death have been of a totally different character. The solitary glands have not apparently suffered more nor perished sooner than the surrounding mucous membrane; consequently no small round ulcers have been formed. But the entire mucous membrane, in larger or smaller tracts, where the inflammation has reached a certain height, has fallen into the state of gangrene, and these gangrenous portions, which are generally seated on the prominent folds of the membrane, subsequently becoming detached, leave large ulcer-like excavations.

Some difference in the appearance of the intestines in which the disease has taken this course, are produced by the age and constitution of the patient, and the period of the disease. In young adults, previously healthy, the mucous membrane around the gangrenous parts is intensely red. In old and feeble persons it is often almost devoid of redness. At an early period of the disease the sloughs are of a dark green colour and firm consistence; at a more advanced stage they are of a yellowish white colour, and soft or spongy, and many of them have already become detached, leaving irregular excavations. Of this form of the disease, and its various aspects just indicated, I have seen several instances. It is well illustrated in this plate from Cruveilhier's great work, (plate 3, livraison 31.) Cruveilhier gives this as an example of enteritis, and seems to

regard it as a different disease from dysentery; but the symptoms as well as the structural changes were altogether those of the latter disease. The general paleness of the intestine is accounted for by the patient having been in the puerperal state, and having suffered severely from flooding.

In this form of the dysenteric lesion, as well as in those previously described, where the solitary glands were especially affected, the disease, it will be observed, attacks by preference the transverse rugæ and other prominent parts of the mucous membrane, the chief exception to this rule being met with in the cæcum, which, though nearly devoid of rugæ, is yet a very frequent seat of dysenteric inflammation; and the ulcers, whatever their size and shape, originate by a process of sloughing, and not by simple ulceration.

In Cruveilhier's plate you will notice that several small round sloughs are represented, which seem to indicate that here, besides the diffuse inflammation and sloughing, there was also, at some points, especial affection of the solitary glands. But this combination of the two forms of the disease is frequently more unquestionable than in the instance here depicted.

Two forms of structural change, then, attend cases of acute dysentery of the second degree of severity. In the chronic stage they still retain distinctive characters. In the one we have large ulcers of irregular shape, chiefly occupying the situations of the rugæ and longitudinal bands, while, in the other, the predominant feature is constituted by the small round ulcers. When the inflammatory action has continued long in a sub-acute form the submucous coat in both cases is found much thickened, and at an advanced period of the disease, much indurated in the situation of the ulcers. The contraction of these thickened parts in the manner of the cicatrices of burns sometimes is productive of strictures of the intestine. Of this, however, I have seen no instance at the Millbank Prison.

Allow me here, though it be a digression, to say a few words respecting the appearance presented by healing ulcers of the large intestines, an appearance which is characteristic and suggests the process by which the cicatrization is effected. A recent writer on dysentery has described the process, but, I think, not very correctly, in the following terms. "In an ulcer disposed to heal, lymph is regularly diffused over the surface, forming a gelatinous-looking coating, which becomes gradually darker in colour, rises to a level with the edges of the ulcer and the surrounding membrane, and then slowly contracts, puckering to a greater or less extent the adjacent mucous membrane."*

* Dr. Parkes, op. cit. p. 17.

In this description too exclusive a share in the process seems to me to be given to the lymph effused on the floor of the ulcer, and too little notice is taken of the state of the edges of the sore. A fibrinous exudation undoubtedly takes place on the floor of the ulcer as well as in the interstices of the tissue forming its base; this effused fibrine becoming organised as it is slowly poured out layer by layer. But that the ulcerated surface is brought to a level with the surrounding mucous membrane wholly by this exudation of new matter, is opposed to what I have observed. In the cases in which I have had the opportunity of examining healing ulcers of the large intestine, a remarkable change in the margin of the ulcer seemed to accompany the effusion of fibrine on its floor. The edges of an ulcer which is still in an active state and spreading are perpendicular or even overhanging, but when the ulcer begins to heal its edges become rounded, the margin of the free surface of the mucous membrane being drawn down, as it were, to meet the floor of the ulcer. And now from this edge a delicate lamina, distinguished from the floor of the ulcer by a less dead white colour, shoots inwards till the whole surface is covered. This delicate lamina is doubtless the epithelium. For the epithelium is reproduced on the cicatrices of ulcers of mucous membranes, as the epidermis is on those of the cutaneous texture, although the tubular follicles are never restored.

The appearances presented by healing ulcers of the colon from a case of chronic dysentery, are tolerably well shown in this drawing and in this preparation.

Dysentery, in its most severe degree, is frequently fatal in a very few days. The inflammation affecting a large extent of the mucous membrane reduces it with extreme rapidity to the state of sphacelus. In the parts most intensely inflamed, the whole mucous membrane is found swollen, and of a dark purple colour, or its texture is disorganised, and its colour black, green, or brown.

The effects of this destructive form of the disease were seen at a rather early stage in a feeble young man, who, when he had been two years in prison, was attacked with dysentery, and died after nine days' illness. His body being opened the morning after his death, the mucous membrane of the large intestines was found completely disorganised through a great part of their extent. In the upper half of the large intestine this condition existed only on the transverse rugæ and on the prominent ridges produced by the longitudinal muscular bands. In the lower half of the descending colon the gangrenous parts predominated over the portions of the membrane which

yet retained their organization; and throughout the sigmoid flexure and the rectum, what had been the mucous membrane formed one uninterrupted surface of disorganized tissue, of dark-green colour, rough and warty to the touch, of firm consistence, and half a line to a line in thickness, subjacent to which was a layer of black coagulated blood; and the whole submucous coat was hardened, the blood in its vessels being coagulated. The solitary glands were not seen enlarged in any part of the intestines. I regret that I had no drawing made from this remarkable specimen of the diffuse gangrenous inflammation of the mucous membrane in dysentery; but the same morbid condition is represented in this plate from Cruveilhier's great work (Pl. 5, Livraison 40), and in several of the plates in Mr. Annesley's Treatise on the Diseases of India. It is shewn, too, in this drawing, which was taken from a part of the colon of a young man who died in prison on the tenth day of an illness which partook of the characters of dysentery as well as of fever. The morbid appearances found after death corresponded to the symptoms observed during life, for the ileum presented the inflammation and sloughing of the agminated and solitary glands that belong to continued fever, while the colon presented appearances which answered exactly to Rokitansky's description of the dysenteric process. We see represented in the drawing the different stages of the disease: in one part the swollen and purple-red state of the mucous membrane, and the tumid condition of the submucous tissue, forming prominent masses; then thin crusts of disorganized epithelium and lymph lying upon the inflamed surface; and, lastly, the dark sloughs, involving the whole thickness of the inner coat of the intestine.

When inflammation of this degree of intensity affects a large extent of the intestine, death, as I have already said, generally ensues very speedily. This is not, however, always the case; and, when life is prolonged for several weeks, we see further stages of the disease—or perhaps I should rather say, changes in the parts which the disease has destroyed, viz. the softening and the separation of the sloughs.

The man from whom the diseased intestine represented in this drawing was taken, was attacked with dysentery seven weeks before his death. We see the still congested state of the mucous membrane where it has not been disorganized, and the great thickening of the submucous coat; we observe, also, in the upper half of the intestine, ulcers of various sizes, evidently due to the sloughing of prominent parts of the rugæ, for in one or two the slough is only partially detached; and in the lower half, the state of uninterrupted slough. This last-mentioned state

extended from the middle of the descending colon to the termination of the rectum. The slough resembled wet cotton wool, tinged yellow, and seemed to be chiefly formed by the cellular coat, to which the dead mucous membrane was hanging in shreds. Here and there a part of it had been thrown off, and the muscular coat was bare. In the rectum, the muscular coat also was disorganized, and the walls of the canal, at the same time that they were thickened, were so soft, that the fingers easily broke through them. Portions of the transverse colon and sigmoid flexure are preserved in these two preparations.

It seems scarcely possible that life should be long maintained with a large portion of the alimentary canal in such a condition as this; but where the gangrenous process has not affected the coats so deeply, the patients survive for some time the loss by sloughing of the mucous membrane, and of much of the submucous tissue through more than half the length of the large intestine.

I shall conclude this account of the structural changes observed in fatal cases of dysentery occurring at the Millbank Penitentiary, by directing attention to this drawing and these preparations, which shew the co-existence of the small pin's-head ulcers with the diffuse sloughing of the mucous membrane.

Before comparing the morbid changes I have been describing with those observed in the dysentery of other parts of these islands, of the Continent of Europe, and of India, allow me to dwell for a few moments on some of the characters of the process by which these changes are effected. In its nature, dysentery is indubitably an inflammation. Respecting the tissue primarily affected, there likewise can be no doubt. It is that tissue, composed principally of capillaries, amorphous substance, and nuclei of cells or cytoblasts, which invests the exterior of the solitary glands, and holds together the small tubular follicles of the mucous membrane. The epithelium, the *membrana propria* of the mucous membrane and tubular follicles, and apparently the solitary glands likewise, having no vessels of their own, must, in the normal state, derive their supply of nutritive material from this vascular tissue, through the intervention of the liquid part of the blood which exudes through the coats of the capillaries. Now when inflammation is lighted up in the vascular tissue of which I speak, not only is the quantity of the extravasated liquid increased,—it is also altered in quality. The first effects of this condition, besides turgescence of the membrane, are the pouring out of a muciform secretion from the tubular follicles in increased quantity, and the separation of the epithelium of the mucous membrane

upon and around the solitary glands: at the same time the walls of the capillaries and the *membrana propria* itself must be ruptured or dissolved at some points, so as to allow the escape of the blood disks which are mingled with the discharged mucus. As the inflammation becomes more intense, the blood in the capillaries around the solitary glands at length stagnates and coagulates. These glands then lose their vitality, and, together with the portion of surrounding tissue in which the circulation has ceased, are thrown off as small sloughs: thus are formed round ulcer-like excavations, which are large and deep in proportion to the extent to which the circulation had been arrested in the capillary net-work. If the inflammation do not subside, fresh portions of the mucous membrane, including follicles and a part of the subjacent vascular tissue in which the blood has ceased to circulate, are detached, and thus the small round ulcers continue to extend by a process of sloughing as long as the inflammation continues active. The separation of only the superficial layer of the mucous membrane, forming the mere erosions which are sometimes seen, must be owing to the movement of the blood having ceased only in the most superficial stratum of the capillary net-work.

Respecting the mode of formation of the larger ulcers—those which extend for the most part along the prominences of the rugæ—it seems unnecessary to say many words. The drawings now before you appear to demonstrate the fact that these ulcers, as well as the more extensive loss of the inner coat of the intestine, which occurs in the worst cases of the disease, are due to a process of mortification and sloughing.

The ulcers, however, when they are once formed, and when the inflammation is subdued in severity, may continue to extend slowly by a different process, namely, by a solution of the tissues; this, indeed, is not only possible, but most probable. We can explain the *separation* of the dead portions of tissue while the disease is active only by supposing that they are dissolved at their line of junction with the still living tissue; and this solution must be effected by the fluid poured out by the capillaries. Now it is probable that, when the inflammation is no longer active, the mucous membrane dies in small portions, and that these dead particles of tissue are wholly or in great part dissolved by the fluid exudation, and not cast off in solid sloughs.* The slow increase of ulcers in depth is doubtless due to the same process. There seem also to be cases where shallow solutions of continuity are originally

* The process of ulceration is described by Rokitansky as a dissolving or eroding of the tissues by a peculiar exudation.—Op. cit. t. i. p. 231.

produced in the mucous membrane by a similar process—I allude to cases in which the mucous membrane has been lost without there being any marks of active inflammation in the subjacent and surrounding membrane—cases where the membrane is by some writers said to be destroyed by softening. This, however, must occur very rarely in dysentery,—at all events, in the dysentery of adults. In this disease, when acute, the ulcers are certainly produced by the process before described, *smaller or larger portions of the mucous membrane perishing, and being thrown off as solid sloughs*. This process is, however, not peculiar to dysentery: it is seen conspicuously in continued fever when the affection of the glands of the small intestines exists in a very active

form; and probably also in all mucous membranes in which loss of substance is rapidly produced as a consequence of acute inflammation.

I should not have said so much on this topic, did not the language of modern morbid anatomists seem to indicate that they regard true *ulceration* as the most usual cause of the solutions of continuity so often seen in the mucous membrane of the intestinal canal. The earlier observers were in the habit of regarding all dark and much congested portions of mucous membrane as mortified. Modern anatomists avoid this error; but in the instance to which I have to-day called your attention, they appear to have overlooked the occurrence of mortification or sphacelus where it really occurs.

LECTURE II.

Character of the intestinal lesion in the dysentery of Europe, and in tropical dysentery—Have two diseases been confounded under the one term "Dysentery"?—Morbid appearances in other parts of the body than the large intestines.—Inconstancy of hepatic abscess as a complication of dysentery—Malaria does not prevent the developement of tubercular disease, and the existence of phthisis does not prevent the attacks of dysentery—Typhus or typhoid fever is often combined with dysentery—Symptoms of dysentery—Accuracy of Sydenham's description—Complete absence of feverishness in some cases—Sthenic character of the symptomatic fever in the two less severe degrees of the disease—Symptoms indicating the part of the intestines affected—Symptoms indicating the degree of severity of the inflammation—Sources of the bloody, mucous, and puriform discharges—The third and most severe degree of the disease attended by symptoms which indicate that the blood has undergone a morbid change—Nervous affections associated with dysentery at the Millbank Penitentiary—Their anomalous character—They have not been observed elsewhere.

IN my former lecture I described the lesions which have presented themselves in the large intestines of persons who have died in the Millbank Prison while suffering from dysentery. It will not be necessary that I should occupy much time in showing that the more prominent of those morbid appearances have been generally observed in fatal cases of the dysentery of Europe.

The best accounts of the morbid anatomy of the disease, from observations made in these islands, are those of Dr. Cheyne* and Dr. Abercrombie.† Both of these physicians describe the redness and roughness of the inflamed mucous membrane, the small round ulcers, the sphacelated state of the rugæ, the more extensive sloughs, and the ulcers of various forms which must have been left by the separation of such sloughs; and Dr. Abercrombie, the truthfulness of whose descriptions gives to his medical

works a classical value, mentions also small firm tubercles, which were evidently enlarged solitary glands. Sir John Pringle* and M. Broussais† found the same lesions in the fatal cases of camp dysentery observed by them in many campaigns on the continent of Europe. And I may mention that M. Broussais, in his remarkable work on chronic inflammations, asserts his belief that the ulcers of the large intestine have their origin in the solitary glands.‡ Again, the dysentery associated with the fevers from which the British troops suffered so severely in the disastrous Walcheren expedition, has been well described by Dr Davis.§ And from his account it is obvious that this Walcheren dysentery was, in its anatomical characters, identical with the disease from time to time observed at Millbank.

The same result is afforded by an examination of the works of more recent French and German authors who have observed and described the disease.

We may therefore at once proceed to the inquiry, whether any peculiar characters of the changes in the large intestine essentially distinguish the dysentery of tropical countries from the disease known by the same name in this climate? Dr. Abercrombie, admitting the identity of the two diseases as to their nature, concluded that the dysentery of British systematic writers and tropical dysentery differed in the extent of intestine affected; the disease being confined to the rectum or the lower part of the colon in the former, and extending in the latter through the whole course of the colon, and often affecting also a considerable part of the small intestines. But though the cases in which the whole length of the large intestines is affected are comparatively rare in this country and frequent in India, yet cases do occur in this climate where the extent of the disease is as great as in the dysentery of the tropics. From the extent of the disease, therefore, no constant distinctive character can be drawn. The *form* and *nature* of the anatomical changes in the large intestine,

* Diseases of the Army, fifth edition. London, 1765, p. 237-248.

† Histoire des Phlegmasies, fourth edition. Paris, 1826, t. ii. p. 517-642.

‡ Ibid. t. iii. p. 76 et seq.

§ View of the Fever of Walcheren, by J. B. Davis, M.D. London, 1810, p. 155-182 and p. 191-194.

* Dublin Hospital Reports, vol. iii. p. 1-75.

† Pathological and Practical Researches on the Stomach and other Viscera of the Abdomen, 3rd edition. London, 1837, p. 204-60.

also, afford no ground for distinction; they are in both cases precisely the same. In proof of this, reference need only be made to the works of Mr. Annesley and Dr. Parkes. The former writer describes the intense redness of the mucous membrane which precedes the further changes, the abrasions of its surface, the small and clustered ulcers, the large distinct ones, the sphacelated state of the membrane characterized by a green or greenish brown colour, and disorganized texture, and lastly the state in which the sloughs are partly detached. All these morbid changes, as I have shown, are met with in fatal cases of European dysentery. Dr. Parkes enters into a minute description of the different forms of ulcers, all of which, he believes, may be traced to their origin in inflammation of the solitary glands. He also gives instances of the diffuse gangrenous inflammation of the mucous membrane of the colon, although, as I have before mentioned, he does not regard it as a form of lesion belonging to true dysentery.* It cannot be doubted, therefore, that in their nature and form, as well as their seat, the principal anatomical lesions are the same in the dysentery of India and in that observed in different parts of Europe.

This point being settled, another question must now be considered. How has it arisen that modern writers have in many instances given such contradictory accounts of the morbid anatomy of the disease,—that some have denied the frequent occurrence of ulceration, and the special participation of the small glands in the diseased process, whilst others have characterised the disease as being in its origin *always* seated in those glands;—that one writer regards dysentery as, essentially, erythematous inflammation terminating quickly in gangrene, while another regards this diffuse gangrenous form as a comparatively rare variety of the disease?

With reference to most of the writers here alluded to, and amongst them must be included M. Chomel and M. Cruveilhier, the only conclusion we can arrive at is that their opportunities of observing the disease have been too limited to enable them to take a comprehensive view of its morbid anatomy,—that they have observed only epidemics in which one form of lesion greatly predominated. We may ascribe the incomplete description of the dysenteric process, given by M. Rokitsky, to the circumstance that although he has enjoyed ample facilities for observing the morbid changes in the body after death, he has had but few opportunities for tracing the connection between these

changes and the symptoms of the disease and its causes. But neither of these conclusions can be adopted in the case of Dr. Parkes. He has evidently seen all the principal forms of the structural lesions, has watched the symptoms attending them, and has known the circumstances under which the disease in the respective cases, arose. He seems, however, to exclude the *diffuse erythematous and gangrenous inflammation* of the large intestines from the category of the structural changes belonging to *ordinary dysentery*, and he thereby suggests, though he does not formally propose the question, whether the anatomical changes described by different writers as characteristic of dysentery, do not include the lesions proper to two distinct diseases,—one characterized by diffuse inflammation, quickly running into gangrene, and the other by inflammation of the solitary glands leading to the formation of numerous distinct ulcers.

In order to determine this question, we must consider not merely the two principal forms of local morbid change in the colon, just mentioned, in their relation to each other, but also the relative frequency with which disease in other parts of the body is associated with the one and the other of these forms of altered structure respectively; the symptoms, local and general, that attend each during life, and the circumstances under which each occurs, or the causes by which it is produced. The essential difference or the identity of the two forms of disease can be concluded only after an examination of all these points. At present, however, we can merely inquire whether the local lesions, considered by themselves, compel us to the conclusion that these two varieties belong to distinct diseases. And this question, it appears to me, must be answered in the negative. There is every grade of transition from the one form of lesion to the other. In cases where the small round ulcers produced by the destruction of the solitary glands predominate, there are frequently, and in severe cases perhaps most frequently, seen at the same time long strips of mucous membrane in the state of sphacelus, or large ulcers formed by the casting off of such sphacelated portions of membrane. And, on the other hand, when the extended gangrene is the predominant form of the disease, the portions of mucous membrane not totally disorganized, frequently present the solitary glands enlarged or sloughing, or ulcers resulting from their total destruction. Several of the preparations now on the table illustrate this fact. It cannot be denied, however, that often where the erythematous inflammation and gangrene affect continuously nearly the whole length of the large intestine, no sign can be perceived of especial affec-

* See, for example, the dissection at page 62 of Dr. Parkes's work, "Researches on the Dysentery of India," and his observations on the appearances in the colon at page 64.

tion of the solitary glands. Let us see whether this is capable of explanation without supposing the disease to be, in such cases, essentially different in its nature. It will be remembered, that even in the mildest cases of dysentery; and where the special affection of the solitary glands was most evident, the inflammation had still extended over the inner surface of the intestine to a considerable distance around the glands: there were redness, desquamation of the epithelium, with effusion of fibrine, and sometimes loss of vitality of a superficial layer of the mucous membrane subjacent to the epithelium. In these cases we may suppose that the blood was but little altered from the healthy condition, and that the inflammation, therefore, which was diffused around the foci of the disease did not take a destructive form. But there are conditions of the circulating fluid where inflammation, set up in one point of a tissue, not only spreads with great rapidity through the capillary net-work, but also induces at once, in all the parts it reaches, complete stagnation of the circulation and gangrene of the tissue. And if in a case of dysentery such a state of the blood existed previously, or was produced at the same time that inflammation attacked the solitary glands of the large intestine, the consequence would be that no small points of disease indicating the situation of the affected glands would be seen,—all these would be lost in the extensive and inflammatory congestion and mortification. This hypothesis describes correctly the kind of inflammation which exists in the cases of dysentery here referred to, but it may appear not quite satisfactorily to account for the *total* absence of all appearance of enlarged and sloughing solitary glands on those parts of the mucous membrane of the colon which are only in the state of inflammatory congestion,—such as are seen in some parts of this drawing. To explain this, however, I would suggest that possibly only the inflammation set up by the primary cause of the disease has the tendency to produce especial tumefaction and mortification of the glands, and that inflammation secondarily excited does not affect them more than other parts of the mucous membrane. Assuming this, we may explain the cases in question by saying that in these instances all the glands attacked by the primary cause of the disease are involved in the gangrenous parts, while the inflammation in the portions which are not disorganized has arisen secondarily by sympathy with the neighbouring intensely affected portions, and not from the direct operation of the original cause of the disease;—and that on this account the glands are not apparently affected.

It should be borne in mind, too, that the solitary glands of the large intestine are not

at all times in the same state of development; and that when dysentery attacks them in their lowest condition as to size and activity of function, they will in all probability show themselves less conspicuously in the inflamed mucous membrane. This circumstance may account for the absence, in some cases of dysentery, of the distinctive character usually given by the especial activity of the diseased process in these glands.

It is then possible, I think, to account for all the forms of local lesions found in dysentery without adopting the view that two diseases of essentially different nature are engaged in producing these lesions. But still, the difference of anatomical characters in different cases is sufficiently great to accord with such a view, if the affections of other parts complicating the disease in the large intestine, if the symptoms and the influences causing the disease in the respective cases, should present well marked differences.

Keeping this question in view, then, I proceed to notice the *lesions found in other organs than the large intestines*, when cases of dysentery have terminated fatally.

In 5 out of 28 dissections of cases of dysentery not complicated with typhus, I have found redness and roughening of the mucous membrane of the ileum extending from the ileocolic valve to a greater or less distance up the intestine. The same appearances are noticed in about the same proportion of cases by writers on dysentery who have observed the disease in all climates. This redness and roughening of the epithelial surface of the mucous membrane of the ileum seems not to be a part of the primary disease, but to be due to the extension of the inflammation from the colon to the ileum by sympathy of surface. In a small proportion of cases the solitary glands of the ileum appeared enlarged, but not to such an extent as to constitute a feature of any importance.

The inflammation of the lower portion of the ileum, as a complication of dysentery, has been more frequent in the diffuse gangrenous form of the disease than in the form where the effects of inflammation are more especially evident in the solitary glands; but this circumstance affords no ground for regarding the diffuse gangrene of the colic mucous and submucous coats as a distinct disease, for the greater intensity of the inflammation in this form of dysentery, and its greater disposition to spread, sufficiently explain the more frequent affection of the ileum in such cases.

The absorbent glands connected with the inflamed colon rarely present any strongly marked signs of disease. This is remarkable, since the mesenteric glands are so constantly affected in typhoid fever, although the evidences of inflammatory action are

often much less extensive in that disease than they are in the colon in dysentery.

The other organs which have been the seat of inflammatory disease in cases of dysentery are the lungs, the pleura, and the peritoneum. The lungs are the only organs besides the ileum inflammation of which has frequently complicated cases of dysentery. In several cases the dysentery and the pneumonia have commenced at the same time, and have run their course together; and in one case inflammation, terminating in gangrene of the lung, came on in the course of a dysenteric attack. In the vast majority of cases, however, the dysentery has run its course without being complicated with any pulmonary symptoms. The morbid appearances met with in all the parts of the body I have mentioned attend with equal frequency both forms of lesion in the large intestine. The morbid appearances found after death, therefore, afford no sufficient reason for believing that two distinct diseases have been confounded under the term "dysentery."

When the close relation subsisting between dysentery and suppurative disease of the liver in India is considered, it cannot but appear remarkable that, amongst the many hundreds of cases of dysentery which have occurred in the Millbank prison during the last seven years, not one has been complicated with hepatic abscess. The medical records of the establishment, too, which reach back to the year 1824, afford no grounds for even a suspicion that such cases ever occurred amongst the prisoners. Dr. Budd* has adduced abundant reasons for the belief that, where hepatic abscess is associated with dysentery, the former disease is in all or most cases an effect of the dysentery, through the blood conveyed by the portal vein to the liver having been vitiated by morbid or putrid matters absorbed from the ulcerated or gangrenous surface of the large intestines. He has gone further, and has shewn, that, in a large proportion of the cases in which abscess of the liver exists without dysenteric disease of the large intestine, there is ulceration of some other of the mucous membranes which return their blood to the liver through the portal vein. But, although ulceration or sphacelus of the intestinal mucous membrane is capable of exciting abscess of the liver, and is its most frequent cause, yet some special conditions must be present, in order that the effect shall be produced: for the frequency with which ulcers seated in different parts of the gastro-intestinal mucous membrane, or even ulcers in the same parts, but due to different diseases, occasion abscess of the liver, is far

from being the same in all cases. Thus, as Dr. Budd has pointed out, hepatic abscess is never seen in conjunction with the ulcerated intestine in typhoid fever, and is very rarely associated with the ulceration of the intestines in phthisis. It has not, however, been owing to the peculiar seat or nature of the intestinal lesion that the dysentery of Millbank has been unattended by the hepatic complication; for in this dysentery in the Millbank prison the disease of the mucous surface, both as to its seat and in its nature, has been the same as in the dysentery of India, with which hepatic abscess is so frequently associated. The generally less severe character of the disease at Millbank likewise cannot be the cause of the difference; for amongst the cases of hepatic abscess with dysentery recorded by Mr. Annesley and by Dr. Parkes, there are several in which the amount of disease in the large intestine was inconsiderable. We must, then, seek some other explanation of the fact that dysentery amongst the prisoners at Millbank has not led to the formation of abscess in the liver; and in this inquiry we must first notice the important circumstance that the association of hepatic abscess with dysentery has not been equally frequent in all countries. When we examine the numerous dissections of fatal cases of dysentery in India recorded by Mr. Annesley, Mr. Twining, and Dr. Parkes, we find that hepatic abscess existed in rather more than one-half the cases. In the 51 cases, for example, detailed by Mr. Annesley, there were 26 in which the dysentery was attended with abscess of the liver. On the other hand, M. Broussais, who relates 17 cases, with dissections, of fatal dysentery, does not mention his having found abscess of the liver in any one instance, although he generally notices the condition of that viscus. And Rokitansky,* in his dissections of cases of dysentery, has never found the liver visibly diseased. Again, in China, where dysentery is very fatal to Europeans, the infrequency of hepatic disease is very remarkable. This is testified to by several medical officers of the army and navy who have had opportunities for observing the diseases of our troops both in that country and in India.† It is established, too, by the statistics of the hospital-ship Minden, which was stationed at Hong Kong during the military and naval operations in China, and was under the superintendence of Dr. John Wilson.‡ Amongst 61 fatal cases of dysentery in which the bodies were examined after death, there

* Med. Jahrbücher des Oesterreich-Staates, Bd. xx. 1840, p. 81.

† Transactions of the China Medical-Chirurgical Society for the year 1845-1846, pp. 14, 25, and 49.

‡ Medical Notes on China. London, 1846, p. 258 et seq.

* Diseases of the Liver. London, 1845, pp. 49-73.

were only two in which hepatic abscess was found.

The infrequency of hepatic abscess in these instances might be ascribed to the greater coolness of the climate, compared with that of India. It is certainly conceivable that the high temperature of the climate of Bengal may induce vascular turgescence of the liver, and thus favour the formation of abscess under the influence of such an exciting cause as a vitiated state of the portal blood; but difference of temperature cannot be the only reason why dysentery gives rise to hepatic abscess in one country and does not in another; for in the West Indies, where the prevalence of dysentery is often very great, and where the heat is intense, hepatic abscess is by no means a frequent complication of the disease. In Europe, too, abscess of the liver has been observed in some epidemics of dysentery, while in others it has been absent. During the epidemic which prevailed in Dublin in 1818, Dr. Cheyne met with hepatic abscess in 4 of the 30 cases which he has published; but, as I have before mentioned, M. Broussais did not find it in the camp dysentery which he observed in the years 1805 and 1806. It would therefore appear that the malaria causing the dysentery has at some times and in some places the property of predisposing to abscess of the liver, and at other times and in other places has not this property. In the case of the prisoners at Millbank, however, another influence possibly comes into play: it is easy to conceive that persons whose diet is most regular and unstimulating, and in quantity not superabundant, would be less liable than others to suffer from a turgid state of the capillary system of the liver, and, consequently, would be less prone to the occurrence of suppuration in that organ. At all events it is certain that, besides difference of climate, and difference in the properties of the malaria itself, there is another cause which may affect the frequency of this serious consequence of dysenteric disease of the colon. All the best authorities on the diseases of India concur in stating that hepatic abscess is extremely rare among the natives of that country, though dysentery is very prevalent amongst them.* This may be owing to the original constitution of the Hindoos; but it is impossible not to remark the great similarity which exists between their simple and spare diet and that of the criminals in a British prison. In both instances it is possible that the diet contributes to render the hepatic disease an infrequent complication of dysentery.

There are two *general diseases* of the

system with which I have found dysentery very frequently combined: one a chronic disease, namely, tubercular phthisis, or the tubercular cachexia generally; and the other an acute disease, namely, continued or typhoid fever.

The old notion, that pulmonary phthisis is less frequent in malarious districts than in those that are healthy, was revived two or three years ago in France. The state of the atmosphere which produced fevers of various kinds afforded a protection, it was said, against tubercular disease, and it was proposed to send persons who evinced a phthisical tendency to pestilential localities in order to preserve their health.

The converse of this doctrine seems to be held by Rokitansky. He says that persons affected with tubercular disease of the lungs in active progress are never, or only very rarely, attacked by either typhus, or cholera, or dysentery.*

Now, if the former of these doctrines include the atmospheric cause of dysentery, amongst those kinds of malaria which prevent the development of tubercular disease, and if it be admitted that the dysentery at Millbank has been caused by an atmospheric poison, then the doctrine in question will be opposed to the fact that, in the Millbank Penitentiary as well as in several other prisons, both dysentery and tubercular disease have prevailed together during many successive years.

The mortality from dysentery, and other bowel complaints, in the Millbank Penitentiary, though not absolutely great, has been nearly five times as considerable in proportion to the population as the mortality from the same diseases amongst adults in the metropolis generally. Yet tubercular phthisis has been the most frequently fatal disease; the rate of mortality from phthisis, and the other tubercular affections, having been nearly four times as high amongst the prisoners as amongst persons of the same period of life, in the general population of London.

In an American prison, that of Sing Sing in the state of New York, there has been the same prevalence of dysentery and other diseases undoubtedly produced by the agency of malaria, and the same excessive mortality from tubercular phthisis.

The results of the examinations of the bodies of prisoners who have died in the Millbank Penitentiary are equally at variance with Rokitansky's notion that dysentery will not attack persons labouring under tubercular disease. Amongst 27 cases during the last six years in which death was caused principally by tubercular disease of the

* See, for example, Twining, on the Diseases of Bengal, p. 3. Parkes, op. cit. p. 118.

* Med. Jahrb. d. Oester. Staates, Bd. xvii. 1838, p. 226; Bd. xix. 1839, p. 423.

lungs, there have been 10 in which that disease was combined during the last few days or weeks of life with active dysenteric disease of the large intestines. As Rokitsky regards only the gangrenous inflammation of the colon as the true dysenteric process, it is necessary to say that in three of the cases the disease was of that character. With regard to the other cases, however, the symptoms during life, the prevalence of dysentery in the prison at the time, the signs of active inflammation in the mucous membrane of the colon or rectum, and the absence of tubercular matter from the margins and bases of the ulcers, left no doubt that the lesions found in that part of the alimentary canal were really the effects of dysentery, and not of the tubercular disease itself.

But let me remark, that, although in these instances tubercular ulcers of the large intestine were not mistaken for dysenteric ulcers, yet in their seat the ulcers of tubercular disease and those of the discrete form of dysentery are identical, and their characters in the chronic stage very similar. They are both seated, at their commencement, in the solitary glands, and, during their extension, assume similar forms, and frequently acquire the same thickened edges. In the base and edges of the tubercular ulcer, however, small points or masses of tubercular matter can generally be detected.

It is interesting here to notice not only the remarkable proneness of the glands of the intestines to disease, but also the especial liability of one set of these glands to one disease, and of another set to other diseases. Thus, while the glands of the large intestines are the seat of dysentery, those of the small intestines are the special seat of the intestinal lesion in typhoid fever. Tubercle attacks the glands in the whole length of the intestinal canal, those of the small intestines, however, most frequently; while another chronic disease, carcinoma, frequently affects the submucous vascular tissue of the large intestines, beginning perhaps in the solitary glands; and but rarely attacks the small intestines.

With respect to the combination of dysentery with typhus or typhoid fever, of which Rokitsky denies the occurrence,* I shall at present only remark that at Millbank prison it has been frequent; and that in the fatal cases the characteristic lesions of the two diseases were many times found perfectly developed. The drawings I now show represent the morbid appearances in the small and the large intestines of two such cases.

I fear that the length to which my ob-

servations on the morbid anatomy of dysentery have run has been tedious. I have been led into this error, if such I have committed, by the varieties of opinion and statement to be found in the writings of the best authors on the subject I have been discussing.

I should have no such excuse to offer were I equally prolix in speaking of the *symptoms* of dysentery, for, respecting these, in all important points, the most perfect agreement exists.

Sydenham describes the disease as beginning with "a rigor succeeded by heat of the surface, and afterwards by tormina and purging. The febrile symptoms" he says, "may be absent. The attack then begins with the tormina which purging soon follows. Extreme pain and bearing down of the intestines attend each of the alvine discharges, which are very frequent. The discharges themselves consist of mucus, but now and then, after many of these mucous stools, one of a feculent character is passed without much pain. The mucous discharges are generally mixed with blood. But sometimes no blood is seen in the stools throughout the whole course of the disease."*

This description of dysentery, as it was observed by Sydenham in London in the latter half of the 17th century, comprehends nearly all the most prominent features which have been assigned to the disease by later writers, in whatever part of the world they have observed it.

The dysentery amongst the prisoners at Millbank, as I said in my former lecture, has presented three grades of severity. The cases belonging to the two less severe degrees of the disease have been by far the more numerous, and these would for the most part be accurately described by the passage I have quoted from Sydenham.

The occasional absence of febrile disturbance noticed by this close observer of disease, is especially important in a practical point of view. For, the cases having this character, we are apt to treat lightly; and then, from prompt measures not having been adopted in the early stage, a troublesome and obstinate diarrhoea often remains for a long period.

In the cases of the second degree of severity there is almost always heat of skin, thirst, and loss of appetite. The tongue is red, and less moist than natural, and the pulse slightly accelerated. But still, even in these cases, the feverish disturbance is usually not more than may be regarded as simply symptomatic of the local inflammation. And, as Willis remarks, in his account of the "*Dysenteria Cruenta*" which

* Oester. Jahrb. Bd. xvii. p. 225.

* Sydenham, *Observat. Med. circa Morb. Acut. Hist. et curat. Lect. iv. cap. iii.*

raged in London in the year 1671, there is no rapid exhaustion of the strength. This is a character distinguishing most cases of the two milder degrees of dysentery from typhoid fever and the exanthematous fevers.

Some differences in the symptoms, dependent on the part of the large intestines which is affected, are important in relation to the treatment of the disease. Sydenham's description applies more especially to those cases in which the rectum and sigmoid flexure of the colon are the parts affected. Here the tenesmus is extreme, and is accompanied with pain and a sense of weight in the sacrum, and sometimes with irritation of the bladder: symptoms which are explained by the nervous sympathies and the anatomical relations of the rectum. The fæces in these cases, too, are often solid or nearly so when discharged.

But where the lower portions of the canal are *not* affected, tenesmus is necessarily absent; the stools are less frequent, and the mucus and blood are mixed with the fæces when discharged. This is more especially the case when only the cœcum and the ascending colon are affected, and then, indeed, the blood and mucus can seldom be distinguished from the liquid fæculent matter with which they are mixed. These are probably the cases to which Sydenham referred when he said that sometimes no blood is seen through the whole course of the disease.* It is easy to see how these differences arise. The contents of the small intestines naturally enter the colon in a perfectly liquid state, and gradually acquire consistence as they approach the rectum. Now, if the cœcum and ascending colon are the seat of inflammation, these liquid matters are at once propelled onwards, carrying with them and concealing the morbid secretions of the mucous membrane, and are discharged as liquid fæces. But if the inflammation is seated in the sigmoid flexure, or in the rectum, the matters poured into the colon from the ileum are detained for some time in the ascending colon and the transverse colon, which are healthy, and there acquire consistence before they are discharged. It seems, indeed, that their passage onward is resisted by spasmodic contraction of the intestine immediately above the inflamed portion. The mucus and blood poured out by the inflamed membrane are, therefore, expelled without admixture of fæculent matters; and when at length the spasmodic contraction of the middle portion of the intestine gives way, the contents of the upper

portion are discharged in a solid, or nearly solid, state. From this account it is obvious, first, that the absence of blood and mucus from the discharges proves no difference in the nature of the disease; and, secondly, that the degree of consistence of the excreted fæcal matters indicates with some degree of accuracy the seat of the dysenteric inflammation. There are, however, exceptions to the latter rule. Masses of long-retained fæces, *scybalæ*, may be discharged at the commencement of a case in which the cœcum is the chief seat of the disease. And, on the other hand, there may be such an increased irritability of the canal that the contents of the intestine, as soon as they enter the colon, are propelled through the whole length of the large intestine, and discharged in a quite liquid state, although the rectum be the only part really affected with inflammation. The morbid irritability of the alimentary canal is sometimes so great that food or drink taken into the stomach excites an immediate discharge of the contents of the colon. This is frequently seen in the last stage of severe cases of dysentery, and is occasionally observed in very slight cases. Although, therefore, the degree of consistence of the fæces is a point worthy of observation, it cannot constantly be depended on as an indication of the *seat* of the disease. A more constant guide is the situation in which pain and tenderness chiefly exist.

The *severity* of the disease may generally be estimated from a consideration of the local and general symptoms taken together, and more particularly from the amount of pain and tenderness, the frequency and quantity of the discharges, and the degree of febrile disturbance. But I may remark, that when the lower part of the intestine is the seat of the disease, a tolerably accurate estimate of the extent of mucous membrane affected may be formed from the mere quantity of bloody mucus discharged.

The source of this bloody and mucous discharge in the acute stage of dysentery is not the solitary glands, but the tubular follicles of the mucous membrane. This is rendered certain by the microscopic characters of the discharged matters. When I spoke of the anatomy of the mucous membrane, I mentioned that the granular matter contained in the solitary glands was composed of solid particles of flattened figure and strongly defined outline, resembling the nuclei of certain cells or globules; while the contents of the follicles were, at one time, epithelial particles, at another round nucleated globules, exactly resembling mucous globules. Now the bloody mucus discharged in dysentery is composed of such nucleated globules, generally mixed with numerous blood-discs. Sometimes the globules are seen here and there still connected together in a cylindrical

* Sydenham may here have alluded to the cases in which mucus is discharged in large quantity unmingled with blood. In these cases, which are rare in comparison with those of the ordinary "bloody flux," the inflammation of the large intestine is of a languid character, and has tendency to assume a chronic form.

mass, such as they form while in the tubular follicle. The puriform matter occasionally discharged in acute dysentery has probably the same source. In the chronic stage of dysentery, also, pus, or a puriform matter, is often excreted. But here this matter seems to have a different source. It probably comes, in the slighter cases, from the enlarged and diseased solitary glands, and in the more severe cases from the numerous ulcers which have been left after the destruction of the glands, and of smaller or larger portions of the mucous membrane.

The principal characters which have distinguished the *cases of dysentery of the greatest severity* observed at Millbank, are indicated by Sydenham, when he says that, under certain circumstances, "fever arises, the tongue becomes thickly coated with a whitish mucus, or even black and dry, the strength and powers of the system are depressed, and all the signs of an ill-conditioned fever manifest themselves." These are the cases in which the mucous and sub-mucous coats are, after death, found extensively mortified and sloughing. Here the pain is generally severe at first, but afterwards ceases to be complained of, while the functions of the sensorium become disturbed. In some cases the pain has ceased suddenly, and then a greatly increased feebleness and acceleration of the pulse, and a diminution of the heat of the surface occurring at the same time, seemed to denote the accession of mortification in the inflamed intestine. In a few of these cases no pain in the abdomen has been complained of throughout the whole course of the disease.

The *discharges* from the intestines in this form of dysentery are muciform and bloody at the very commencement of the attack. Afterwards they are generally quite watery, though still bloody, and, when mortification has taken place, are excessively foetid. At a later period of the disease, the discharges are no longer bloody, nor so watery as at first. They generally consist of very liquid fæces, in which occasionally portions of the sloughing coats of the intestine can be distinguished.

The local as well as the general symptoms of this most severe form of dysentery differ, therefore, in kind as well as in degree from the symptoms of those less aggravated cases of the disease, in which the lesion consists, not in diffuse gangrene of the mucous membrane of the large intestine, but in destructive inflammation, confined to the solitary glands, or at most affecting only the prominences of the rugæ to a limited extent. The *most important differences in the symptoms* are those which indicate that the blood has undergone a morbid change in composition, and that the powers of the nervous system are disturbed. These symptoms are,

the general febrile disturbance, the quick and feeble pulse, the dry and often brown tongue, the loss of muscular power, frequently intense headache, delirium, and sometimes subsultus tendinum. Many of these symptoms show themselves before mortification of the mucous membrane can have taken place, though they afterwards increase in degree. They seem, in fact, not so much the consequence of the local lesion as the manifestation of that altered state of the circulating fluid which causes the inflammation of the large intestine to take the diffuse gangrenous form. This form of the disease has, at the Millbank prison, generally attacked the weakest subjects; but there have been exceptions to this rule. Three strong and apparently healthy men have perished from it, and the same thing has been observed elsewhere. We must conclude, therefore, that the peculiar character assumed by the disease in these cases has been due, not to anything originally morbid in the systems of the patients, but to the atmospheric poison, which produces the dysenteric inflammation around the solitary glands of the large intestines, being itself modified, or perhaps I should rather say, combined with another poison, which alters the composition of the blood.

The difference, then, between this *diffuse* gangrenous form of dysentery and the more common *discrete* form, for to that question I must once more return, seems to me to be the same in nature and degree as that which exists between the malignant scarlatina and the benign form of that disease. And this seems, indeed, to be the view taken by those Indian practitioners who have had the largest experience in the observation and treatment of dysentery. They distinguish this form of the disease as the adynamic dysentery, while the milder form they term simple dysentery.

Thus far I have noticed only those characters of the dysentery of Millbank prison which it possesses in common with the same disease in other localities. I must now mention a class of symptoms which has been observed in connection with dysentery, as far as I know, only in that establishment. I allude to the disorders of the nervous system, to which a separate chapter is devoted in Dr. Latham's account of the epidemic which prevailed in the Penitentiary in the year 1823. "No part of the disease," Dr. Latham there says, (p. 78), "was more striking and characteristic, none more formidable and difficult to treat, than that which declared itself through the medium of the brain and nervous system." Some slight nervous affections, tremors, cramps or spasms, and various degrees of mental despondency, were observed by Dr. Latham and his colleague, Dr. Roget, at their first visits to the Penitentiary, but were regarded

by them only as symptoms of constitutional weakness. "In process of time, however," Dr. Latham observes, (p. 79) "disorders of the brain and nervous system became more and more frequent, and of various kinds; headache, vertigo, cramps, and twitchings of the limbs, delirium, convulsions, and apoplexy. But since these disorders did not immediately discover themselves in all their variety and magnitude, it was not until after much observation that we were enabled to tell their genius and character, and to know that they constituted one form of the predominant disease; that they were not contingent upon the flux, nor the flux contingent upon them; that either might exist separately, although they were generally found in combination; and that both arose from a morbid condition, essentially the same, but falling upon different parts." Of the nervous affections here referred to Dr. Latham gives many striking examples.

Now the affections of the nervous system which formed so remarkable a feature of the epidemic of 1823 have been witnessed by myself during a more recent outbreak of the disease at the Penitentiary. Bowel complaints in some form or other have almost always been prevalent. Not so the peculiar nervous disorders described by Dr. Latham. They have shown themselves only during a limited period, comprehending the last few months of the year 1841, and the greater part of 1842.

It was in the autumn of the former year that I first met with several cases of nervous affection which were perfectly new to me. Two or three women, otherwise apparently in perfect health, complained of inability to open their mouths except a very short distance, and on examining the muscles which bring the jaws together I found them rigid. Others had cramps in their hands, so that the fingers were drawn tightly together without being much flexed, and could not be moved by the patients themselves; while the attempt by another person to alter their position gave great pain. These symptoms were unattended by diarrhoea, constitutional disturbance, or any other morbid state whatever, and they soon passed off. The patients who were the subjects of them were females, and were not many. I was interested, however, in the appearance of these nervous disorders from their correspondence with some features of the disease described by Dr. Latham.

During the months of December 1841 and January 1842 several cases of dysentery of unusual severity occurred amongst the prisoners, and in the succeeding months they became so frequent as to constitute an epidemic. Then it was that the nervous disorders appeared in a more formidable shape. One young man, 17 years of age, of

most healthy aspect, was attacked with dysentery of active inflammatory character, but presenting no feature calculated to cause alarm. On the seventh day of his illness, cramps, attended with agonising pain, seized his limbs, and soon affected also the trunk of his body. These cramps were as severe as are ever seen in cholera, but were not attended by the depressed circulation and coldness of the surface observed in that disease. On the contrary, the pulse was full and bounding, and the surface hot, and steaming with a profuse perspiration. The patient in the midst of his sufferings retained perfect consciousness, and complained of a peculiar sense of heat all over his body, which he described as the blood boiling in his veins. This sensation seemed to distress him more than the pain produced by the cramps. The spasms abated from time to time, never, however, entirely leaving him, and each fresh paroxysm was more violent than the preceding one. At length they became so severe and general that his body was twisted from side to side, and his features dreadfully contorted, as we see the body and features of a patient in a fit of epileptic convulsions. His mind, however, remained clear to the last. Calomel and opium in large doses, venesection, cupping along the spine, warm baths, and antispasmodics, were the remedial means employed with some temporary relief. The spasms abated for a time after each of these measures, but soon again became more terrible than before; and at length the muscles of inspiration seemed to be implicated, and the sufferings of the patient were cut short by suffocation. This happened on the 11th day of the dysenteric attack, and the 3d day of the nervous affection. The body was examined 24 hours after death in the presence of Dr. Burrows. The lesions I have described as belonging to the milder form of acute dysentery were found in the large intestines. But the brain and spinal cord were perfectly free from any appearance of disease.

Another variety of these severe nervous affections was seen in a young woman, 22 years of age, who had just recovered from an attack of dysentery which had commenced a month previously. She was suddenly seized, late in the evening, with general cramps, and loss of feeling in the extremities. The next day I found her lying extended in bed, with her legs, arms, and trunk, quite rigid, and complaining of pain across the loins, and of a dreadful sense of oppression at the pit of the stomach. The skin of the extremities was quite insensible to external impressions, and she appeared to suffer no pain in them. The cramps gradually ceased,—first in the trunk, and then in the extremities. On the 4th

day of the attack her limbs were no longer rigid, but she had lost the power of moving them. On the following day there was a temporary return of cramps in the legs and forearms, accompanied with pain; but they soon finally ceased, and the power of moving the limbs returned. The patient's nervous system, however, had evidently suffered a severe shock. Her pulse, which, during the continuance of the spasms, had been scarcely at all altered from its natural character and frequency, became rapid and feeble; hence, although dysentery had reappeared, together with frequent vomiting, on the 4th day of the nervous attack, active measures to check it could not be thought of. The dysentery and vomiting continued, and hastened her dissolution, which took place on the 19th day from the first appearance of the spasms. Here, again, the large intestines presented after death the effects of dysentery in an early stage; and the mucous membrane of the duodenum was red, thickened, and mammellated, apparently from inflammation; but the brain and spinal cord were quite free from diseased change.

A third fatal case occurred, in which dysentery on the 4th day of its duration became associated with severe cramps in the muscles of the extremities, the neck, and face. The cramps ceased at the end of four days, and death was caused on the 22d day of the dysentery by inflammation of the lungs. The brain and spinal cord in this instance were not examined.

Now in these three severe cases the symptoms indicated an affection of the powers of sensation and motion, while the higher sensorial faculties were unimpaired. And of like character were a great number of slighter cases, which were frequently occurring amongst the prisoners. They consisted for the most part of cramps in the extremities, with now and then a similar affection of the muscles about the lower jaw.

But there were other cases in which the brain was manifestly implicated. The patients, sometimes affected with slight dysentery, sometimes having no affections of the bowels whatever, complained of intense headache, and had a heavy expression about their eyes; while, except in the instances where dysentery existed at the same time, all their bodily functions were properly performed. Others lay with their heads buried beneath the bed-clothes, not sleeping nor in a state of complete stupor, but in a condition of mixed stupor and apparently wayward obstinacy. It was with the utmost difficulty that any information as to their sensations could be drawn from them: their state had a resemblance to some forms of hysteria, yet there was no characteristic hysterical symptom. And on the other hand, there was no undue heat of the surface

of the head, nor any other sign of vascular excitement. The state here described was sometimes combined with cramps in the extremities, or with slight dysentery, but often it occurred alone. Here, again, the patients were in most instances females.

Had I seen only those nervous affections which consisted in cramps, convulsions, and partial loss of sensation in the limbs, I should certainly not have placed in the same category with them, as part of the prevailing epidemic, one remarkable case in which the nervous disorder assumed the form of cataleptic ecstasy. But the variety of nervous disorders last mentioned made it evident that not merely the motor and sensitive functions, but the faculties of thought and volition also, might be disturbed by the noxious influence from which the prisoners were so generally suffering, and thus afforded grounds for regarding every unusual affection of the nervous system which occurred at the same time as an effect of the same general cause. The case to which I refer was that of a young man, 22 years of age, who was first attacked with an affection of the bowels in the form of profuse serous diarrhoea; quarts of watery fluid being discharged from his bowels in the course of 24 hours. He complained of intense pain in the left hypochondriac region, and of tenderness of the whole abdomen. Notwithstanding the absence of all general signs of the presence of inflammation, I was induced to use local depletion and mercury, with opium, the means which had proved most successful in the treatment of other forms of the prevailing intestinal disease. In this case they wholly failed. And then metallic astringents, with opium, were substituted at the suggestion of Dr. Burrows, who had been requested by the Superintending Committee of the Penitentiary to afford his valuable professional aid at the time when the epidemic prevailed most extensively. The new remedies immediately arrested the diarrhoea, but now a fresh train of symptoms presented themselves. The patient's mind seemed no longer to take cognizance of the impressions made on his senses. He sat up in bed frequently repeating half aloud a sentence consisting of two or three words. His eyes were open, but he did not seem to observe surrounding objects: when questions were put to him he did not answer, or only repeated the last word or two of the question. When his chin was depressed he mechanically protruded his tongue, and kept it protruded until his lower jaw was raised again; he then withdrew his tongue, and allowed his mouth to be closed. When his arm was raised he kept it in the position given to it till it was returned to its former place by another person. Food and medicine he swallowed when they were put in his

mouth, but never expressed either repugnance at the one or desire for the other. He appeared conscious of no suffering. His skin was cool, his tongue moist, pale, and nearly clean, and his pulse slow and rather full. In this state he continued three days; the nervous symptoms then gradually passed off, and merely slight diarrhoea remained. It may appear questionable whether the occurrence of this case during the epidemic of dysentery in the year 1842 was not accidental, and whether in its origin it was not wholly unconnected with the cause or causes by which that epidemic was produced. But besides the occurrence at the same time of other cases in which the minds of the patients were certainly affected, though in a less degree, and besides the testimony of Dr. Latham to the appearance of nervous symptoms of the most unusual and various characters in the year 1823, there was the fact that the nervous affection in this case was immediately preceded by diarrhoea, tending strongly to establish an alliance between it and the other forms of disease which more especially characterised the epidemic.

In their nature all the nervous disorders seemed to resemble each other in being merely functional: that is to say, they were not dependent on inflammation or any appreciable change of structure either in the nervous centres or in their investing membranes. In this respect they resembled the

cramps and convulsions generally attendant on cholera; but, as I have before remarked, the patients who suffered from them presented nothing of that state of collapse,—of depressed circulation, and diminished animal heat, which characterises cholera.

I have sought in the works of many writers on Dysentery for evidence of the occurrence of nervous affections of a similar kind in conjunction with that disease in other localities, but have found none. Even Dr. Copland, who has himself had the opportunity of witnessing dysentery in many climates, and appears to be acquainted with almost all that has been written on the subject, makes no mention of any instance in which the disease was attended with nervous affections such as those observed at Millbank.

It is, however, well known that the most formidable of all functional affections of the nervous system, tetanus, is especially prevalent in hot climates. And this fact must be borne in mind when we attempt to explain the nervous disorders which have during two epidemics of dysentery affected the prisoners in the Penitentiary. The inquiry, however, into the causes on which the prevalence of dysentery and these nervous disorders in the Penitentiary depended, must be postponed to the next lecture, when I shall, in conclusion, offer a few remarks on the treatment of dysentery.

LECTURE III.

Cause of the dysentery in the Millbank Penitentiary—Influence of the seasons and the weather on its degree of prevalence—Its alliance with cholera and fever—Causes of the bowel complaints prevalent in other prisons and in some workhouses and lunatic asylums—Susceptibility of the influence of malaria produced by imprisonment—Characters of the site of the Millbank Penitentiary—General doctrines of the causes of dysentery—It is produced by a poison absorbed into the system from without—Mode of action of this poison—Cause of the peculiar nervous disorders observed in the Penitentiary—State of the nervous system produced by separate imprisonment—Treatment of dysentery—Of the simple inflammatory or sthenic form—Of the asthenic form—Conclusion.

THE first subject for inquiry to-day is the cause of the dysentery which has from time to time prevailed at Millbank. The physicians who had the medical charge of the Penitentiary during the epidemic of the year 1823, reported as their final opinion that the disease had been produced by a local noxious influence. They adduced good reasons for holding this opinion, and subsequent occurrences have shewn its correctness. Diarrhoea of a very mild character has seldom been altogether absent from the Penitentiary: dysentery has been a frequent disease there; and in one year this disease has prevailed as a severe and fatal epidemic.

It cannot be doubted, therefore, that the cause of the disease is a noxious influence fixed on the spot, but capable of undergoing variations in its power of action. The term noxious influence, however, is a very general one. Cannot the nature of this influence be more closely defined? I think it can. Here, as in other instances where dysentery is endemic in prisons, workhouses, or lunatic asylums, the cause really producing it is, I believe, a malaria rising from the surface of the ground around the building. There are other influences from which dysentery might be supposed to arise, namely, diet, the water used as drink, defective ventilation, and defective sewerage. None of these, however, can have been the efficient cause of the disease in the Penitentiary. I think it unnecessary to detail on the present

occasion the facts by which this has been rendered certain. I shall therefore at once proceed to adduce those reasons which, to my mind, prove the dependence of the disease on a gaseous poison or malaria derived from the soil of the surrounding grounds.

One of these reasons, and an important one, is the relative frequency of the cases of dysentery and bowel complaints in general at different seasons and in different states of the weather. They prevail most in the autumn and in the spring, especially in a wet autumn following a hot summer, or in a mild spring when the preceding autumn was wet and the winter severe; in other words, at those times when, from the state of the soil and atmosphere, the decomposition of the organic matters in the soil is necessarily most active. It may be recollected that the epidemic of the year 1823 commenced at the close of a very cold winter, and that the bowel complaints especially became prevalent and severe when mild spring weather ensued. The rise and progress of the epidemic of the year 1842 have a similar history. The latter half of the preceding year was remarkable for long-continued rains; and in the month of October the low garden grounds to the north of the prison were inundated, owing to the river overflowing its banks. A cold winter followed. In the latter part of February, 1842, the weather was mild, while the atmosphere was humid; and then it was that cases of dysentery suddenly became very numerous amongst the prisoners, and continued so throughout the month of March. From the beginning of April to the middle of July the weather was dry and cool, and the dysentery gradually subsided; but at the close of July very hot weather set in, and then the disease became prevalent more or less throughout the country, and again attacked a large number of the prisoners in the Penitentiary. At the end of September the hot weather was succeeded by a cold and dry state of the atmosphere, and the prison once more became very healthy. Lastly, about the middle of October, a moist and foggy state of the air ensued, and then dysentery returned, together with fever. This coincidence between the prevalence of dysentery and bowel complaints on the one hand, and particular states of the weather on the other hand, has been observed in

years when those diseases have been less prevalent than in the year 1842, and it seems to me reconcileable with no other theory of the cause of the diseases in question than that which ascribes them to the influence of a malaria rising from the soil.

Another class of facts, strongly supporting the same theory, are those which shew the close alliance between dysentery and other diseases which are more indisputably of miasmatic origin, namely, common cholera and fever. Not merely have epidemics of dysentery in the Penitentiary been preceded or followed by the prevalence of one or other of those diseases, but an attack of dysentery has often been ushered in by cholera, or has been combined with fever in the same patient. The direct transition from the choleric state, attended with rice-water evacuations, to a condition characterised by all the symptoms of inflammatory dysentery, has been often observed, especially in hot summers and autumns.

I have already mentioned the frequent co-existence of typhoid fever and dysentery in the same patient, in speaking of the morbid anatomy and of the symptoms of the latter disease. Dr. Latham, too, noticed the association of a fever with the other disorders which constituted the epidemic in the Penitentiary in the year 1823, and he has given an account of its relation to those other disorders according so closely with what I have myself observed, that I shall quote the passage:—

“While the flux of the bowels and the disorders of the brain and nervous system,” Dr. Latham says, “prevailed to their greatest extent, the cases of fever were rare. It was not until these complaints began to subside, that the fever shewed itself in a sufficient number of cases at once to make us accurately acquainted with its type. At no time did it pervade the prison to an equal extent with the other two forms of disease, but it had a just claim to be considered as a part of the disease of the Penitentiary; and the manner in which it was mixed up with the disorders of the bowels and the brain and nervous system led to the belief that they had all a natural relation to each other, and that they all sprang from one and the same morbid condition of the constitution at large.*”

The fever thus associated with dysentery at the Penitentiary has, during the period in which I have observed it, been generally characterized by the predominance of intestinal symptoms; sometimes, in fact, the affection of the larger bowels formed so important a part of the disease that it was difficult to say whether the patient was la-

bouring under fever complicated with dysentery, or under dysentery with unusually active symptomatic feverishness. In other cases the principal disease was evidently idiopathic fever, the symptoms of the bowel affection being by comparison only faintly developed.

I must here, however, remark that fever at the Penitentiary has not always had these characters: sometimes other organs than the intestines, namely, the brain or the lungs, have chiefly suffered. In a few remarkable instances, too, the sole characteristic feature has been profuse and constant sweating; the other symptoms being headache, pain in the loins, a thickly-coated tongue, a quick and feeble pulse, and great general debility. In these cases life was sustained, and health at length restored, only by the aid of wine and bark very freely administered. Cases of similar kind seem to have been observed by Dr. Latham, though, in the instances he notices, the sweating and debility did not come on till the second week of the disease, while in those I have seen they existed from the first. The fever, however, of most frequent occurrence in the Penitentiary has been that in which abdominal symptoms predominated, and in which, after death, not only the ileum, but the colon also, was found affected in various degrees.

The close alliance subsisting between dysentery and continued fever in London strongly impressed the mind of Sydenham. This is shewn not merely by his denominating a certain fever that was associated with dysentery the “dysenteric fever,” but also by his oft-quoted remark that “dysentery was the very fever itself, with this sole difference—that it was turned inwards, and discharged from the system through the bowels.”

A close connection, then, subsists between dysentery and fever; and the prevalence of dysentery, and of the bowel complaints allied to it, is greatest at those seasons, and in those states of the atmosphere, which most favour decomposition of organic matter in the soil. Now these two facts, together with the constant, or almost constant, presence of the disease in a mild form, and the absence of other causes capable of accounting for it, satisfy my mind that the dysentery observed in the Penitentiary at Millbank is really produced by malaria.

But several objections may be opposed to this conclusion. The first I shall notice is, that dysentery, or at all events, diarrhoea, is a common disease in prisons and work-houses, and in them appears to be produced by deficient nourishment; an increase in the diet having, it is said, in many instances, caused the disappearance of the disease.

To this objection I would answer, that

* Account of the Disease lately prevalent in the Penitentiary. London, 1823, p. 118.

the prevalence of bowel complaints in the establishment here referred to has not been caused by the poorness of the diet. During the summer of the year 1842, I visited many prisons, workhouses, barracks, and lunatic asylums, in various parts of England, with the view of gaining information which might throw light on the origin of the disease prevailing at Millbank. And I found that the degree of prevalence of bowel complaints in the different establishments bore no constant relation to the poorness of the diet, any more than it did to defective ventilation, or impurity of the water which the inmates drank. Dysentery and diarrhoea prevailed in barracks and lunatic asylums where the dietary was abundant, and were absent from prisons in which the allowance of food was scanty. In many instances, too, where a salutary effect had been produced by an increase of the dietary, the bowel complaints, before rife, had only been rendered less prevalent; they had not disappeared. It seemed, therefore, impossible to believe that poorness of the diet was the primary and essential cause of the disease. On the other hand, I found that the frequency of bowel complaints in the different establishments I visited *did* bear a relation to the character of their sites. Where those complaints were of frequent occurrence, there the site of the building was low, the ground around it damp or imperfectly drained, and the subsoil often formed of peat or clay. Where bowel complaints were infrequent, the site was elevated, the soil dry, and the subsoil generally composed of gravel. The conclusion, therefore, I arrived at respecting the origin of bowel complaints in these different institutions was, that they were really caused by malaria, and that their frequency and severity had been affected by the diet only in so far as poorness of the diet had in some cases produced a great susceptibility of the influence of the malaria, and the subsequent increase of the diet diminished this susceptibility.

The second objection to the theory which ascribes the bowel-complaints prevalent in the Penitentiary to the influence of malaria, is that the inhabitants of the immediate neighbourhood do not suffer in a similar way. This is true. Even the private families residing in the Penitentiary are seldom at all affected with the prevalent bowel complaints, and very seldom indeed affected in a severe degree. But surely this is no valid objection. For it must be remembered that these free persons are living under conditions very different from those which surround the prisoners. They are not constantly confined to the atmosphere of the locality; they commonly drink in the course of the day some fermented liquor, as well as other stimulants of the nervous system,

such as coffee and tea; their bodies are kept in a healthy state by active and voluntary exercise, and their minds by cheerful and varied trains of thought. The prisoners have none of these advantages. It might be expected, therefore, that the former class of persons would be insusceptible of disease from causes which might affect the prisoners very generally. There is, indeed, nothing wonderful or remarkable in one class of persons enjoying a comparative immunity from the effects of morbid agents which prove hurtful, and even destructive, to those of another class. During the prevalence of almost every epidemic malady, we see that the poor are cut off, while the rich generally escape. And this has been especially observed of dysentery when it has been most destructive to our troops, not only in military campaigns during war, but also in time of peace, when the barracks have had unhealthy sites. The officers have been far less affected than the private soldiers.*

The consideration of these objections, then, tends by no means to weaken my conviction that the bowel complaints prevalent in the Penitentiary have been caused by malaria. On the contrary, it seems to me to render this theory more probable, since it shows why the effects of malaria on the prisoners have been so well marked, though the neighbourhood of the Penitentiary has not manifestly the features of a malarious locality.

For it has been asked, whence comes the malaria? There is, indeed, no marshy ground near the prison, no considerable extent of stagnant water: there are none of the more obvious sources of miasm. Still, if we examine closely the state of the ground around the Penitentiary, we find in it many points of resemblance to those tracts near the borders or mouths of rivers where dysentery is so often an endemic disease. The tracts of country to which I allude are low, and have a damp alluvial soil, containing much organic matter. Now such, in a less degree, is the character of nearly all the open ground around the Penitentiary. The building itself stands upon an artificial hill, but the surface of the surrounding ground is below the level of high water in the river. The soil is loose, and contains much organic matter, and the subsoil is formed in most parts of an imperfect clay, beneath which are alternate strata of peat-earth, marl, and sand. The ground has hitherto been imperfectly drained. But the chief defect of the site seems to consist in the beds of sand just mentioned being full of water, derived, in all probability, from rain which had fallen on distant and higher grounds. In rainy

* Pringle, Diseases of the Army; Tulloch, Reports of the Health of the Troops; Annesley, Diseases of India.

seasons this water rises through the many breaks in the superficial clayey stratum, and keeps the soil itself, at all events its deeper layers, in a very wet state. These characters show that the exhalation of miasms from the ground around the prison is by no means impossible. And there will, I think, be no difficulty in admitting even the probability that sufficient malaria is produced there to affect persons in whom, from mental as well as physical causes, the power of repelling the attacks or morbid agents is weakened. Indeed, that spot must be a remarkably healthy one where no disease attributable to malaria would show itself amongst persons in the condition of prisoners. And in the Millbank Penitentiary, at the periods when dysentery has chiefly prevailed, the prisoners' susceptibility of disease must have been unusually great, owing to the long terms of confinement then enforced. I believe, therefore, that the dysentery and other bowel complaints which have been prevalent in the Millbank Penitentiary are attributable to its site. But, at the same time, I must remark that the site is not an eminently unhealthy one: this, indeed, is proved by the fact, that the inhabitants of the neighbourhood, and the families residing in the prison itself, have seldom been affected with any disease attributable to an endemic influence; and I may add, that the site would never have been discovered to be other than perfectly healthy had not a prison been built there.

My remarks on the causes of dysentery in the Penitentiary and similar institutions have been extended; for my personal knowledge of the facts seemed to justify my entering into details on the subject. In speaking of the disease as it is seen under other circumstances, I shall be more brief, as my conclusions must here be drawn, for the most part, from the observations of others, and must consequently be less positive, and less entitled to attention. I cannot, however, altogether refrain from noticing the extremely unsatisfactory character of the doctrines generally propounded respecting the causes of dysentery*. It seems to be admitted by most writers on the disease that almost any agent capable of making an injurious impression on the body may produce dysentery. Some of these reputed causes of the disease we may, I think, safely reject. Such are, "acrid ingesta and irritating secretions from the liver, pancreas, and upper part of the alimentary canal," all of which have been supposed capable of producing the disease†. Such causes as

these might occasion irritation of the parts of the canal through which they passed, and consequent purging, but it seems highly improbable that they should produce a severe and rapidly destructive inflammation, confined to the part of the canal they would last reach, and commencing around particular elementary parts of the mucous membrane, namely, the solitary glands. The idea that the autumnal dysentery, diarrhoea, and cholera of this climate, are due to unripe fruit, or other acrid ingesta, if entertained, would be negatived by the fact, that the same complaints prevail during the autumn in prisons, where those causes do not exist, and prevail there to even a greater extent than among the free population. But such an idea is, I believe, no longer entertained by any educated member of our profession in this country. The medical officers of the army in India, however, still reckon the influences I have mentioned among the causes of dysentery; and this seems the more extraordinary when we consider the frequently extreme severity of the inflammation of the large intestine in the dysentery of India, and the acknowledged existence there of a general and more adequate cause for the disease, namely, a noxious state of the atmosphere.

Another class of the causes of dysentery admitted by most writers, comprehends mere high temperature of the atmosphere, or cold and moisture combined, or sudden alternations of heat and cold, and especially the suppression of perspiration by cold dews during the nights succeeding to hot days*. But it is difficult to conceive why these causes should produce dysentery any more than any other internal inflammation, while it is obvious that a high temperature favours the rise of malaria from the soil, and that hot days and cold night-dews are characteristic of those countries and seasons in which malaria is most abundantly generated.

In India, and in all climates where dysentery is a frequent disease, the influence of the season and weather on its degree of prevalence is the same as I have shown it to be at Millbank. Its close alliance, too, with typhus fever, and with cholera, has been generally observed. Even the fatal Asiatic cholera which visited Europe fifteen or sixteen years ago, manifested a close relationship to dysentery. The transition of cholera into dysentery was often seen in particular cases, and the epidemic of cholera in several places passed gradually into epidemic dysentery. Moreover, although cholera was not

* When this lecture was delivered, the author was not acquainted with the excellent article on dysentery, in the "Elements of Medicine," of the late Dr. Robert Williams.

† See Annesley, *Researches on Diseases of India*, vol. ii. p. 234, et seq.; Parkes, *Remarks*

on Dysentery, &c. p. 131, et seq.; Copland, *Dictionary of Practical Medicine*, art. Dysentery, par. 70, 71, and 72; O'Brien on the Dysentery of Ireland, p. 24.

* Twining on *Diseases of India*, p. 2, 3, and 4; Annesley, *op. cit.* p. 244; O'Brien, *op. cit.* p. 24-28.

essentially characterised by disease of the large intestine, yet in many instances it was attended by a lesion of that part of the canal very similar to the lesion characteristic of dysentery. This fact is illustrated by one of Cruveilhier's plates (Livraison 14, Pl. 5.)

From these facts, then, as well as from the consideration of the whole character of the disease, I infer that dysentery is always produced by a poison introduced into the system from without, and that in most instances this poison is generated by the decomposition of matters contained in the soil. Many other of the reputed exciting causes of dysentery, such as intemperance, or exposure to cold, may have a share in the production of the disease; not, however, by acting as the efficient cause, but merely by disturbing the general health, and thus rendering the body obnoxious to the influence of the atmospheric poison which it previously resisted.

The most general source of the poison producing dysentery is certainly the surface of the ground, and the soils generating it in the greatest abundance are those which are rich in organic matter, and are imperfectly drained. But still there are facts which show that dysentery may be produced independently of the source of malaria just mentioned. It has not unfrequently broken out in ships at sea, especially in tropical latitudes. In these instances the poison of dysentery may have been produced as an exhalation from bilge-water, or from decaying vegetable or animal matters in the ship; or the water used for drinking may have become putrid, and the poison having been developed in it, may have been carried into the system through the stomach. For there is no difficulty in admitting that the same noxious matters commonly disengaged from the surface of the ground under the influence of heat and moisture may be produced by the decomposition of organic matter in water, and that the water containing them being taken into the stomach, and thence absorbed into the blood-vessels, may produce the same injurious effects on the body as result from the admission of those matters with the air into the lungs.

I must here also remark that there appear to be other conditions of the atmosphere besides heat and moisture which favour the development of malaria from the soil. The degree in which dysentery prevails in a particular locality is not always obviously proportionate to the circumstances which are known to promote the decomposition of organic matter, and the disengagement of the gaseous products of decomposition. Dysentery is in some years unusually prevalent over considerable tracts of country without any obvious cause. In these cases, however, the disease is especially severe and

general in the spots where it is at other times endemic in a milder form, and where, from the character of the soil and disposition of the surface of the ground, the presence of malaria might be expected. In the same way, Asiatic cholera, when it visited Europe, showed a preference for the banks of rivers and low moist spots, although some general state of the atmosphere was undoubtedly necessary to its production.

Some unknown state of the atmosphere, too, appears to have a share in determining whether the malaria shall produce a mild diarrhoea, a severe dysentery, cholera, or fevers of different kinds. For many of these diseases appear in the same locality at different times, without our being able to determine exactly why at a particular time one disease and not the others should prevail. Sometimes, indeed, two or more of these diseases coexist on the same spot, and it might hence be supposed that these variations in the effects of malaria are due to the greater or less intensity of action of the poison, and to varying states of the system of the persons on whom it acts. But this supposition would not accord with the fact that all forms of endemic disease are not met with in every malarious locality; that ague, for example, is never seen in certain spots where other endemic diseases are rife, and that in some localities where ague is prevalent other endemic maladies are absent. This fact, indeed, seems to show that there are as many distinct varieties of atmospheric poison as there are forms of disease belonging to the class of endemic and epidemic disorders.

I must notice still another question connected with this part of my subject; namely, how the poison, when it has entered the body, produces the diseased changes. Does it itself circulate with the blood, and on reaching the part of the body with which it has some chemical affinity disturb its normal composition, and thus excite diseased action? Or, does the noxious agent act at first on the blood and produce with some of the elements of that fluid a new compound which becomes the immediate cause of the local disease? This question in the present state of our knowledge it is impossible to answer; but it seems to me equally impossible to doubt that in dysentery as well as in fever the intestinal lesion is the effect of a chemical action taking place between the glands, or the tissue immediately surrounding them, and a morbid matter circulating with the blood. The time, perhaps, will come when chemistry shall be able to detect the subtle agents which produce these and other disorders in our bodies, and shall explain the play of affinities which, disturbing the normal constitution of particular parts, causes destructive inflammation to arise in them.

The poison of dysentery and that of typhoid differ much in their action on the human body. The poison of fever has an especial affinity for the glands of the small intestines, and also produces almost immediately an evident change in the constitution of the blood. The poison of dysentery attacks the glands of the large intestines, and in the more common sthenic form of the disease produces no obvious change in the condition of the circulating fluid. There is, however, as we have before seen, an asthenic variety of dysentery in which the blood does apparently undergo a change in its composition and vital properties; and here we must suppose that the poison of dysentery is modified in its properties, or that it is combined with some other noxious matter capable of disturbing the normal constitution of the blood.

It remains to inquire what cause or causes produced those *nervous disorders* which at different periods have appeared amongst the prisoners in the Millbank Penitentiary. How has it happened that disorders of this kind have shown themselves prominently in connection with dysentery only in that establishment?

The first important fact to be noticed in relation to this inquiry is, that these nervous disorders have not been constantly prevalent in the institution. They have appeared only at those times when dysentery was epidemic in the prison, or was about to become so. This fact suggests the inference that some alliance exists between the cause of the dysentery and the influence giving rise to these nervous disorders: an inference which is strengthened by another important fact, viz. that tetanus and neuralgia have been observed as endemic diseases in malarious countries.

With respect to tetanus, no doubt exists but that it is far more common among adults in hot climates than in temperate ones, and in hot seasons than in those that are cool; while the tetanus or trismus of infants is seen chiefly in pestilential countries, such as the West Indies, and in temperate climates only under circumstances which favour the belief that it is produced by a noxious state of the atmosphere. Now it has been observed that the adults attacked by idiopathic tetanus in hot climates are for the most part persons who have suffered hardships, or have been more than usually exposed to the deleterious influence of the climate. The infants we may suppose to have been predisposed to suffer from the disease by the delicacy of their whole constitution, and especially by the excitability of their nervous system.

Will not these facts help us to explain the occurrence of peculiar nervous disorders in the prisoners at Millbank? Besides

confirming the opinion that the efficient cause of these disorders is some kind of malaria, do not these facts also render it probable that a peculiar state of the system of the prisoners has predisposed them to be thus peculiarly affected by a cause which would have produced no such symptoms in other persons?

We have already seen that the effects of imprisonment on the nutritive system are such that severe inflammation and change of structure is produced in the prisoners by a morbid influence which does not affect free persons who are equally or almost equally exposed to it. May not imprisonment so affect the nervous system, likewise, as to give it an extreme excitability comparable to that which predisposes infants to suffer from trismus under the influence of malaria?

If we consider for a moment the effect which long-continued exclusion of light has on the eye, the great sensibility of that organ which results, so that it cannot bear ordinary daylight, suddenly restored, without pain, or sunlight without danger, we shall, I think, find it reasonable to expect that the whole nervous system of prisoners who have been very long confined in complete or almost complete seclusion from society and from all the ordinary sources of mental excitement will manifest an exaggerated sensibility to the influence of unusual stimulants. At all events, facts have occurred during the last few years which prove the existence of this sensitive state of the nervous system in prisoners under such circumstances.

Prisoners sentenced to transportation after being confined for a longer or shorter time in Government prisons in a state of the greatest order and silence, deprived of the society of their fellows and of all the causes of excitement to which they had been accustomed, have been suddenly transferred to convict ships in the river, where they have been thrown together without discipline or restraint of any kind, and exposed to the additional excitement of the parting with friends, and to the tumult which must exist in ships preparing for sea. The effect of this sudden change has been that many of the prisoners have been thrown into fits of epileptic convulsions: not merely men and women previously subject to epilepsy, but those who had never before suffered from the disease, have been so affected. This has occurred, not once only, but several times, many prisoners being attacked with epilepsy in each ship.

It is only during the first few days after embarkation that these symptoms of disordered nervous system have been observed; and no serious consequences have been left. But although so temporary, these phenomena are important from their proving that

imprisonment in a state of seclusion induces, for a time at least, an enfeebled, and in consequence highly excitable state of the nervous system.

Now both in the year 1823 and in the year 1842, when the nervous disorders associated with dysentery occurred in the Penitentiary, the terms of confinement to which the prisoners were subjected were much longer than at present. We may therefore, I think, fairly infer that the excitability of the nervous system of the prisoners was at those times proportionably great.

Can we not now explain the occurrence of the nervous disorders in the Penitentiary? We have seen that they were associated there with other disorders due to the influence of malaria; and that in hot and unhealthy climates analogous nervous affections are met with, in adults of enfeebled frame, and in infants whose nervous system is naturally sensitive. We have seen too that the nervous system in prisoners is brought by confinement to a similar sensitive state; and is it not the natural conclusion from these facts that the peculiar nervous disorders seen in the Penitentiary were due to the action of an atmospheric poison, on persons in whom the nervous system was rendered thus excitable?

My account of the dysentery observed at the Millbank Penitentiary would be incomplete if I omitted to notice the remedial measures which have been found most efficacious in the *treatment of the disease*. I have here no novel mode of practice to announce. But still, the plans recommended by different authors for the treatment of dysentery being so various, it may be of some interest to the College to learn which of these plans has best answered the wishes of the physician in an instance where the disease has presented well-marked characters, and has prevailed, not in one season merely, but through several years.

I shall first speak of those more numerous cases in which the disease had, for the most part, the characters of a simple inflammation of the larger bowel, this inflammation varying in severity, but being attended by no morbid state of the system at large except symptomatic fever.

The chief means I have found successful in these cases, and the means which have rarely failed, where the dysentery was not complicated with some other diseased state, are bloodletting, the administration of calomel with opium, and gentle aperients.

General bloodletting has seldom been required, but free local bloodletting by means of leeches has never been omitted when there was well-marked tenderness of the abdomen, or, in the absence of tenderness,

when the other symptoms indicated the existence of active inflammation in some part or other of the large intestine. The application of leeches, in the number of twelve or twenty-four, has been repeated several successive times at intervals of some hours, when the symptoms were urgent, and were not readily subdued by the means first used.

The calomel and opium have generally been administered in doses of two grains of the former, and one-third or half a grain of the latter medicine, every three, four, or six hours, as long as the character of the evacuations and the state of the local and general symptoms indicated the existence of inflammation, or until the state of the gums showed that the system was affected by the mercury. In the more severe cases the production of this effect on the system to the full extent was required; but when the disease was of only moderate severity it was generally subdued so quickly, that the mercurialization of the system to a further extent than was manifested by a slight swelling and tenderness of the gums was unnecessary.

Mild aperients also have been found most useful in perhaps the majority of the cases, but especially in those where the lower part of the large intestines was the seat of the disease, and where the *fæces* were retained, so that the evacuations consisted almost entirely of mucus tinged or mixed with blood. In these cases the administration of one, two, or three drachms of castor oil has produced free feculent evacuations, and has afforded great relief to the symptoms. The beneficial effect produced by the action of this mild aperient medicine has often been very remarkable, greater than I know how to explain; while evil seemed always to result from allowing more than twelve hours to pass without obtaining a free feculent evacuation, if the frequent discharge of bloody mucus and other signs still indicated the persistence of inflammation. In many cases, however, no aperients whatever were required, free discharges of the feculent contents of the bowels taking place spontaneously. Indeed, where the cæcum and ascending colon were the seat of the disease, the active stage of the inflammation was almost always attended with copious discharges of liquid *fæces*, and the subsidence of the inflammation was in great measure evidenced by the less frequent occurrence of these discharges, and their less liquid character.

Very rarely indeed was a more active purgative than castor oil found requisite in the acute stage of dysentery. In some instances, owing to the irritable state of the stomach, small doses of saline purgatives have been substituted for the castor oil; but

whenever they have acted with much violence they have seemed to do harm. I have, indeed, seen nothing to encourage me to a trial of the plan of treating dysentery by active purgatives, recommended by some writers who have practised in India.

At one time I thought the use of even local bloodletting might in a great measure be dispensed with, and that the inflammation in the large intestine might be wholly subdued by means of calomel, administered in doses of five, ten, or fifteen grains, combined with more or less of opium according as there was a disposition to the retention of the fæces, or to the discharge of frequent, liquid, feculent evacuations. And assuredly the disease was often very speedily arrested by this plan of treatment; the system being quickly brought under the mercurial influence, and the general as well as the local symptoms of inflammation disappearing as soon as the dark-green discharges produced by the calomel took the place of the bloody and mucous, or of the very liquid, though feculent, evacuations. I have subsequently, however, seen reason to prefer the more moderate administration of calomel, aided by free local depletion, as a safer and indeed more sure method of reducing the inflammatory action. In the most severe cases of acute inflammatory dysentery occurring in the Millbank prison, one or two large doses of calomel are still given at the commencement; but afterwards the administration of this remedy is continued in smaller doses. I have relinquished the use of large doses of calomel as the general mode of treatment, not only because it was uncertain in its effects on the disease, and because the ptyalism produced was sometimes very troublesome, but also because the violent mercurial action, in some cases, seemed to cause serious injury to the system, and to lead to the development of tubercular disease. I should here remark, too, that calomel, even in small doses and combined with opium, has in some cases increased the irritation of the large intestine; and that great advantage has then resulted from substituting for the calomel the grey mercury with chalk, of the *Pharmacopœia*, in combination with Dover's powder.

When the symptomatic feverishness was considerable, the skin was generally dry as well as hot. And then some benefit has been derived from the use of antimony, ipecacuanha, and other diaphoretic remedies. But ipecacuanha has wholly failed in my hands as a specific, or in any way active, remedy for the disease.

The local bloodletting, the calomel and opium, and the mild aperients, were the remedies to which the physician looked for the cure of the disease. But there were other remedial means from which the pa-

tients in many cases derived more sensible relief from their sufferings. To those means, however, of which the principal were opiate enemata to relieve the tenesmus, large warm poultices to the whole abdomen, and medicines to allay symptomatic vomiting, I can merely thus briefly allude. I must, however, remark that whenever, from the age or debility of the patient, it appeared desirable to give moderate quantities of a stimulant, such as brandy, no harm ever resulted from its use. On the contrary, a beneficial effect was produced, however active the local inflammation might be. It may be thought that this was owing to the prisoners having been weak from long continued confinement. But there was no strongly marked sign of such weakness, and, when general bloodletting appeared to be called for, it was borne well. Indeed, the principal reason for preferring local bloodletting, even in severe cases, was that it seemed to be more efficacious, and not that any symptoms contraindicated general depletion.

I need scarcely say, that the diet allowed in the earlier stage of dysentery consisted merely of milk and farinaceous articles of food.

Such is the plan of treatment which has been found best adapted to cure the simple acute dysentery quickly, and without injury to the constitution of the patient. It differs from the plan which was found so successful by Dr. Latham and Dr. Roget in the year 1823, chiefly in greater use being made of local blood-letting, and in the main reliance not being placed on the large doses of calomel and opium. This difference in the treatment is, indeed, the necessary consequence of the disease having presented a well-marked inflammatory character during recent years, while in the epidemic of 1823 this character was for the most part wanting.

There have of course been many slighter cases, in which the more active remedial measures were not needed—where no distinct tenderness of any part of the abdomen indicated the necessity of applying leeches, and where the mildness of the symptoms rendered it unnecessary to use the mercurial remedy to the extent of affecting the system in the slightest degree. These cases were generally characterized by moderate tormina, rather frequent evacuations containing some bloody mucus, temporary tenesmus after each action of the bowels, loss of appetite, and thirst; and these symptoms were soon relieved by two or three doses of calomel with opium, and the more continued administration of an oleaginous mixture, which was composed of mucilage, with a small quantity of castor oil intimately diffused through it, and to each dose of which a few minims of laudanum

were generally added. This castor oil mixture I found in frequent use when I first visited the prison, and I have since had constant opportunities of observing the good effects it produces. Its virtue seems to depend on the mild oleaginous purgative being so subdivided that it acts as a moderated stimulus on a large extent of mucous membrane, instead of exerting a more powerful irritating influence on particular parts.

These very mild cases of dysentery are frequent in London during almost every autumn. If not carefully distinguished from the common diarrhoea, and if treated with the aperients and aromatics which so soon arrest the latter disorder, they are, according to my observation, very apt to assume the chronic form, and to be productive of long-continued annoyance and suffering to the patient.

Both in this milder form of dysentery and in the stage of subsidence of the more active form of the disease, strict attention to diet has appeared to me to be of paramount importance. No other influence has so frequently seemed to re-excite the inflammation in the intestine, or to cause the disease to assume a chronic form, as the too early indulgence of the appetite with meats, broths, or succulent vegetables. I have always found it better to withhold these articles of diet, if possible, till the mucous membrane has regained its healthy condition.

At a very early period of the decline of the disease, cretaceous and aromatic medicines, generally combined with more or less of opium, were given with advantage; and, if the disease became chronic, various tonics and astringents were combined with the aromatics and opium. The astringents most frequently found useful were the tincture and infusion of catechu and the sulphate of iron; but other medicines of the same kind, as the sulphates of zinc and copper, acetate of lead, and cinchona bark, were occasionally employed; one astringent often proving efficacious when others failed, and each, in turn, gradually losing its power after its use had been continued for a more or less considerable period. But, although astringent medicines are most valuable means in the treatment of chronic dysentery, their action, according to my observation, should never be carried so far as to produce actual constipation of the bowels, for this has almost always been followed by an aggravation of the disease. Opiate remedies, too, though generally highly serviceable, and even essential, have sometimes arrested the secretion or excretion of the bile, and have thus produced an injurious effect on the local disease as well as on the general health of the patient. In such cases it has been found

necessary either to omit altogether the use of opium, or, diminishing its dose, to combine with it some dandelion or rhubarb. Other details of the treatment of chronic dysentery I cannot enter upon here; but I must remark that strict attention to diet has appeared to me of as great importance in the chronic as in the acute stage of the disease. Animal food and vegetables have been allowed in very moderate proportion, and have been altogether withheld when increased frequency and liquidity of the stools indicated the accession of fresh irritation in the large intestine.

Astringent and aromatic remedies being judiciously employed, and great precautions as to diet being taken, few cases have failed of being cured, except where, as not unfrequently happened, the disease was complicated with the tubercular cachexia. Even when large portions of the coats of the intestine had been discharged from the bowels as sloughs, perfect recovery has taken place in so short a space of time as to prove the great and rapid power of healing possessed by the mucous membrane.

The intractable character of chronic dysentery, as it is seen in persons who have contracted the disease in hot climates, seems to be owing not simply to the extent of the intestinal lesion, but in a great measure to the impaired condition of other abdominal viscera, such as the liver and spleen.

I should not omit to mention that, at various periods in the course of inflammatory dysentery, other enemata than the simple opiate and demulcent ones already alluded to have been found of great service. This has happened when the inflammation has been very severe in the lower part of the large intestine, when the pain and tenesmus has been great, and the discharge of the bloody fluid and mucus very frequent, and when simple opiate enemata have failed to alleviate these symptoms. In these cases the injection of a few ounces of black wash, with a drachm of laudanum, has often given great relief, and has appeared to check the inflammatory process.

Other stimulant injections, as solution of nitrate of silver, have appeared useful under similar circumstances, but on the whole the black wash with laudanum has been found the preferable remedy. At a later stage, when the inner coat of the bowel, having mortified, was becoming detached, similar stimulating injections have appeared to aid the process of separation, and the healing of the solutions of continuity. At all events, the discharges from the bowels, during their use, have become more healthy. Advantage has been derived also from these remedies, especially from injections of a weak solution of sulphate of zinc, when, in the still more chronic stage of the disease, a copious secre-

tion of purulent fluid has taken place from the ulcerated surface of the lower bowel. The discharge has rapidly diminished in quantity, and there has been every possible evidence that the healing of the ulcers was promoted.

The remarks I have hitherto made on the treatment of dysentery apply to the very large majority of the cases,—to those in which the disease has seemed to be a purely local inflammation, and in which the feverishness has not been more than might be symptomatic of the local lesion.

The treatment of the comparatively infrequent but much more formidable cases in which the mucous and submucous coats, in extensive tracts, rapidly pass into the state of sphacelus, I have found to be a matter of much greater difficulty; the altered state of the blood apparently rendering the use of active remedial means inadmissible.

According to my observation, calomel in large doses is not well borne in these cases, and blood-letting, to an extent proportioned to the severity of the local lesion, would be fatal to the patient. Even local blood-letting, when not very cautiously used, has appeared to increase the debility without producing much relief to the local symptoms.

Still I am satisfied that cautious local depletion and small doses of a mild form of mercury are means which ought to be used in the treatment of this formidable disease. Diaphoretics, too, especially ipecacuanha and the warm bath, have appeared to produce a most beneficial effect. But all these remedies generally fail of saving the patient, and they would probably always fail unless they were combined with measures adapted to support the strength of the system. Wine and beef-tea, which in the simple inflam-

matory form of the disease would be hurtful, must be administered freely. In fact, we are obliged to treat the disease with reference rather to the state of the system generally, than to the condition of the inflamed intestine.

With all our caution and all our endeavours, however, these cases will generally terminate fatally; if not in the acute stage, yet in the chronic stage, when, the dead mucous membrane having been thrown off, a constant oozing of the serous part of the blood from the extensive surface of exposed tissues gradually exhausts the patient's strength.

The intractable nature of this disease when fully developed, and the rapidity of its course, make us indeed wish with Sydenham that more specific remedies were discovered; that by their means, not only the cure might be more immediate, but that, in such diseases as the one just now under consideration, the poison admitted into the system might be destroyed, before it had produced in particular organs those deadly effects which Sydenham and the older writers supposed nature to bring about unwillingly, in her efforts for the expulsion of the morbid matter.

The prospect of such a remedy being discovered, even for dysentery, is not altogether desperate, for we possess a specific remedy for a disease which has a cognate cause, and which is frequently combined with dysentery, namely, ague. The knowledge of the specific virtues of Cinchona bark we do not, it is true, owe to medical science, but we must remember that not many years have elapsed since the discovery of a new specific for another disease, syphilitic perioritis, rewarded the zeal and perseverance of a late respected fellow of this College.

