LECTURES ON THE PRACTICE OF MEDICINE.

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LECTURE III.—INFLAMMATION.

1. *Definition.*—2. *Etymology.*—3. *Frequency and Importance.*—4. *Pathology. Minute Changes. Results: a. Resolution; b. Suppuration; c. Fibroid Substitution; d. Ulceration; e. Gangrene. Inflammation of Serous and Mucous Membranes.*—5. *Causes.*—6. *Symptoms. A. Local Signs of Inflammation: a. Redness; b. Swelling; c. Heat; d. Pain. B. Constitutional Pyrexia. C. Deranged Functions of Inflamed Part.*—7. *Varieties.*—8. *Treatment.*

1. *Definition.*—Inflammation of a tissue consists in the increased transudation into it of materials from the blood, and an alteration of its nutrition, consequent on some irritation.

Many definitions of inflammation have been given by different writers. It was long taught that inflammation consisted essentially in an exudation of lymph, which might either become organised, or soften into pus. This definition, however, is not applicable to all forms of inflammation; for, although in some the matter transuded from the minute vessels does take the form of lymph, yet lymph is rarely seen in inflammation of mucous membranes, of brain, and of cartilage. Nor can inflammation be defined as a local hyperæmia with stasis of blood in the capillaries; for, although these phenomena are common in inflammation, it has been shown that, even in vascular tissues, exudation of liquor sanguinis, although favoured by increased arterial afflux, may occur without it; and, moreover, we know that inflammation may take place in tissues which are non-vascular, the increased supplies being then drawn from the vessels of the adjacent parts. The definition which I have now placed before you is most in harmony with our present knowledge of the subject.

II. *Etymology.*—The term "inflammation" is derived from the Latin word *inflammo*, to burn; and was originally employed many centuries ago, from the fact that, in external inflammations, increased heat or burning is one of the most obvious and prominent phenomena.

III. *Frequency and Importance.*—Next to pyrexia, inflammation is the most common morbid process which comes under the notice of the medical practitioner. Most diseases, in fact, either commence as inflammation, or are complicated with inflammation in some part of their course; while others simulate inflammation so closely as to require considerable skill to discriminate them. In after-life, you will constantly be called upon to decide whether inflammation exists or not. Death is, in a great many instances, due to inflammation; and in not a few instances death is prevented by inflammation. A local inflammation glueing the peritoneal covering to adjacent parts often prevents a fatal escape into the peritoneal cavity of food from an ulcerated stomach, of feces from an ulcerated bowel, of pus from an hepatic abscess, or of gall-stones from an ulcerated gall-bladder. You will perceive, then, how important it is that you should acquire an accurate knowledge of the inflammatory process. This, I have no doubt, you have already done from attendance on the lectures on General Pathology. In this lecture I shall do no more than recall to your minds the leading facts of the process, and more especially those which have a practical bearing upon the diseases which we shall have hereafter occasion to discuss; and for additional details I would refer you to the able articles of Dr. Burdon Sanderson and Mr. Simon in the second and fifth volumes of the second edition of Holmes's *System of Surgery*.*

IV. *Pathology.*—The minute changes which take place in a tissue as the result of inflammation have long been studied; and our knowledge of them has been rendered much more accurate by the researches in recent years of Lister, Cohnheim, Stricker, and Sanderson. The plan of investigation has consisted in producing inflammation artificially in the transparent web of a frog's foot, or in the mesentery or tongue of the same animal, or in the wing of the bat. The changes observed have been as follows:

1. Disorder of the circulation;

2. Transudation through the minute vessels of liquor sanguinis and blood-corpuscles;

3. Alterations in the nutrition of the tissue.

1. *Disorder of the Circulation.*—The first change consists in an acceleration and increase of the circulation of the irritated part. The vessels immediately begin to dilate without any antecedent stage of contraction; the dilatation being progressive, and commencing in the minute arteries, but followed by a similar change in the veins. The rate of movement of the blood is at the same time altered. At first it is much quicker than natural, so that both the actual quantity of blood existing in the part, and that passing through it in a given time, are increased. There are satisfactory grounds for believing that the acceleration of the blood-stream is due to an irritation of the centripetal nerves of the injured part, reflected by the vaso-motor centre through the centrifugal nerves to the vessels, and influencing them in some way (not yet sufficiently understood), so as to facilitate the flow of blood through them.

The second change observed is a retardation and diminution in the flow of blood through the part. The retardation usually commences rather suddenly, and is first observable in the veins; but the rapidity of the current may be often seen to vary in contiguous vessels; the current in some being rapid, and in others oscillating to and fro; while in others the blood is stagnant. The small arteries and capillaries often exhibit at the same time irregular bulgings and contractions. At last the current becomes everywhere arrested; and the red corpuscles accumulate in the capillaries, and become so closely packed that their outlines can barely be distinguished. This is the condition long known as *inflammatory stasis*, and is believed to be due to some impairment or alteration in the vital properties of the walls of the blood-vessels with which the circulating blood comes in contact. Around the area of stagnation there is *congestion*, or fulness of the vessels with slow movement of the blood; and at a further distance around there is, to adopt Sir James Paget's expression, *determination*, or fulness with rapid movement of the blood.

2. *Transudation through the Minute Vessels of Liquor Sanguinis and Blood-corpuscles.*—In a state of health, transudation to a certain extent is constantly taking place through the coats of the minute vessels into a tissue; but in inflammation, as soon as retardation of the current of blood commences, this transudation is increased. The transuded matter varies in different cases. Sometimes it is mainly serum, as in certain inflammations of the pleura and in the submucous infiltration of an inflamed mucous membrane; at other times it is coagulable lymph, as in inflammations of the pericardium, peritoneum, and pleura. The exudations of inflammation differ from the liquor sanguinis of normal blood, in containing more albumen and a great excess of chlorides and phosphates. Pus is said to contain three times as much chloride of sodium as the serum of blood; while in pneumonia the lungs are strongly impregnated with chlorides, which disappear from the urine. But it is not the liquor sanguinis alone which transudes. The blood-corpuscles, and especially the white corpuscles, pass through the coats of the vessels, and escape into the inflamed tissue. Many years ago (1841), it was shown by Dr. C. J. B. Williams that, when the capillary circulation was becoming retarded, the white corpuscles in the vessels became increased, and showed a remarkable disposition to adhere to their walls; and that white corpuscles also appeared outside the vessels, undistinguishable from those within. But he did not see his way to admit that the corpuscles outside and inside the vessels were identical. Dr. William Addison (1842) and Dr. Augustus Waller (1846) were the first to describe the emigration of white blood-corpuscles through the walls of the blood-vessels; and both of these observers maintained that the escaped blood-corpuscles became pus-corpuscles. About the same time, it was shown by Virchow that pus-corpuscles and white blood-corpuscles are indistinguishable. The statements of these observers, however, made little impression, and were soon forgotten; and it is only since the independent researches of Professor Cohnheim of Berlin in 1867 that the transudation of blood-corpuscles has come to occupy an important place in the pathology of inflammation. For the details of the method of transudation, I must refer you to Dr. Sanderson's memoir;* and I shall now confine myself

* An excellent summary of the minute changes which take place in inflamed tissues will also be found in Dr. T. H. Green's *Introduction to Pathology and Morbid Anatomy*. London: 1871.

* "Simultaneously with the retardation, the leucocytes (white corpuscles), instead of loitering here and there at the edge of the axial current, begin to crowd in numbers against the vascular wall, as was long ago described by Dr. Williams. In this way the vein becomes lined with a continuous pavement of these bodies, which remain almost motionless, notwithstanding that the axial current sweeps by them as continuously as before, though with abated velocity. Now is the moment at which the eye must be fixed on the outer contour of the vessel, from which (to quote Professor Cohnheim's words) here and there minute colourless button-shaped elevations spring, just as if they were produced by budding out of the wall of the vessel itself. The buds increase gradually and slowly in size, until each assumes the form of a hemispherical projection, of width corresponding to that of a leucocyte. Eventually,

to stating that it has been satisfactorily demonstrated that, as soon as retardation of the circulation commences, and before complete stasis, the white corpuscles crowd in large numbers against the vascular wall, and pass through the wall, not by little rents or fissures, but by a process of amoeboid digestion of the protoplasm of the wall. The red corpuscles also sometimes pass through the walls of the capillaries, thus accounting for the occasional presence of blood in inflammatory exudations.

3. *Alterations in the Nutrition of the Tissue.*—The nutritive changes, although they differ according to the structure of the part affected, all indicate an abnormal activity of cell-life. The first change is one of cell-proliferation. This is particularly active in tissues, which, like skin and mucous membrane, are naturally maintained by a multiplication of their cellular elements; but, even in such a tissue as cartilage, the cells, which under normal conditions undergo no alterations in form, become enlarged and altered in shape, while new cells are formed in their interior by enlargement and subdivision of their nuclei and protoplasm; and the matrix softens and liquefies (Fig. 1). By a continuance of this

Fig. 1.—Section of Cartilage artificially inflamed, magnified 250 diameters. *a*, Healthy cells and matrix. *b*, The cells becoming spherical. *c*, *d*, The same, undergoing endogenous proliferation. *e*, New cellular structure resulting from the inflammatory process, pervaded by vessels, *f*, (Cornil and Ranvier.)

process, the natural structure of the connective and supporting tissues, such as areolar tissue, bone, cartilage, and muscle, is completely destroyed, while the newly formed elements either degenerate into pus or die, or become developed into a permanent tissue of inferior organisation to that of the original structure.

Such is a brief outline of the minute changes which take place in inflammation. We may now consider from a practical point of view some of the results of the inflammatory process.

1. In the first place, inflammation may terminate in what is called *resolution*. Here there is congestion and retardation of the flow of blood, but the quantity of material transuded through the coats of the vessels is small, or is absorbed without affecting any material alteration in the nutrition of the tissue. This is the most favourable mode of termination of inflammation. When the process of resolution is very sudden and rapid, it is sometimes called *delitescence*; and when the inflammation which thus suddenly recedes from one part of the body makes its appearance in another, as in articular rheumatism and in mumps (where the inflammation is sometimes transferred from the parotid to the mamma or testicle) there is said to be *metastasis*. This diversion of the inflammation from one part of the body to another we sometimes endeavour to imitate in treatment by what is termed *counter-irritation*, *derivation*, or *revulsion*.

2. *Suppuration* is a very common result of inflammation and almost invariably occurs when the process is intense and acute. It is unnecessary for me to describe to you the minute characters of pus; but I may remind you that it consists of minute rounded corpuscles suspended in a liquid similar to liquor sanguinis; that these corpuscles cannot be distinguished from the white corpuscles of the blood; that they can multiply by division; that they are masses of contractile protoplasm, which, like amoebae, can alter in form, and have the power of spontaneous movement; and that they can, therefore, migrate in the tissues. The liquid of the pus is the transuded liquor sanguinis, but the corpuscles are derived from two sources. Some, and especially those met with in the early stages of inflammation, are the white corpuscles which have escaped from the vessels—blood-corpuscles, in fact, in the wrong place; but others are due to the proliferation of the cellular elements of the inflamed tissue, whose nutrition has become altered in the manner already described. By a process of cell-growth, then, the natural tissue of a

solid organ may become *liquefied* into pus. When skin or mucous membrane is inflamed, the pus, as soon as formed, is discharged; but in inflammation of other parts the pus collects in a cavity, and the collection is called an *abscess*, or it becomes infiltrated through the tissues of the inflamed part in what is called *purulent infiltration*. An abscess usually finds its way in time to the skin or a mucous surface, and discharges itself; but if it remain long imbedded in the tissues, the liquid becomes absorbed, the cells atrophy and undergo a fatty disintegration, and in time the abscess may dry up into a cheesy or calcified mass.

3. *Fibroid Substitution* is another result of the inflammatory process, and especially when this follows a chronic course. Here the inflamed part, instead of liquefying, becomes solidified from the gradual formation of new fibrous tissue, which takes the place of the natural structure. The young cells, which have escaped from the vessels, or formed in the part, in the manner already described, become aggregated, and present a distinct nucleus in their interior (granulation-tissue); channels become hollowed out in this new tissue lined on each side by cells, into which blood penetrates from the neighbouring vessels, and in this way new vessels are gradually formed, while at the same time the granulation-tissue becomes slowly developed into a fibrillated tissue. Some of the young cells become elongated and spindle-shaped; others disappear; and the intercellular substance splits up into delicate fibrils, in which at certain intervals granulation-cells remain permanently imbedded, and constitute fixed connective tissue corpuscles. This is the process which takes place in the so-called cirrhosis, or interstitial inflammations, of the liver, lungs, and kidneys, and also during the healing of wounds by the "first intention," and in the organisation of the granulations by which the cavity of an abscess is filled up after the discharge of the pus. It is by the same process also that the lymph which is thrown out in an inflamed serous membrane becomes organised, so as ultimately to form fibroid thickening of the membrane and firm adhesions between its opposed surfaces, in what is called *adhesive inflammation*. An important feature of this process is that the cellular mass, during its conversion into fibrous tissue, undergoes contraction; and thus it is that the site of a cut on the skin is for ever marked by a puckering called a cicatrix, while similar cicatrix-like depressions may be seen on the surface of an internal organ which has been the seat of interstitial inflammation.

4. *Ulceration* is the term applied when the liquefying result of inflammation described under the head of Suppuration involves any of the surface-textures of the body, such as the skin, mucous membrane, articular cartilage, or the lining membrane of an artery, to such an extent as to produce a loss of substance. The breach of surface is called an *ulcer*. Like a closed abscess, an ulcer may burrow deeply, and eat through all the tissues with which it comes in contact. An ulcer heals or *cicatrises* by granulation-tissue becoming developed into fibrous tissue, as already explained.

5. *Gangrene, Mortification, Sphacelus, or Sloughing*, is another result of inflammation. The vitality of the inflamed part may be so impaired, and the stagnation of blood so complete, that the part dies. Mortification is, therefore, most apt to occur when the inflammatory process is intense, and when there are in operation other causes of impaired vitality and weakened circulation, such as a weak state of the circulation throughout the body, an unhealthy condition of blood, or pressure upon the inflamed part. As soon as vitality is entirely destroyed, all pain in the part ceases, and the ordinary chemical changes of putrefaction take place in the dead mass, which, acting like a foreign body, excites fresh inflammation in the surrounding tissues. By suppuration and ulceration, a *line of demarcation* is ultimately formed between the dead and living tissue, and ultimately the former is completely detached.

Mortification is not an equally common result of inflammation in all parts of the body. It is most common in the vascular connective and supporting tissues—skin, areolar tissue, and bone. The next most common site is the mucous membrane of the digestive canal. Thus sloughing is not a very rare result of scarlatinal ulceration of the fauces, and is a very common result of the inflammation of the intestines which occurs in enteric fever and dysentery. It is a very rare result of inflammation of the lungs, and is scarcely ever met with in the liver, spleen, kidneys, heart, or brain.

Gangrene, however, may occur independently of inflammation. Among other causes, may be mentioned occlusion of the afferent artery by embolism or other processes; diminished power of the heart in conjunction with diseased arterial coats, as in "senile gangrene"; occlusion of the small arteries and capillaries by pressure, or by the presence of certain poisons, such as ergot, in the blood; extreme heat or cold; and powerful corrosive substances. When gangrene is due to a deficient supply of blood, the dead part shrivels up, and the gangrene is said to be *dry*; but when it is due to inflammation, or when

the hemisphere is converted into a pear-shaped body, the stalk end of which is still attached to the surface of the vein, while the round part projects freely. Gradually the little mass of protoplasm removes itself further and further away; and, as it does so, begins to shoot out delicate prongs of transparent protoplasm from its surface, in nowise differing in their aspect from the slender thread by which it is still moored to the vessel. Finally, the thread is severed, and the process is complete. The observer has before him an emigrant leucocyte." (Sanderson, *loc. cit.*)

there is at the same time venous obstruction, the part may remain tense from accumulation of fluid, and the gangrene is termed *moist*.

There are certain modifications of the inflammatory process, according to the tissue involved, which are of some practical importance, and which I must briefly mention. I refer more particularly to the varieties of inflammation observed in serous and mucous membranes.

1. In *Serous Membranes*, the inflammatory process commences with hyperæmia. Liquor sanguinis and white corpuscles transude from the vessels into the serous cavity; and the epithelial cells of the serous membrane enlarge and become granular, and multiply by endogenous growth (Figs. 2 and 3). Instead of being smooth and glistening, the

Fig. 2.—A fragment of healthy Mesentery treated with nitrate of silver, and showing its cellular structure. Magnified 250 diameters. (Cornil and Ranvier.)

membrane becomes opaque and rough, from the deposit of coagulated lymph glueing the opposed surfaces together, or forming a slightly adherent rough or reticulated membranous covering. Along with this, there is usually more or less serous fluid, which is turbid from the

Fig. 3.—A fragment of Mesentery artificially inflamed and treated with nitrate of silver. a. Epithelial cell proliferating. b. Pus-globule. g, g. Epithelial cells which have become globular and prominent, and in process of detaching themselves from the fibrous bands, i, f. Epithelial cell swollen, but still adherent. (Cornil and Ranvier.)

presence of migrated blood-corpuscles and flakes of lymph. Sometimes none of this liquid is thrown out; and, when present, the quantity varies greatly. Occasionally the quantity is so great that the serous sac is greatly distended, and there is danger from its pressure on adjacent parts. When recovery takes place, this liquid is absorbed; but the fibrine undergoes organisation in the manner which I have already described, and produces permanent thickening of the serous membrane, and firm bands of adhesion between its opposed surfaces. It is rare for the exuded matters to take the form of pus; but sometimes this does happen, and more especially when the patient's system has been in a very low condition—when air, or some putrid matter, such as the contents of the bowel, has obtained access into the serous cavity; or when the inflammation has been excited by some morbid poison, such as that of small-pox, in the blood.

2. In *Mucous Membranes*, we find two distinct forms of inflammation—the catarrhal and the membranous. Of these, the former is by far the more common.

a. Of *catarrhal* inflammation, the characters are mainly three; viz., increased vascularity; tumefaction of the membrane from submucous transudation; and an increased secretion of mucus from the free surface, at first clear and tenacious, but gradually becoming opaque yellow, less viscid, and puriform. There is nothing here like an exudation of fibrine from the blood. The exudation takes the form of an increased formation of mucus, although of course the materials for this are transuded from the blood. On minute examination, it is found that from the first the epithelium-cells become enlarged and granular, and abnormally multiplied; and that the number of mucus-corpuscles is greatly increased, some of these corpuscles being apparently escaped

blood-corpuscles, but the majority being formed by endogenous multiplication within the epithelial cells (Fig. 4). As the inflammation advances, these little rounded corpuscles become still more numerous, and indistinguishable from those of pus. The only differences, in fact, between a mucus-corpuscle and a pus-corpuscle are, that the latter is smaller, and has several nuclei instead of one. At the same time, the

Fig. 4.—Catarrhal Inflammation of the Conjunctiva. a. Epithelial cells undergoing endogenous proliferation. b. Subepithelial connective tissue. (Kindsfleisch.)

follicular glands in the mucous membrane become distended with secretion, and may impart a “granular” appearance to the inflamed mucous membrane. Little drops of pus may form in the inflamed follicles, and, at last bursting, may leave small ulcers. The process of proliferation commencing in the epithelial cells may also extend to the subepithelial connective tissue, and thus produce more extensive breaches of surface or ulcers. It is important to remember that the redness and swelling indicative of catarrhal inflammation during life may be no longer visible in the dead body.

b. In *membranous, croupal, or diphtheritic* inflammation, there are also hyperæmia and tumefaction of the mucous membrane; but its peculiarity consists in this, that the free mucous surface becomes lined with a distinct membranous layer of more or less thickness, but often resembling wash-leather. The so-called “false membrane” is generally described as composed of fibrine assuming a fibrillated arrangement, and enclosing in its meshes epithelial cells more or less transformed into pus; but the recent observations of Wagner, Cornil, and Ranvier, (with which my own agree, seem to show that in most instances, at all events, there is no fibrine at all, but that the membrane is made up entirely of altered epithelium-cells, some of which become infiltrated with an albuminoid substance, lose their nuclei and cell-wall, and become converted into homogeneous branching masses.* (Fig. 5.) This cellular structure accounts for the fact that, however

Fig. 5.—Modified Epithelium-Cells from a Diphtheritic False Membrane. (Cornil and Ranvier.)

thick and consistent these false membranes may be during life, they are usually breaking down and becoming disintegrated by the time that a *post mortem* examination is made. Membranous inflammation is often preceded by catarrhal, of which it appears to be merely an intensified form; and we constantly observe every stage of gradation between the two. In croup, of which I show you here a specimen, the inflammation may be membranous in the larynx, and catarrhal in the

* See Cornil and Ranvier, *Manuel d'Histologie Pathologique*, 1869, p. 90.

bronchi, with a gradual transition from one to the other. Moreover, you must remember that, although membranous inflammation is often excited by a specific poison, as in diphtheria, yet an inflammation which is anatomically identical may be produced by other causes, such as severe irritants of a non-specific character. Children, who sometimes swallow by mistake boiling water from the spout of a kettle, are liable to die of croupal inflammation of the larynx.

[To be continued.]

ABSTRACT OF TWO LECTURES

ON

THE GEOGRAPHICAL DISTRIBUTION OF TYPHOID FEVER IN ENGLAND AND WALES.*

Delivered at St. Thomas's Hospital, London.

By ALFRED HAVILAND, Esq.

PRESUMING that the specific morbid poison which causes enteric or typhoid fever in man is either primarily engendered *de novo* in his body, is contained in his excreta, or is formed within or without his body, independently of specific disease, during the process of decomposition, the facts already described show that there are certain physical, geological, and meteorological conditions which favour the kindling and the transmission of the typhoid *fomes*, and certain others that are opposed to its development and extension, both which sets of conditions are such as *a priori* we should, from the history of the disease, be led to expect.

1. In an elevated part of country, having an abundant rainfall and a watershed of hard unabsorbent rocks, such as some of the primary formations, the carboniferous limestone and millstone grit, which afford a good natural drainage, through which the meteoric water flows in torrential streams, pure, forcible, and quick, and beneath which the spring-water has to be sought at a considerable depth, we find conditions opposed to the development of the typhoid poison.

2. The opposite set of conditions are, low margins to the catchment basins, a corresponding scanty rainfall, a gently sloping and absorbent watershed, terminating in a flat district through which the rivers wind their way in sluggish course; for there the springs are mostly superficial, being derived from an absorbent soil overlying one of a tenacious character like clay, and easily admitting not only the percolation of rain, but of every soluble matter that may be lying on the surface at the time of its fall.

3. The parts most exposed to the direct influence of the sea and other winds, and even inland localities, are liable, when the mouths of main drains are partially open during the ebb of the tide, whether in rivers, as in the Thames, or on the sea-coast, to have their sewer-gases forced back into the houses or into the streets through gullies, producing, especially during the night and early morning, when the vital powers are at their lowest ebb and the body fatigued, the most poisonous effects.

After this simple outline, I think it will be evident that the study of the geographical distribution of disease is the only true mode of becoming acquainted with these conditions, the only safe guide in all our legislation intended to mitigate the evils arising from this and many other diseases, which owe their spread to similar conditions; and that in all our designs for the disposal of sewage, by attending to its teachings, many of the fatal errors already contracted in great drainage-works and in model sewage-farms would be avoided.

Of one thing there can be no doubt—and upon that alone hangs either our success or failure in any attempt the legislature may make to prevent the ravages of typhoid fever—before anything else is done, pure water must be provided for all towns and villages. The disposal of the sewage will become a very secondary consideration when pure water is supplied. The poor suffer from *drinking* the poison—the rich from *inhaling* it; the poor *must* be cared for, and the first step to be taken is to ensure them pure water. There are few villages in England and Wales where this great boon could not be achieved temporarily until a more complete system throughout the country be organised.

I shall now give a short history of some of the epidemics which have been notable during the last twelve years; and we shall find that, although the cause ever remains the same, the variety of modes in which

man is liable to inhale it is so remarkable, as to form another proof, if one were needed, of the dire necessity of a perfectly independent water-supply throughout the country, even in those parts where the conditions are so favourably antagonistic to the life of the poison.

According to the researches of Hirsch, typhoid fever is, unlike typhus, met with in the Tropics, where, probably, it has often been mistaken for remittent fever. It has been recorded as having broken out in Bengal, Madras, Sumatra, Java, and Syria, Algeria, Egypt, and the Isle of Bourbon; and Dr. McWilliam, in his Niger expedition, gives such an account of its phenomena as to leave no doubt that he came into contact with it as well at Sierra Leone. In North America, it is described as existing from Greenland to the Gulf of Mexico. In Central America, in California, in Brazil, and Peru it is common. New Zealand and Van Diemen's Land have also been the seats of this disease, and perchance many other countries not in this list. (Dr. Murchison.)

The lecturer then proceeded to give a short history of each of the epidemics of enteric fever, which had been examined by Drs. Buchanan, Ord, Bristowe, and other physicians appointed by the Medical Department of the Privy Council. He remarked that the elder Disraeli had written the *Curiosities of Literature*, and he knew not where to find better material than was contained in the reports of the above-mentioned gentlemen for a work on the *Curiosities of the History of Typhoid Fever*; although, as regarded one on its *Amenities* as a sequel, some difficulty might be experienced even by a Disraeli or a Simon.

These histories told the same tale throughout, which might be summed up in the simple words, "poisoned air and water", resulting from either leakage or soakage. The better classes suffer from the inhalation of mephitic air within their houses, as in the cases of Windsor and Scarborough, and a thousand other unreported instances; and the poorer classes, whilst from the absence of water-closets in their dwellings they escape the dangers of their richer neighbours, fall victims to the poison by drinking the water of superficial wells having direct or indirect communication with sewage. Both classes are, however, subject to the same form of poisoning; and, as an instance, Dr. Ballard's lucid exposure of the milk-fever in Islington some years ago was given.

Mr. Charles E. De Rance of the Geological Survey had furnished him with several instances of the connection between fever and geological conditions. In a letter which he had just received, that gentleman remarked: "The old Northmen, when they first colonised West Lancashire, appear invariably to have chosen sites on the 'middle sand gravel', owing probably to the easy access to water which it afforded—as Preston, Lancaster, Chorley, Leyland, Euxton, Kirkham. But this sand, from being extremely free from clay, porous, and open, is exceedingly liable to surface contamination. Thus at Leyland, where enteric or typhoid fever was prevalent in the village, the principal well is close to the churchyard, having sand dipping towards it. A specimen of this water is in the South Kensington Museum, and is described in its *Food Catalogue*. At Euxton the conditions are the same, and the water smells horribly."

Freebridge Lynn, in which Sandringham, Grimstone, and West Newton lie, is in the same category with Witham: it has a mortality below the average, it is true, although only one less than the annual average death-rate from typhoid fever—viz., 8. Freebridge Lynn had, in 1861, a population of 13,486; for the ten years represented on the chart the mean population was estimated at 6,728 males and 6,794 females. Fifty-four deaths were registered under typhus among the males, and 55 among the females, or as 8.0 to 8.1 as the average annual rate of mortality to every 10,000 living. It is bounded to the north by Docking, and to the south by the river Nar, which separates it from Swaffham. Its western boundary is divided into two equal parts; the northern has an aspect facing the Wash, and the southern lies on the bank of the Nar, in its course to join the Ouse.

On looking at Greenhough's Geological Map of this part of Norfolk, we find that the three districts—Docking, Freebridge Lynn, and Downham—succeed each other in a straight line from north to south. In my chart, Docking and Downham are each coloured blue of the third degree; the first having a death-rate of 10 (9.6) and the last 11.2. The mean death-rate of this portion of Norfolk would then be, if we exclude King's Lynn, which has an annual average mortality of 10.3, equal to 10 (9.8).

We find chalk prevailing from the north to the south of this group, skirted to the west by what Woodward, in his outline of the geology of Norfolk, calls *carstone*, but which now is designated upper greensand, lying on gault or golt, a provincial term for clay of any kind, but which has been applied to that particular formation lying between the upper and lower greensand; it is also called Tetsworth or Folkestone clay. An old term for the upper greensand is *firestone*. To the west, again, of this formation we see in Freebridge and Downham districts two

* Continued from page 149, Feb. 10th, 1872.