

Zymotic diseases : their correlation and causation / By A. Wolff.

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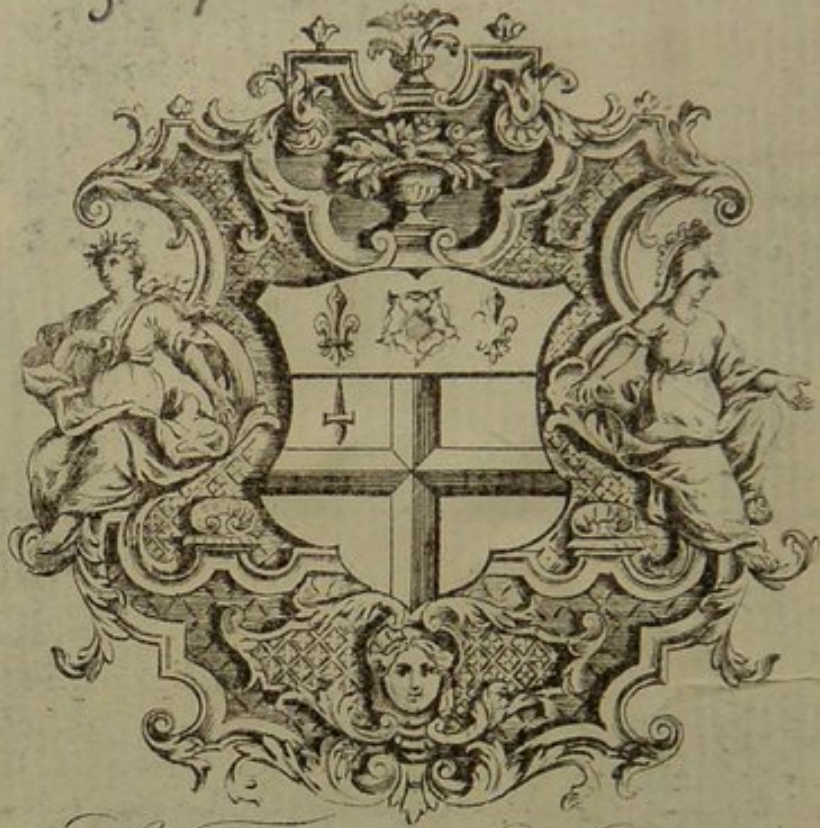
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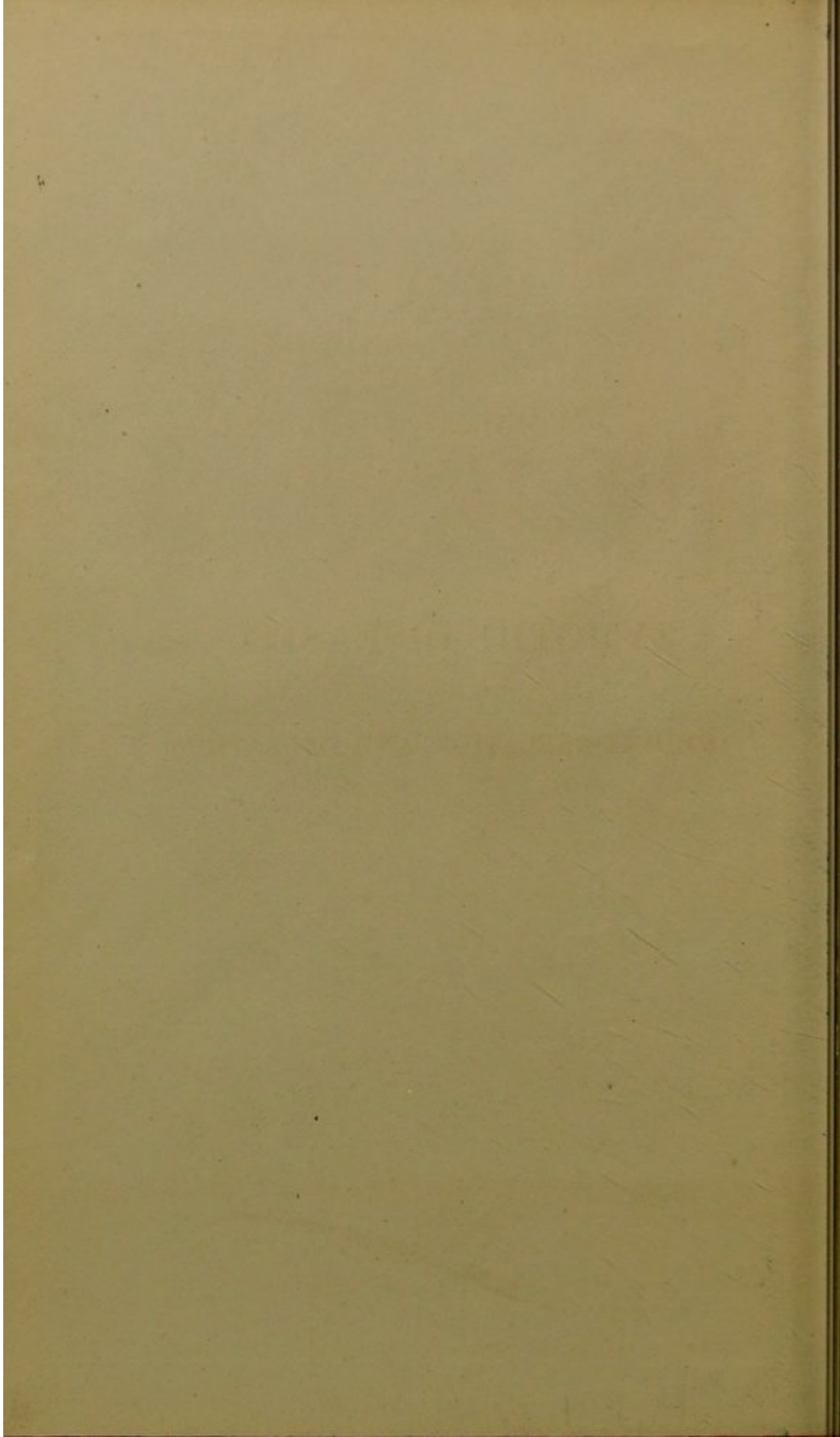
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ZYMOTIC DISEASES:

THEIR CORRELATION AND CAUSATION.

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ZYMOTIC DISEASES:

THEIR

CORRELATION AND CAUSATION.

BY

A. WOLFF, F.R.C.S.



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P R E F A C E.

IN a pamphlet published in January, 1871,* I dissented from the doctrines generally prevailing with regard to the causation and the nature of the (so called) zymotic diseases. I expressed a doubt as to the existence of the special poisons to which they are supposed to owe their origin, and an opinion that the correlation that unites them is more intimate than has been generally supposed. The present volume is a further effort in the same direction.

The doctrines advocated in the following pages are not original. Most of the views expressed in them may be found scattered in the writings of various medical authors, ancient and modern.

I have used the word zymotic, the term used in the reports of the Registrar-General, and the most popular mode of designating the diseases we are about to consider, without reference to any theory of their causation.

In the sketches of the different diseases I have

* "The Correlation of Zymotic Diseases."

selected those most commonly met with, and with whose phenomena we are most familiar. To have included all the diseases known as zymotic would have largely increased the bulk of the volume without adding to the strength of the argument.

These sketches have no pretension to be considered as finished pictures. They should rather be looked upon as photographs taken partly from nature and partly from well-known pictures by celebrated artists.

A. WOLFF.

66, GOWER STREET,
December 14th, 1871.

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ZYMOTIC DISEASES.

INTRODUCTION.

RECENT investigations by pathologists have tended to cast much doubt upon the doctrines that have been long held respecting the specificity of diseases. Dr. C. J. B. Williams, in his most recent work upon the Pathology of Phthisis, tells us that the conclusion at which he has arrived is that tubercle is not an adventitious product, but an effect of perverted development of the normal elements of the body; even cancer that has been for so long accepted as the type of heterologous product, is now, like tubercle, thought to be, not the growth of a foreign element amongst the textures of the body, but rather a disproportion between the growth and development of the normal elements.

In that large and important class of diseases that is comprised under the term zymotic, the doctrine of specificity still holds its ground, and writers as well as lecturers on these diseases, teach that zymotic diseases depend upon specific poisons, and that

each variety of zymotic disease has its own special poison. Recognising the fact that the cause of zymotic disease is something external to, and distinct from the body, various attempts have been made to discover the actual material cause of these diseases. At one time it was supposed that the cause was a fungus, and inquirers searched the vegetable world to discover the fungi that are associated with disease. Some investigators claimed to have discovered the fungus that causes intermittent fever, and various experiments were made to test the truth of the supposed discovery; other observers favourably placed hoped to have discovered in a fungus common in rice-fields the cause of cholera, but further experiments proved the fallacy of these supposed discoveries.

Investigations were also made in another direction, and those minute forms of organized matter that are met with on the extreme confines of the animal world were thought to be in some manner the cause of disease. It is noted that these minute organic beings are present in different stages of the decay of organized textures, and it is assumed that their presence is due as a cause rather than as a consequence of such decay. One of the earliest amongst the hypotheses that have been put forward on this subject, is that the atmosphere is constantly pervaded by a number of so-called morbid poisons. The nature of these poisons is not described, but

it is supposed that they gain access in some manner to the bodies of those specially predisposed (it is not explained in what this predisposition consists), and that having obtained access they cause the disease that is their special consequence, and that during the course of this disease the mass of poison is indefinitely increased, and becomes the cause of a further extension of the disease in the bodies of those with whom it may have contact.

Grave doubts may, however, be entertained whether the truth really lies amongst any of the theories that have been glanced at above, and even whether it be possible to maintain the distinct and separate character of the various forms of zymotic disease. The object of the following pages will be to show that the origin of these diseases can be accounted for, and the phenomena exhibited during their course explained, without assuming the existence of certain entities called morbid poisons, to show that in every stage they are governed by the same laws that regulate other organic processes, healthy and diseased. In short, that the genesis, the course, and the distribution of the so-called zymotic diseases are shaped by the same great natural laws that govern growth and regulate decay.

We know that health consists in a proper equilibrium between the waste caused by functional activity and the repair supplied by normal assimilation, and that when this equilibrium is disturbed

and the destructive overcomes the assimilative action, disease ensues, that may result in the death of a texture, of an organ, or of the individual.

A consideration of the manner in which death usually occurs shows, that it may be roughly divided into three modes.

1. *Natural Death*.—When the different organs of the body have gradually and synchronously lost the power of continuing their functions, and death results, from what is graphically described as Natural Decay.

2. *Premature Death*.—When some organ essential to life takes on an action of disintegration, and death ensues as a consequence, the other organs being healthy, as when—*e.g.*, a kidney, heart, or liver becomes the seat of organic disease.

3. *Accidental Death*.—In this class would be comprised the diseases we are about to consider.

All the organs being healthy, and during the perfect performance of their functions, some cause external, and in a measure accidental, causes them to take on an action that is an interruption of their natural healthy functions, that disturbs the harmonious balance of waste and repair of the tissues—that causes these tissues to take on an action that we recognise as a form of inflammation—that leads to their disintegration, and may result in their death.

Whenever it has been possible to trace zymotic disease to its commencement we shall find such

origin invariably marked by the presence of decaying organic matter, of organic matter that is gradually undergoing a process of disintegration, and we must bear in mind what active molecular motion is continually going on in bodies that we roughly class as dead—a molecular motion that possibly is the more intense from being in the one direction of disintegration—the molecular action that converts muscle into adipocire is probably as intense as that acting during the greatest functional activity of the living structure.

Abundant evidence will be found in the following pages of the extension of this disintegrating, this dying action in accordance with a well-known physical law, and it is to this extension we must look for the true explanation of the cause of zymotic disease.

That decaying organic matter is the cause of zymotic disease is one of the earliest doctrines held upon this subject; although the manner in which this cause acted was not clearly pointed out, the obvious and constant coincidence of the two conditions could not fail to lead to the idea that they stood in some manner in the relation of cause and effect.

The two main arguments that have been adduced against this doctrine are:—

1st. That persons who from their social position are brought into constant contact with this sup-

posed cause, are not specially prone to become the subjects of zymotic disease.

2nd. That the supposed disease-producing substance retains its maleficent power after long intervals of time, as, *e.g.*, when an article of clothing that has been worn by a patient is packed into a drawer or box, and causes a fresh outbreak of the disease weeks or months later, when worn by another person.

It is possible to answer both these objections. That butchers, nightmen, and others whose occupations cause them to be constantly subjected to the influence of decaying organic matter do not necessarily become the constant subjects of zymotic disease, is explicable upon the doctrine of immunity, as will be more fully explained in the ensuing pages.

The natives of a fever-infested district are not the chosen victims of the disease, which fixes in preference upon the recent sojourner.

In reply to the second objection, the physicist may be reminded that the processes of growth and of decay may be temporarily arrested, the substance still retaining what is well understood as the "potential energy" of continuing either process when placed under favouring conditions: Perhaps the most familiar and striking example of this fact is the case of the Egyptian mummy whose winding cloths contain some grains of corn. Here the

process of decay in the mummy and the process of assimilation and growth in the corn-seed have been arrested for ages; yet when placed under favouring conditions both processes are at once resumed—the corn-seed germinates and grows—the mummy crumbles into dust.

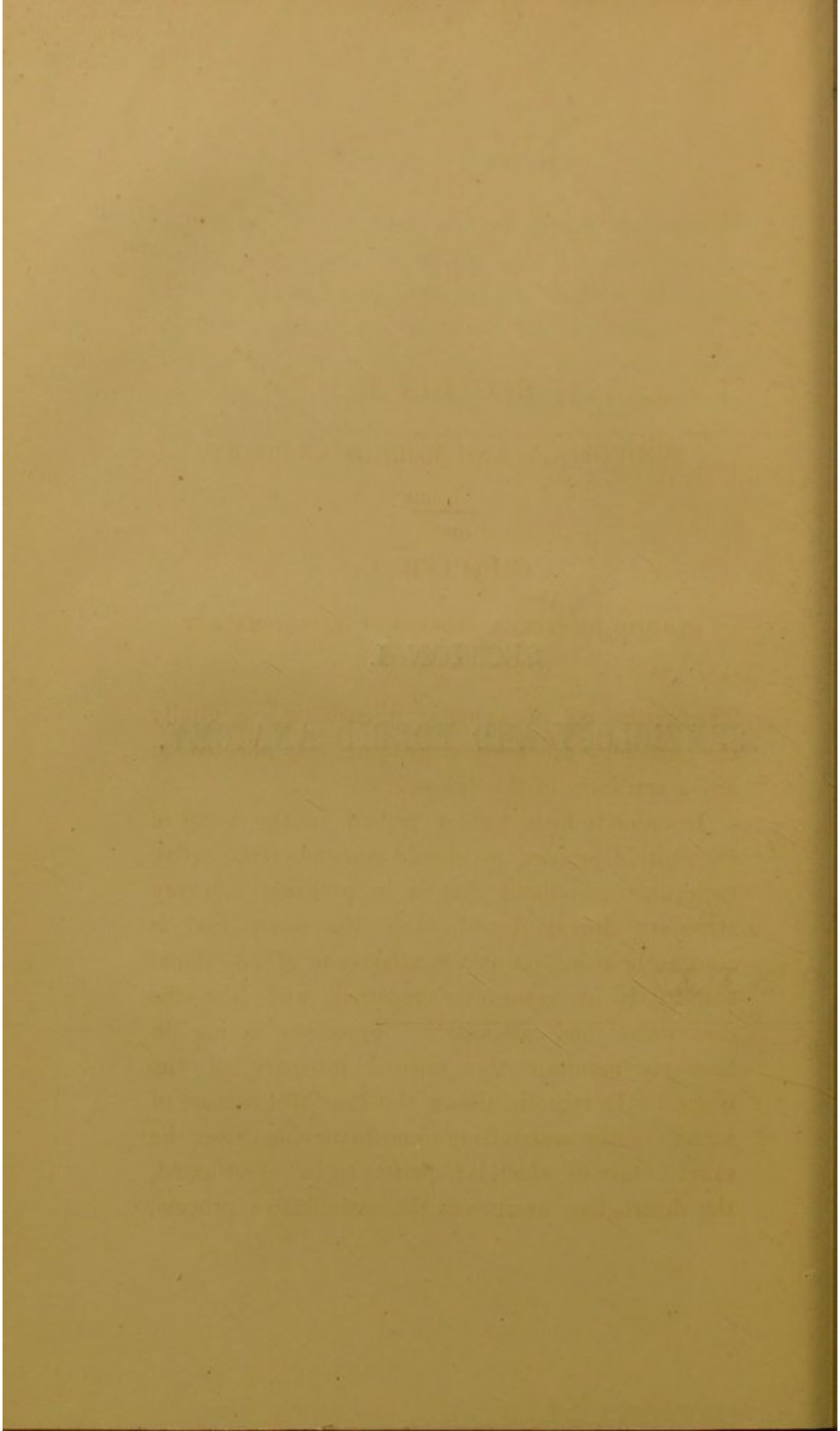
The doctrine advocated in these pages brings the phenomena exhibited by zymotic disease within the range of laws that are well understood by the physiologist and pathologist. It rejects the idea of diseases as special entities or as the result of peculiar organisms whose nature and origin are unexplained, and it brings the human body within the action of those great physical laws that govern all other forms of matter.

It would be difficult to over-estimate the importance of clearly and distinctly understanding the cause and the nature of zymotic disease—important both in a prophylactic and curative sense. For when the correlation of zymotic diseases shall be fully recognised, the efforts of physicians to avert or counteract the effects of the disease-producing cause will be directed to a single and well-defined point; and the recognition of decaying organic matter as the invariable cause of the prevalence of zymotic ailments; and of faulty hygienic surroundings as one of the great factors in their diffusion, will show that the duty of guarding society against pestilence should be divided between the physician and the

legislator—that whilst the physician indicates the point from which the attack is to be expected, and points out the means by which its evil effects are to be averted, the duty of the legislator is to frame laws that shall place the population under the most favourable possible condition for offering an efficient resistance.

SECTION I.

SEMEIOLOGY AND MORBID ANATOMY.



SECTION I.

SEMEIOLOGY AND MORBID ANATOMY.

CHAPTER I.

ZYMOTIC DISEASE, A PROCESS OF INFLAMMATORY DISINTEGRATION.

THE pathology of zymotic disease is essentially the pathology of the texture primarily affected in any given form of the disease.

In order to form a clear picture in the mind of the morbid process, we should remember the active molecular movement that is in progress in every structure during health—how the waste that is constantly going on as a consequence of functional activity is as constantly repaired, and how the destructive and assimilative processes acting in harmony maintain the normal integrity of the tissues. In zymotic disease this healthful balance of action is interrupted; from some disturbing cause, the exact nature of which is hereafter to be investigated, the destructive overcomes the assimilative process,

and in the symptoms exhibited during the course of any form of zymotic disease, and in the structural changes that may at times be found as their consequence, we can clearly trace the different stages of textural irritation, textural inflammation, and textural death. For in each and every form of zymotic disease, whatever be the structure affected, the action is essentially the same. It is disintegration of tissue we witness, not the production of any new abnormal structure, but the death, or an action tending toward death, of the already existing tissues.

When we study the pathology of the various forms of zymotic disease we find these facts clearly borne out both in the symptoms and also in the structural changes that we are able to trace after death. The symptoms are found to be precisely those that would naturally follow on inflammatory action excited in one tissue where the diseased action originates, and extended from that tissue, in accordance with natural and well-known laws, to other tissues, and sometimes to all the structures of the body ; and if the disease have resulted in death the dead-house gives no contradiction to these views, but shows in the dead structures how the process has been essentially a process of inflammatory disintegration.

Physiological observers have furnished us with abundant proof that the various processes of in-

flammation are different stages of disintegration of tissue tending towards death. The experiments of Magendie prove that when from starvation the cornea perishes, it perishes by a process of inflammation and ulceration. It is perhaps in a structure like the cornea that the process of inflammation can be best studied. In the state of health the balance of waste and repair is so exact that although we know that molecular change must be going on, the organ maintains its perfect transparency, and even under the microscope exhibits no trace of structure. But when from some cause of irritation the balance is disturbed, this condition is at once changed, and we can watch the inflammatory process changing the appearance of the organ, injuring its function, and if the disease goes on unchecked, destroying its structure.

Stricker and Burdon-Sanderson in the course of their investigations into pyogenesis have carefully watched the microscopical appearances of the inflammatory process, and have detailed the progressive changes of structure from the first moment of irritation through the various stages of disintegration. Perhaps no better description can be given of healthy structure disintegrated by a process of inflammation than is contained in the description by Dr. B. Sanderson of inflamed cartilage. "The normal cartilage cell, like every other active cell, is a mass of protoplasm containing a nucleus."

“When cartilage is irritated, as, for example, by scraping its surface, the cells in the neighbourhood of the irritation enlarge, and consequently expand their capsules. The protoplasm of which each cell consists becomes more granular, and soon it is found that the mass contains two corpuscles in its interior instead of one, and that each has a gathering of protoplasmic matter around itself. This process of division is repeated in each segment until every cavity contains a mass of nucleated cells, which at length assume characters corresponding with those of newly formed pus corpuscles, while at the same time the original interstitial substance gradually wastes away, and is finally represented by a sponge-like stroma, in the holes of which the young cells are contained.”

But perhaps the strongest evidence as to the fact that these inflammatory processes are acts of disintegration, that as far as regards the texture inflamed they are in fact a death action, is contained in the work of Mr. Simon on Inflammation :—

“It is not in regard of these last-mentioned extreme degrees (rodent ulcer—gangrene) that the student runs the risk of overlooking the reality of destructive changes as an essential part of inflammation. But both for pathology and practice it is needful that he recognise the same reality in cases where it is less obvious—that he learn to trace

the anatomy of inflammatory destruction from its grossest to its most minute phenomena.

“Let him examine inflamed muscle, as for instance in the post-mortem examination of a compound fracture or a recently made stump. He will find the structure weakened so that it easily gives way with pressure or traction; he will see under the microscope that the substance tends to fall into irregular fragments, that the natural striation is more or less replaced first by an almost homogeneous appearance and afterwards by an appearance of aggregated granules; that with these granules of albuminous matter into which the muscle has resolved itself there is mixed, even from an early date in the inflammation, a noticeable quantity of minute oil-drops, that often these oil-drops appear before the disintegration of muscle has made much progress, and then arrange themselves in such mutual relation, transverse or longitudinal, as to suggest that the sarcous elements have changed themselves particle by particle into oil; that little by little the oil-drops multiply to such an extent as to be the chief visible objects, the limitary membrane of a fasciculus seeming now to be almost filled with finely divided oil diffused through some scanty connective albuminous material; that the limitary membrane within which the muscular material is thus emulsionized tends also itself to undergo dissolution and let its pro-

ceeds confuse themselves with the similar débris of neighbouring fasciculi till more or less bulk of muscle is reduced to a state of oleo-albuminous liquidity. And from this point, if the observer have opportunity of watching the changes which lead to convalescence, he will see that gradually the liquefied material diminishes in volume; that in proportion as it vanishes the adjoining parts adapt themselves to the altered relation; that eventually only a scar-like puckering of substance—a kind of tendinous intersection—remains to mark the place where muscular material has irrecoverably melted away.”—MR. SIMON, *Holmes' System of Surgery*, Art. “Inflammation.”

In tracing zymotic disease as it affects the various textures, we shall find that the signs that are generally accepted as pathognomonic of inflammation are not absent, that, as in the more common forms of inflammation, the pain, the heat, the redness, the swelling, attest the nature of the molecular changes that are in progress.

We will now examine the more important varieties of zymotic disease, trace the symptoms that are produced as the various textures are primarily affected, and inquire into the textural changes that are discovered by post-mortem examination.

CHAPTER II.

TYPHUS—CHOLERA—ENTERIC FEVER.

TYPHUS.

Texture primarily affected—The Blood.

THE course that the symptoms of zymotic disease pursue when the action affects the blood primarily is best seen in a case of typhus fever.

The disease is usually ushered in by some days of undefined illness, the patient complains of feeling unwell, has severe headache, pains in the back and limbs, a feeling of weakness and languor, disinclination for usual occupation, loss of appetite, thirst, the countenance has a peculiar dusky hue, the conjunctivæ are injected. The symptoms are not sufficiently defined to excite alarm—the most prominent are the intense headache and a feeling of excessive weakness that is greater than appears explicable by the other symptoms. The tongue is white and appears to be coated with a creamy fur, the pulse is full and soft and not excessively quick. After two or three days there is probably a decided rigor, the nights become disturbed by sleeplessness

and delirium that sometimes ceases during the day to be renewed the following night, the pulse rises, and the normal temperature is increased to 103 or 104. The patient is now forced to take to his bed, and the symptoms gradually become more severe. About the fifth or sixth day an eruption of small dusky, pinkish spots is perceptible over the abdomen, chest, back, arms, and thighs. These spots are slightly raised above the surface and disappear momentarily on pressure. The headache ceases now to be a prominent symptom and is replaced by excessive drowsiness, amounting almost to stupor; the patient lies in bed supine, and the cerebral functions are so much impaired that he appears like one suffering from concussion of the brain; he takes little or no notice of surrounding objects; speaks only when roused, and then subsides at once into a condition of apparent insensibility with feeble moaning or muttering delirium. The tongue becomes brown, this change commences in the median line, and the dark brown fur gradually encroaches on the surface until the entire upper surface is covered by it; the organ is protruded with difficulty or with a tremulous movement; the tremor is noticeable also in the hands. The secretion of saliva stops, and the mouth becomes quite dry; the teeth are covered with sordes. As the case proceeds power over the sphincters is lost; the patient is unconscious of the passage of fæces. The urine is either retained, and has to

be drawn off by catheter, or it dribbles away; he lies upon his back and appears unable to maintain even that position, but keeps slipping down to the lower part of the bed; there are muttering delirium and subsultus tendinum; the eruption becomes darker in colour, and looks like small spots of purpura; the spots do not now disappear on pressure; there is a peculiar fœtid odour both from the breath and the skin; sometimes the skin over the sacrum becomes dark and sloughs. As the case draws near its termination the symptoms become more intense, the stupor becomes coma; the pulse, from being rapid and weak (120 to 140), becomes uncertain, fluttering, and is with difficulty to be felt; the temperature falls, the extremities become cold, and unless a decided improvement takes place (and this improvement sometimes occurs very suddenly and decidedly), the patient dies about the twelfth or fourteenth day.

Post-mortem Appearances.

It has been noticed that putrefaction is more than ordinarily rapid after death from typhus fever, and that the stage of muscular rigidity is of short duration and ill-defined.

There is dark discoloration of the skin in patches, and this is more marked in the course of the large venous trunks.

The muscles are darker than usual, and their

texture is less firm than after death from other causes. This softening of muscular tissue is most marked in the heart, and all observers are agreed that the muscular texture of this organ is much softened after death from typhus; indeed, this is, perhaps, with the exception of the alteration in the blood, the most decided and constant post-mortem appearance.

The substance of the heart is said to be flabby, soft, and easily torn, and Dr. Murchison says that under the microscope these softened hearts exhibit all the appearance of well-defined fatty degeneration.*

The lungs are generally found to be congested.

The brain shows a remarkable freedom from structural change, notwithstanding the prominence of head symptoms during life. Sub-arachnoid effusion is, however, often present.

There is no special appearance in the stomach and intestines.

The kidneys and liver are generally found gorged with fluid blood, and not unusually exhibit signs of disintegration in being softer than natural, and their substance easily broken down.

The blood is much changed in typhus fever; it is dark and fluid; such clots as are found, instead of being firm in substance and pale in colour, are

* The particulars as to post-mortem appearances are from observations made by Jenner, Murchison, Peacock, and others.

black and very soft. The red corpuscles, diminished in number, when examined under the microscope are found aggregated in heaps, instead of arranging themselves in the usual rolls. They are also found to have lost their circular form, and to have irregular crenated edges, as if undergoing an actual process of solution. It has also been observed that the blood from the body of a patient who has died of typhus becomes rapidly putrid.

In the symptoms and their consequences here delineated we can distinctly trace the nature of the diseased action that has been in progress.

The blood exposed through the respiratory organs to contact with decaying organic matter has taken on the disintegrating action. This partially devitalized blood, as it circulates through the various organs, produces those functional disturbances that we see marked in the symptoms. The brain being especially the organ in which impairment of function is most obvious, evidences the earliest and most distinct deviation from normal action—the headache, the diminished perceptive power, the stupor, the loss of co-ordination of muscular action, the delirium, and finally, the coma—all these symptoms occurring in a brain that shows no structural lesion, are evidently the consequences of the circulation through that organ of the disintegrating blood. The heart, filled with this diseased blood, continues

in its own substance the disintegrating process: its muscular structure softens, breaks down, becomes fatty—in short, begins to die; the feeble action, the diminished impulse, the first sound impaired or lost, mark during life the progress of this disintegrating process; the gorged liver and kidneys, the darkened and softened muscles, arise from a similar cause. The glands supplied by the diseased blood cease to furnish their usual secretions, the flow of saliva stops, the mouth is filled with epithelial débris, giving to the teeth and tongue the aspect characteristic of the disease. Minutely examined, the petechial spots tell the tale of fluid and disintegrated blood that has exuded from the capillaries; even the respired air returns from the lungs charged with unusual elements of disintegrating tissue, and, submitted to chemical tests, betrays the presence of large quantities of ammonia.

CHOLERA.

Our knowledge of the physiology of the nervous system is so imperfect, and the phenomena that attend its diseases so obscure, that there is great difficulty in distinctly tracing the effect of disintegrating action in these structures. We might easily predicate that whatever structure is attacked and disintegrated by zymotic disease the nervous system, and particularly that portion whose especial function appears to be connected with the processes of organic life, would be early and extensively affected by the morbid action. Observation and experience show this to be the fact. In all the various forms of zymotic action we notice that when the diseased action is set up in its intensest form, life is destroyed before any special kind of symptoms is established; whatever be the prevailing form of disease, be it Cholera, Plague, Typhus, Scarlatina, Variola, we meet with cases where death takes place so rapidly, with symptoms that in all these cases are so nearly identical, that it is only by a knowledge of the prevailing epidemic, or by the subsequent family history, that we are enabled to give a name to the disease that has destroyed life.

In cholera this intense action is most frequently

witnessed. "When the cholera reached Muscat, in some instances only ten minutes elapsed from the first seizure before life was extinct. In one case a merchant on board the *Conde de Rio Pardo* was in the act of closing a bargain, when he suddenly vomited twice, fell down, and expired. Many natives at Hoobly were attacked while walking in the open air, and having retched, complained of vertigo, blindness, or deafness, fell down and expired in a few minutes. Mr. Gordon gives the case of a tailor at Bellary, who expired with his work in his hand. At Punderpore, also, the disease is said to have raged with such severity that 350 persons died in the streets as if knocked down dead by lightning."—

Dr. R. WILLIAMS, Dr. GUY.

Dr. Hodges, on *Plague* of 1665, writes that "some of the infected ran about, staggering like drunken men, and fell down dead in the streets, or they lay there comatose and half dead; some lay vomiting as if they had drunk poison, and others fell dead in the market in the act of buying provisions."

M. Bacot, on *Typhus* in Army of Peninsula, 1812, says that "the patients usually came into the hospital complaining of chilliness, languor, and depression, both of strength and spirits. Their countenance was wan and melancholy, and the surface of the body unusually cold to the touch; giddiness of the head was a frequent complaint, and deep and constant sighing was an universal symptom.

I have seen numbers of men brought into the hospital so attacked die in twenty-four or thirty-six hours after admission, without a prominent symptom, insensible to every kind of stimulus, and never having had any increased vascular action or accession of heat from the moment of their attack to the hour of their death."

Dr. Williams tells us that "in the severe forms of fever that prevailed during the years of the Cholera Indica in this country, it was not unusual to see patients brought into the London hospitals after only a few hours' illness with an attack of typhus, their bodies cold and covered with petechiæ, the pulse little excited, their faces bloated and almost purple, their conjunctivæ red, die in a few hours or a few days without any very prominent symptoms, except, perhaps, expectorating a small quantity of blood from their loaded lungs. These patients were apparently destroyed by the great depressing action of the poison, no organic lesions of any moment being discoverable on the most minute examination after death."

Sydenham, on *Small-pox*, writes:—"Sometimes, instead of fever being produced, the patient sinks at the beginning of the disease, when the morbid matter cannot disentangle itself and come out, by reason of the confused and irregular motion raised in the blood, bloody urine and purple spots succeeding and closing the scene."

Every physician must have met with cases during an epidemic of scarlatina where a child in apparently good health up to a given moment was suddenly seized with vomiting and purging, and then rapidly sank into a state of collapse, death ensuing without any of the characteristic symptoms of the disease being developed, and the physician must have felt some doubt as to the cause of death until, other cases of the disease in the same household occurring, and following the usual course, satisfied his mind as to the nature of the destructive agent.

These descriptions apply to those intense forms of zymotic disease when death occurs from what would formerly have been described as nervous shock, before there had been time for the characteristic symptoms to develop. Usually this effect upon the nerves of organic life is represented by what is generally described as the premonitory stage, and does not form a very prominent feature, except in the disease that we are now about to consider.

An attack of cholera very commonly begins in the night, some hours before dawn; the person seized may have had some premonitory looseness of bowels for some days previously, or may have retired to rest apparently in perfect health; he is roused by a feeling of sickness, quickly followed by excessive vomiting and purging; at first the stomach and intestines are emptied of their usual contents, and

as the vomiting and purging continue, the matter expelled consists of a clear serous fluid rendered slightly turbid by a white flocculent matter; soon violent and most painful cramps set in, commencing in the hands and feet, and extending to the legs and arms; the belly of the muscle is contracted into a hard ball, causing intense suffering; the skin is cold and covered with a clammy sweat;* but there is a sensation of intense heat about the pit of stomach, and there is great thirst. The drink taken to allay this thirst is immediately expelled from the stomach; the change in the aspect of the patient is great from its extent, and startling from the suddenness with which it has occurred; within a very few hours from the commencement of the attack—sometimes within a single hour—he appears to have aged by many years; the features are sharpened in outline, there is a circle of livid darkness round the eyes, which appear deeply sunk in their sockets—the corneæ are hazy and flaccid, like those of a body that has been dead for some hours; the hands and nails have a bluish tinge, and the skin of the fingers is corrugated as if it had been immersed for hours in water; the voice is quite changed; it has become husky and whispering; the vomiting and

* This coldness is not merely a sensation; there is a decided and very considerable diminution of temperature that has been known to fall as low as 83° Fah.

purgings still continue; the cramps become more violent, the blue tinge spreads from the fingers and toes up the arms and legs; the face becomes livid; no pulse can be felt at the wrist; there is total suppression of urine; but notwithstanding these terrible symptoms and the frightful suffering they entail, the consciousness and intelligence remain unimpaired; the brain appears to be the only organ not affected.*

From this state there may be no reaction: the patient may die after lying in the condition above described for a period varying from ten to fifty hours (this is given as the average duration); or an improvement may set in which is marked by a return of warmth to the surface and cessation of the purging and cramps; the pulse can now be felt at the wrist beating feebly and rapidly, the countenance loses in some degree the deathlike hue it has hitherto had: there is no longer the blue tinge of the hands and feet—this stage is marked by a return of the urinary excretion, and the changed appearance of the discharges from the bowels indicates the presence of bile. Although the extreme and immediate danger may now be looked upon as past, the patient rarely passes

* The author has seen many patients (during the epidemic of 1849) retain perfect consciousness to within a very few minutes of death. He has heard patients converse rationally and calmly, though in a low, hoarse whisper, until their voices have been literally hushed by the advent of death.

at once into a state of convalescence; in the great majority of cases the stage of collapse is succeeded by a condition that is exactly like an attack of continued fever. The pulse becomes rapid, the face flushed, the tongue is covered with a brown fur, the teeth with sordes; there is often delirium and generally stupor, the conjunctivæ are injected; the discharges from the bowels are dark and offensive; this condition may last for ten or twelve days, and frequently proves fatal; when recovery takes place, the convalescence is like that from a severe attack of typhus.

Post-mortem Appearances.

The post-mortem appearances in cases that have proved fatal in this stage of collapse throw no light upon the nature of this disease. Some observers have found signs of congestion in the brain and its membranes, and some have even marked serous extravasation; but these occurrences are not sufficiently constant to be of importance.

In the chest the lungs are found sometimes gorged with dark-coloured blood, and in other cases collapsed against the spine; the heart is generally filled with dark blood.

The spleen, liver, and kidneys are found gorged with blood, but show no structural change.

The intestines are filled with the same sort of white serous fluid that was discharged from the

bowels and stomach during the attack ; in some cases the mucous lining of the intestinal canal has been found softened and easily destroyed.

It is stated that blood drawn during the cold stage is black like tar, and of the consistence of syrup.

Dr. Keir of Moscow found the blood-vessels of the vertebral column and spinal cord more or less loaded with blood, which was sometimes effused between its arachnoid and dura-mater; partial softening of the substance of the spinal cord was sometimes met with, and marks of inflammatory congestion in the larger nerves were detected.

Although the symptoms during life and the appearances after death have been carefully studied, observers have not hitherto been able to trace the structure that is primarily affected in this terrible variety of zymotic disease. The phenomena during life present in an intense form that condition of collapse that we see occurring in a minor degree as the earliest symptom in bad attacks of the other varieties, and death commonly takes place before the stage of reaction sets in. The investigations of Dr. Keir appear to point to the spinal cord and ganglionic nervous system as the parts where structural change may be detected ; but the observations have not been sufficiently numerous to enable a decided opinion to be given on this point.

ENTERIC FEVER.

The Mucous Lining of the Intestinal Canal Primarily Affected.

This disease frequently makes its attack in a very insidious manner. The patient feels unwell for several days without any very prominent symptoms; he feels a degree of languor, disinclination for exertion, headache, thirst, frequently there is a slight diarrhœa; the skin feels warmer than natural, pulse quick (110 to 120); there is loss of appetite, the feverishness is increased at night, the tongue is white in the centre, but the tip and edges are more than usually red.

This condition may continue without any very marked change for a week or ten days, the patient not being sufficiently ill to take to his bed; as the disease progresses the symptoms become more pronounced, the heat of skin and thirst increase; the thermometer indicates a temperature of 103 or 104; the pulse becomes more frequent (120 to 140); the looseness of the bowels is increased, and the evacuations have the appearance characteristic of this disease—they are thin and of the colour of yellow ochre; the urine is scanty and high coloured; the cheeks have a hectic flush like that seen in a

phthisical patient; the pupils are dilated. During the second week of the disease, commencing generally at about from the eighth to the eleventh day, an eruption may be perceived on the skin of the abdomen, chest, and back, of small rose-coloured spots very slightly raised above the surface, and disappearing momentarily on pressure. These spots are not generally very numerous and are rarely closely aggregated. They die away after four or five days, and are succeeded by a fresh lot. These successive crops go on appearing and dying away during the whole remaining course of the disease; they do not become petechial like the eruption of typhus.

In this the second week of the disease the abdominal symptoms become more grave; there are five, six, or more evacuations daily; the abdomen is tender on pressure, and pressure excites a gurgling sensation, especially in the right iliac fossa; the abdomen looks enlarged, and feels tympanitic; the tongue now becomes brown in the centre and dry, the tip and edges still remaining red, and sometimes the entire organ looks intensely red and dry, and is furrowed by deep horizontal cracks. At this stage of the disease there is sometimes delirium, especially at night, and the patient lies supine with the eyelids partially closed, and in a dull listless condition, and when roused answers questions languidly. Very frequently bed-sores occur over the sacrum and other

parts subjected to pressure. This condition may continue with but slight change into the third or fourth week of the disease, or longer, when there may be gradual alleviation of symptoms and slow restoration of health; the change is usually very gradual, and is not marked by any special crisis.

The disease leaves the patient very weak and emaciated; in some cases instead of this gradual recovery the symptoms increase in severity, the tongue becomes brown, the teeth are covered with sordes, the slight delirium merges into coma, and the patient sinks with the symptoms of the last stage of typhus; even when the patient appears to be going on well and progressing towards recovery, there may be a sudden attack of sickness and intense abdominal pain, followed by collapse, the pulse excessively quick and weak, or even imperceptible, the countenance anxious; the physician knows by these symptoms that perforation through the peritoneal coat of the intestines has occurred, and that the result will be almost certainly fatal. The usual duration of the disease is from twenty to thirty days.

Post-mortem Appearances.

The body is generally much emaciated; there is no appearance of the eruption; the tendency to rapid putrefaction is not marked.

The brain and its membranes generally show no structural lesion.

The muscular structures are natural; there is sometimes a softening of the muscular substance of the heart.

The lungs are frequently found gorged with dark-coloured blood, and the bronchial tubes are often charged with mucus; the structure of the lungs is generally unimpaired.

The blood is frequently found dark and fluid, especially in those cases that have gone on to a typhoid stage, and Virchow records that in these cases the red corpuscles are crenate, and betray by other signs that they are undergoing a process of disintegration.

The kidneys and liver are generally healthy.

The principal post-mortem appearance is found in the intestinal canal. The mucous coat of the ileum is often found much congested, thickened, and softened so as to be easily separated from the subjacent textures. Peyer's glands, both the solitary and agminated, show signs of inflammatory disintegration in all stages; the most diseased portion is commonly close to the ileo-cæcal valve, and it is here the greatest change is visible; but the disease spreads sometimes over a very large tract. This change may be traced in all stages. At the first appearance of inflammation the glands are more salient than usual, and give to the mucous membrane of the part the appearance of a pustular eruption that has been compared to variola; where the disease has

proceeded further, and ulceration has taken place, the ulcers are seen surrounded by, and covered with, the débris of the disintegrated tissue. Sometimes the ulceration has destroyed the submucous tissues and left the gut protected from perforation by only the peritoneum; where this has taken place can be perceived by examining the peritoneal surface of the gut, where a dark patch shows the point of ulceration; when this last protection has yielded to the disintegrative action, and perforation has taken place, the usual appearances after death by peritonitis are found. If one of the glands be minutely examined, the usual signs of inflammation will be found—much granular matter and abundance of oil globules.

The mesenteric glands near the affected portions of the intestine are much enlarged; they are often seen as large as filberts, intensely congested, and, when cut into, pus is frequently found in their substance.

The symptoms traced in the above sketch, and the consequent changes observed upon post-mortem examination, explain the nature of the changes that take place in enteric fever. Animal matter in a state of putrefaction is brought into contact with the mucous lining of the intestinal canal, and the disintegrating action is extended to those structures. The diseased action is evidenced as might be expected, especially in the tract of the absorbents, for we must bear in mind that Peyer's patches, both the solitary

and the agminated glands, must not be classed amongst those glands that pour out secretions into the intestinal canal, but that they are in fact lymphatic glands. These structures take on them the diseased action—they swell, they redden; in fact, they inflame. The inflammation proceeds to ulceration, to sloughing; the structures pass through the usual stages of inflammatory disintegration. Microscopical examination exhibits the usual appearances of dying structure; there is no trace of any foreign material, but there is much granular matter and abundance of oil globules. The character of the alvine evacuations affords additional proof of the nature of the local change; their odour is very offensive, and they are alkaline, whereas healthy fæces are always acid. The mass is made up of undigested food, intestinal epithelium, and blood corpuscles in a disintegrating condition, and a large quantity of crystals of triple phosphate: these evacuations also show a tendency to unusually rapid putrefaction. The amount of urea excreted is increased, the normal 400 grains daily is increased sometimes to double that quantity, or even more; the diseased action, as it extends to other structures, excites in those structures not only symptoms special to enteric fever, but the symptoms that would be naturally exhibited by structures undergoing a process of inflammatory disintegration. If the action be extended to the blood, the symptoms induced are exactly those that

may be observed in typhus, and the patient dies with the brown tongue, the teeth covered with sordes, the coma, and all those symptoms that we see ensue when disintegrating blood is circulating through the various organs. The inflamed mucous membrane of the intestinal canal extending upwards towards the jejunum and beyond the cæcum to the colon, whose glands often take on the diseased action, the enlarged and inflamed mesenteric glands, the gorged and softened spleen, mark the extension of the morbid action. In short, to whatever organ in whatever structure the disease extends, the functional derangement and the structural lesion show alike that the resistance of healthy action has yielded to this extension of the disintegrating process.

CHAPTER III.

VARIOLA — MORBILLI — SCARLATINA.

SMALL-POX—VARIOLA.

Structure primarily affected—Cutis.

WHEN small-pox occurs as a consequence of contagion, the initiatory fever usually commences about twelve or fourteen days after such contact, and in no way differs from the preliminary fever that ushers in the other varieties of zymotic disease. There are the usual dulness, depression, feeling of weakness, sense of illness, loss of appetite, at times nausea and disordered bowels, very frequently pains in the limbs, and almost invariably a distressing aching in the loins; the pulse is more rapid; the temperature higher than in health. After these symptoms have lasted for about forty-eight hours the characteristic eruption commences with the appearance of small papules about the face and wrists; the eruption gradually extends over the neck, chest, and arms, and some hours later appears on the abdomen, back, and lower extremities. The face and wrists are almost invariably the parts where the

eruption first appears. When the eruption has come out the feverish symptoms generally subside. For the first twenty-four or forty-eight hours the eruption consists of a number (varying greatly, from a few scattered papules, to a number so great that the entire surface is almost covered by them) of small reddish papules about the size of a coriander-seed sensibly raised above the surface. On the second or third day the papules become vesicular. These vesicles are at first translucent. The centre of each vesicle is depressed as if bound down to the subjacent structure, giving the appearance that has been termed umbilicated. These vesicles gradually lose their translucent appearance and appear opaque. About the fourth or fifth day each vesicle is surrounded by a red areola. Gradually, as the vesicle ripens, it becomes more pustular in appearance and more distended; the central depression disappears; there is general tumefaction of the skin; the features appear swollen; the eyes are closed up by the tumid lids, and those parts of the skin that are not covered with pustules appear intensely congested. About the eighth day the eruption appears to be at its height, and the feverish symptoms that had subsided from the first outbreak of the eruption reappear, forming what is known as the secondary fever of small-pox. About the eighth day the cuticle gives way, and the contents of the pustule partially ooze out and dry upon the surface, forming

a scab over the pustule, which gradually shrinks, and at last dries, when the desiccated scab drops off, leaving the part of the skin from which it has dropped stained with a dark red spot that only disappears after some weeks. The period of desiccation and scabbing generally lasts from the eighth or ninth to the eleventh or thirteenth day. When the scabs have fallen off the disease may be considered at an end. The secondary fever gradually subsides in accordance with the decline of the local symptoms.

The above is the usual course of an ordinary attack of the disease, but the disease may be milder or more aggravated, forming those varieties that are termed respectively the horn-pock and the confluent.

In the horn-pock (the milder form) the vesicle do not proceed to the pustular stage, but begin to shrink and dry up after the sixth day; the secondary fever does not occur, and the disease usually runs its course within ten days from the first appearance of papules.

The confluent small-pox is much more severe; the number of papules is so great that the skin is entirely covered with them, and as they mature they coalesce, forming large pustulous blebs, especially on the face; the deeper skin structures and the subjacent cellular tissue take on the inflammatory action; the pustules are not surrounded with an areola, but appear flat and white; the eyelids are

much swollen, pustules form on the conjunctiva, and sometimes the cornea is rendered opaque, or the organ entirely destroyed. The parotid glands inflame, and salivation occurs. The secondary fever is much aggravated, and may assume all the appearance of an attack of typhus with the brown tongue, the teeth covered with sordes, the violent delirium and coma.* In this form of small-pox the action is extended to the mucous membrane and the eruption extends to the mouth, nose, pharynx, and trachea. It is after this form of small-pox that patients exhibit in their scarred and pitted faces the local ravages of the disease.

Inoculation.

The history of inoculation has been so often written that it would be purposeless to reproduce it here.

The Chinese and Brahmins claim a high antiquity for the practice, and we know that it had been long practised in Persia, Georgia, and Greece, whence it was introduced into Turkey at the commencement of the eighteenth century, and the story has been often told of its introduction into this country by Lady Mary Wortley Montagu in 1721.

The disease induced by this process in the skin

* One of the most frequent of the complications that arise during the secondary fever is pleuritis; this sometimes sets in with great rapidity and violence, and passes quickly on to empyema.

of persons in a state of health generally followed a very mild course, much resembling what has been described as the horn-pock.

Much speculation has been excited as to the cause of the mildness of the attack of small-pox induced by inoculation compared with disease excited by contagion. That such is the fact is borne out by abundance of evidence. Dr. Watson says of the cases inoculated in the London Foundling Hospital, "Of the seventy-four persons whose histories I have related, though inoculated with variolous matter in different states, though prepared in so different a manner, and a great many not otherwise prepared than by an absence from animal food, not one of them was disordered enough during the whole progress to occasion the least anxiety for the event. None continued in bed an hour longer than they would have been in their best health." This account is quite borne out by the observations of other inoculators.

It is noted also that whereas in the natural small-pox the initiatory symptoms commence twelve or fourteen days after exposure to contagion, the symptoms are rarely delayed beyond the ninth day after inoculation.

That there is no essential difference in the nature of the disease is proved by the fact that a person contracting small-pox from contact with an inoculated case may have the disease in the most violent form.

Vaccination.

The practice of inoculation spread rapidly through Europe, extended to America, and was very generally in vogue until superseded by the promulgation in 1798 of the brilliant discovery that has immortalized the name of Dr. Edward Jenner.

It would be difficult to over-estimate the enormous benefit that the world has derived from the discovery of Jenner that by anticipating the action of small-pox by the incomparably milder action induced by vaccine lymph, the immunity obtained is as complete as by an attack of actual small-pox.

Post-mortem Appearances.

The post-mortem appearances found after death from small-pox are confined to the skin and to those portions of mucous membrane that are in direct contact with the external air—viz., the lining of the mouth, pharynx, and trachea ; these parts are sometimes found after death softened, congested, and covered with inflammatory exudation.

Such internal parts as may have taken on inflammatory action exhibit evidence of such change ; the pleura, for instance, may be filled with serum and covered with recent lymph, but as a rule the viscera are found free from every appearance of special disease.

Dr. Gregory states that amongst the many post-mortems made at the Small-pox Hospital, in no case had the disease extended to the intestinal mucous membrane.

In small-pox we see the effect of zymotic action set up in the skin, and producing those phenomena that necessarily result from such action. The skin inflames and passes through the various stages of inflammatory disintegration: first hypertrophy with the redness, heat, and swelling pathognomonic of inflammation; next inflammatory exudation marked by the vesicular stage, and finally, suppuration. If the purulent contents of one of the pustules be examined under the microscope it exhibits not any product peculiar to small-pox, but what we should naturally expect to find as the result of disintegrated dermic tissue.

It is probable that the true explanation of the mildness of the disease produced by inoculation is that the disease is induced in those persons whose natural healthy resistance to diseased action would have successfully warded off the consequences of mere casual contact. That by inoculation the long-continued contact of molecules in an active state of disintegration forces the healthy skin to take on the diseased action, and that such action is modified by the healthy resistance.

In vaccine lymph it is probable that the disintegrating action is much less intense than in variola,

and that consequently the imparted action partakes of this mildness, although it is sufficient to secure immunity from a renewed attack.

We must remember that Jenner always declared his belief that variola and vaccinia were identical.

MEASLES—MORBILLI.

*Structure primarily affected—Mucous Membrane
of the Respiratory Tract.*

This disease commences with a mild form of pyrexia, quickened pulse, lassitude, and headache. After a few days the mucous membrane of the eyes, nose, mouth, and bronchia become inflamed; there are intolerance of light, frontal headache, sneezing, and dry cough. This condition is speedily followed by a profuse serous discharge from the mucous membrane of the eyes and nose. After these symptoms have continued for three or four days the disease extends to the skin, and an eruption characteristic of this disease is perceived commencing on the face, generally on the forehead, and gradually extending to the chest and arms, then covering the abdomen and back, and about three days after its first appearance extending to the lower extremities. The eruption consists of small dark red spots, aggregated in crescentic-shaped patches, slightly elevated above the surface, and momentarily removable by pressure. If the mucous lining of the cheeks and fauces be carefully examined when the rash first appears, it will be found to be also covered

with small red spots. The feverish symptoms that had preceded the eruption do not disappear when the rash comes out. The cough also continues; the eyes are suffused; the face looks puffed. About the fifth day the eruption begins to fade away from the face, and gradually disappears; first from the face, then from the chest and arms, and lastly from the lower extremities; so that about the seventh day from the first appearance of eruption the disease, in favourable cases, is at an end. The feverish symptoms and cough cease with the eruption. In severe cases the disease extends sometimes to the pleura, and even to the substance of the lungs, and the mortality in measles is principally made up of those cases in which such complication has occurred.

Post-mortem Appearances.

There is no appearance found after death from measles that is peculiar to that disease. When the action has extended to the pleura, or lungs, the pleuritic effusion, the consolidated or disorganized lung tissue, differ in no respect from the changes observed when those parts are affected by inflammation from other causes.

In measles the destructive action attacks the mucous membrane of the eyes, nose, and mouth. The intolerance of light and the sneezing mark the first stage of inflammation, speedily followed by the greatly increased secretion; the membrane is red,

hot, and swollen; the inflammatory action extends by continuity of texture to the skin, first to that part of the skin that is nearest to the inflamed mucous membrane—the forehead and face—then gradually extends to the arms and trunk, and lastly to the lower extremities. The destructive action not being intense, is gradually overcome by the resistance of healthy action, and the natural condition of the affected parts is re-established.

In those cases that have been followed by a fatal result, the dead-house does not reveal any product special to this disease. Where the inflammatory action has extended to vital organs, those organs exhibit the usual results of destructive inflammatory action. In some rare cases after the disappearance of the eruption, the mucous lining of the mouth and nares is destroyed by a sort of gangrene that spreads to the subjacent structures, and causes death. The author met with a case where the entire face, from the frontal ridge to the lower jaw, was quite destroyed by this process.

SCARLATINA.

Structures primarily affected—Epithelial Layers of the Skin and of the Mucous Membrane of the Mouth and Throat.

Willan, speaking of scarlatina, says: "It is truly singular that the slightest of all eruptive fevers, and the most violent, the most fatal disease known in this country, should rank together, and spring from the same origin." To describe in detail all the varieties of scarlatina, from the mild attack that scarcely deserves the name of illness, where slight discomfort and evanescent rash are the only symptoms, to the terrible disease that kills as certainly and almost as rapidly as cholera, and those other severe forms in which the throat mischief becomes the prominent feature, and inflammation proceeds to mortification—to describe all the varieties of the disease that lie between these extremes, and to trace the possible sequelæ, the anasarca, the ascites, &c., would require a volume. We must be content to trace here the usual course of an ordinary attack of the disease.

Scarlatina commences with a sharp feverish attack, shivering, and a violent attack of vomiting is frequently



amongst the earliest symptoms ; thirst, considerable heat of skin (the thermometer marking 104-106) ; rapid pulse, headache, restlessness, loss of appetite. After these symptoms have continued twenty-four to forty-eight hours a rash appears upon the skin, consisting of extremely minute bright red points, disappearing momentarily on pressure, closely aggregated, and spreading rapidly over the trunk, so that after a few hours the chest, abdomen, and back are quite covered. On the second day the eruption spreads to the arms and legs. The fever does not remit upon the appearance of the rash. If the interior of the mouth and the fauces be examined they will be found, like the skin, covered with small red points, that soon coalesce and make the mouth and throat appear of a vivid red ; the tonsils are enlarged, and are frequently spotted with lymph ; the centre and back of the tongue are generally covered with a white fur, through which the enlarged bright red papillæ project ; the point and sides of the tongue are bright red, with projecting papillæ ; the conjunctivæ are injected, but there is no intolerance of light, nor profuse lachrymation. If the attack be severe there is an acrid muco-purulent discharge from the nares ; the breath is rendered fœtid by the inflamed throat and sloughy tonsils ; deglutition is difficult, and the attempt to swallow causes liquids to be violently rejected through the mouth and nose. The temperature continues very high and the pulse

very rapid throughout the attack ; there is great restlessness and frequently delirium.

After the fourth day the eruption begins to fade away, disappearing at first from those parts that were earliest affected, but even after the complete disappearance of the rash and of the sore-throat symptoms, the pulse remains abnormally quick.

The disappearance of the rash is followed by a process of desquamation, and the patient cannot be considered well until the conclusion of this process, which sometimes occupies a considerable time. During the period of desquamation it not uncommonly happens that the cervical, maxillary, and parotid glands swell, inflame, and suppurate, causing large abscesses whose slow progress much retards recovery.*

There are four different periods in scarlatina, at any one of which death may occur.

Ist. The disease may make its attack with such violence that there is no reaction from the original

* Another complication that frequently occurs in scarlatina is painful swelling of the larger joints, the wrists, elbows, and knees, the suffering caused by these local inflammations is very severe, much like the pain in acute rheumatism, and occasionally the inflammation proceeds to suppuration.

Dr. Kirkes points out that the fluid in the serous cavities in a state of health is due to a process of transudation of the fluid portion of the blood ; but that in the cell of the arachnoid and the synovial cavities of joints it is rather owing to a process of actual secretion due to the action of epithelial cells.

depressing effect. The patient passes rapidly into a state of collapse, and dies before the characteristic symptoms are developed. This occurs frequently in the case of infants at the breast, also to parturient women, and some cases generally happen at the commencement of every epidemic.

2nd. At about the fourth or fifth day the patients may become suddenly worse. They become delirious, then comatose—the pulse from being quick becomes tremulous, irregular, and indistinct; the surface cools; the fæces and urine are passed unconsciously, and death takes place on the sixth or seventh day of the disease.

3rd. The local affection in the throat sometimes becomes much aggravated. About the eighth or ninth day of the disease the ulcerations become deep and sloughy; mortification spreads rapidly, and the patient sinks exhausted about the tenth or twelfth day.

4th. About the twenty-second or twenty-third day general anasarca may set in, commencing in the face, and soon spreading over the whole body, and the dropsy may invade the pleural and peritoneal cavities, and cause death in the fourth week of the disease. In those cases in which dropsy occurs, the urine is often smoke-coloured, and when examined it is found that the discoloration is caused by blood: it is also of light sp. gr., 1011 to 1015, and contains much albumen.

Post-mortem Appearances.

When death takes place during the first stage of the disease no structural change can be detected on dissection.

When death has taken place during the acute stage on the fifth or sixth day, or later, the arachnoid membrane frequently is found injected, and with serous effusion underneath; the mucous membrane of the mouth and fauces exhibits traces of the various stages of inflammation, from the severe darkness of sanguineous congestion to actual mortification; in the chest the bronchial lining is found congested, and the lung tissue is often observed to be softened, and showing a tendency to rapid decomposition; in the abdomen the post-mortem appearances are not generally of a marked character.*

The extreme gravity and acuteness of the symptoms that occur when the destructive action attacks the epithelium will be more easily understood when we consider the great extent of the structure and the rapid molecular change that is constantly going

* When the throat affection has caused death, there are not only extensive ulceration and mortification of the structures about the fauces, but the mucous membrane of the nares is found softened and inflamed, and pus is not unfrequently found upon the lining of the larynx and trachea. Dr. Tweedie has observed that the skin shows an unusual tendency to rapid putrefaction.

on in it; covering the entire body, lining the great cavities, continued along the canals and ducts, in immediate contact with the peripheral termination of the nerves—this enormous extent of cell structure is constantly going through a more rapid action of growth and of decay than any other portions of the body.

In those cases of scarlatina where fatal collapse occurs at the first onset of the disease, we see a condition precisely analogous to those cases so often met with of burns where the danger is measured by the extent of surface affected. Liquid not hot enough to vesicate, spilled over a child's chest and abdomen will not unfrequently cause death within twenty-four hours.

In the acute stage of scarlatina, the extreme rapidity of the circulation, 130–150, and the very high temperature, 106–108, attest the great activity of the molecular change that is going on, and the throat affection, with its heat, redness, swelling, lymph exudation, ulceration, and mortification, proves that the change is a process of inflammatory disintegration.

Dr. Tweedie tells us that the skin putrefies more quickly than usual, and Franz Simon found that the turbid, smoky-coloured urine of the second week owes its turbid appearance to the presence of an enormous quantity of epithelial scales. The symptoms during life, the appearances after death, tell the tale of a rapid and violent process of destructive

inflammation. The traces of this inflammatory process are co-extensive with the presence of the diseased structure : the skin, the fauces, the kidneys, the joints, wherever epithelial structure prevails, there are to be found the consequences of this disease.

CHAPTER IV.

PUERPERAL FEVER—ERYSIPELAS— DISSECTION WOUNDS.

PUERPERAL FEVER.

*Structure primarily affected—Lining Membrane
of the Uterus.*

PUERPERAL fever usually commences about the third, fourth, or fifth day after delivery. The patient may have been going on favourably until this period, when it is noticed that she is not quite so well. There is a slight rigor and some tenderness on pressure over the region of the uterus. The nurse notices that the lochial discharge has become scanty, and perhaps offensive, and that the secretion of milk has stopped. Sometimes there are nausea and vomiting. As the disease advances the pulse becomes very rapid, 110–120; the temperature rises. There are hot skin, headache, and thirst. The patient seems to move in bed with pain and difficulty. The expression of the countenance changes to a look of great anxiety; the brows knit, and the upper lip retracted and drawn tightly over the gums. On the second or third day the tongue becomes brown and dry.

The pulse rises to 130-40. The abdominal tenderness has increased to acute pain, so that the slightest pressure is unendurable; the belly is tympanitic; the bowels lose their contractile power, and cause the abdomen to look enormously enlarged with the skin stretched tightly over it; the breathing is rapid and laboured, and frequently there is delirium; the secretion of milk having stopped, the breasts are flaccid; there is no lochial discharge. After this condition has continued a few days there is either a gradual amendment marked by diminution in the frequency of the pulse, abatement of pain and lessened abdominal tension, or symptoms of sinking come on. The pulse gets weaker and more rapid; the breathing more oppressed; there is great restlessness, tossing about of the arms and muttering delirium. As death approaches the patient may perhaps declare that the pain is gone and that she is better, but the unmistakable signs of approaching collapse show how fallacious is this hope. Sinking sometimes comes on with startling suddenness; in other cases the symptoms gradually increase in violence, and death may take place on the fourth or fifth day of the disease.

Post-mortem Appearances.

The post-mortem evidence of death from puerperal fever is generally confined to the contents of the pelvis and abdomen.

The internal lining of the uterus is congested, softened, and sometimes sloughy; the substance of the uterus is sometimes so soft that the finger can be easily thrust through it, and abscesses are at times found in its walls.

The peritoneal covering shows all the signs of acute inflammation, and there is abundant evidence that this inflammation has spread to those portions of the membrane that cover the bowels and line the abdomen.

The bowels are glued together and to the uterus by recently effused lymph, and flakes of lymph are floating in the serum that has been effused into the abdominal cavity.

The history of the symptoms during life and the lesions met with after death in cases of puerperal fever, indicate so unmistakably the nature of the destructive agent that comment is scarcely necessary. The exceptional condition of the uterus after recent delivery makes it specially prone to septic influence, and the anatomical conformation of the parts points out how the morbid disintegrating action once set up spreads by continuity of tissue to the parts affected. The symptoms plainly indicate the rapid spread of destructive inflammation, and the morbid anatomy exhibits its consequences.

ERYSIPELAS.

Tissue primarily affected—Subcutaneous Cellular Tissue.

The term Erysipelas is sometimes erroneously applied to any diffused erythematous blush upon the skin excited by external causes, such as stings, burns, scalds, friction, or the introduction of indigestible food, such as pork, shell-fish, &c., into the stomach. These cases should, however, be eliminated, and the term confined to cases of idiopathic erysipelas, such as we see occur most commonly in the head and face, and sometimes, though much more rarely, on the trunk or limbs, and the traumatic erysipelas that occurs in any part of the body that has been subjected to injury by accident, operation, or otherwise.

An attack of erysipelas is generally, but not invariably, preceded by some constitutional symptoms: languor, chilliness, hot, dry skin, thirst, and furred tongue; the bowels are generally constipated; when acted on, the motions are dark coloured and offensive; the urine is high coloured and scanty; quick pulse; there are headache and a feeling of oppression about the head, sometimes drowsiness. (Frank has pointed out that when erysipelas is about to attack

the head, there are invariably pain, tenderness, and swelling of the lymphatic glands of the neck.) After these symptoms have continued for a period varying from some hours to a few days, the local symptoms manifest themselves. A portion of skin, at first perhaps of limited extent—an eyelid or patch of skin of the cheek—becomes red, tense, shining, and swollen. The swelling and redness do not gradually merge into the healthy texture, but there is a defined limit; the redness disappears momentarily on pressure; the skin feels thickened and brawny. After some days bullæ may appear on the affected part, filled with a straw-coloured serum. The swelling and redness rapidly extend, and if the head and face be the part affected, the deformity caused by the swelling is extreme; the eyes are closed by the enormously puffed up lids; the nose, lips, and other features partake of the swelling. After some days, the pain, at first tingling, becomes burning and smarting. If the attack be severe, there is probably delirium. After three or four days the redness of the parts first affected becomes less vivid, whilst the disease is perhaps still spreading in other parts. The tumefaction gradually subsides, and generally after eight or ten days, in favourable cases, the disease terminates by a shrinking and subsequent desquamation of the skin.

That severe form of the disease that is recognised by the term phlegmonous erysipelas usually follows

upon the receipt of some local injury. The general constitutional symptoms that usher it in are the same as those that have been marked as preceding the milder form, but are more intense; the local signs are, that recently closed wounds re-open, granulations become flabby and unhealthy looking, the edges of wounds look glazed, and cease to produce healthy pus. There is a red blush that gradually extends and becomes of a dusky hue; the skin is swollen and pits on pressure; soon it becomes hard and tense; the redness changes to a livid violet hue, and in some cases large portions of skin slough; or if this be anticipated by incisions, the cellular tissue beneath will be found in a state of suppuration and often sloughy. This condition of the cellular tissue has no distinct boundary, and the destructive process may extend, large portions of skin and subjacent cellular tissue becoming gangrenous, until an entire limb or a large portion of the trunk is involved in the disease. The general symptoms sympathize of course with the local affection, and in severe cases the patient sinks into a typhoid condition, with feeble fluttering pulse, dry brown tongue, delirium, and finally subsultus and total insensibility.

Post-mortem Appearances.

Mr. Brook says that in fatal cases of erysipelas of the head, the smaller veins of the head are al-

ways found to contain pus: there are generally thickening and opacity of the arachnoid, and sometimes effusion.

Dr. C. J. B. Williams says he has detected pus in the blood of patients who have died of erysipelas.

Mr. De Morgan says that the blood is sometimes black and pitchy, depositing a blackish powder and staining the inner surface of the heart and great vessels, and that the corpuscles are much altered, broken up, and irregular in shape.

Mr. Brook has stated that the lungs are always much congested, and that the smaller pulmonary vessels always contain pus.

The liver, spleen, and kidneys are generally found congested, and their structure softened.

Sometimes there is congestion of the mucous lining of the stomach and intestines.

The changes observed in the skin and cellular tissue are thus described by Mr. De Morgan:—

“The skin, where it has remained untouched by ulceration or mortification, loses after death its red colour and appears shrivelled and brown. Its vascular tissue will be found congested, the veins especially being filled with dark black blood. Pus and serum will be found in its areolæ. The cuticle readily peels off.

“The condition of the cellular tissue may often be seen in all its stages in fatal cases; for as the disease is progressive, some parts will be found in

which only the earlier changes have taken place, while in others the destruction is complete at first. The tissue is vascular and congested, and its areolæ contain serum, which is occasionally tinged with blood. The serum becomes thicker and is replaced by a semi-fluid, whitish or often deep yellow matter. This is still lodged in the areolæ, and does not flow from them when cut into. By-and-by true pus appears, which though still lodged in the cellules of the tissue, escapes when they are laid open. In the last stages the areolar tissue is found broken down, the pus is diffused in layers, and contains the fragments and shreds of cellular membrane."

The swelling and redness of the integument and the fact of any erythematous blush being called erysipelas have led to erysipelas being considered as a skin disease; but the facts that the subcutaneous cellular tissue is invariably affected, that such affection is more constant than the condition of skin, that the diseased condition is more persistent in the cellular membrane than in the skin, for the swelling often continues after the skin has returned to its normal state, make it probable that the cellular tissue is the structure primarily affected, and that the skin takes on the action secondarily. That this action is a process of inflammation is abundantly proved by the symptoms during life and the appearances on dissection. The redness, swelling, heat, and pain are evidenced more dis-

tinctly perhaps in this than in any other variety of zymotic disease; the general symptoms are such as invariably occur whenever active local disintegration is going on.

It has been shown that the changes found after death in the cellular tissue are such as would naturally be found as a consequence of destructive inflammation; that the same process has extended to the skin; and in severe cases the blood and the viscera exhibit the products due to a wide extension of the inflammatory process. In this disease we can distinctly trace the progress from simple congestion to actual death of structure.

DISSECTION WOUNDS.

Closely allied to phlegmonous erysipelas in symptoms and consequences, is the diffuse cellular inflammation caused by wounds received during dissection. In these cases, that most commonly occur from wounds received during the examination of bodies recently dead, the extremely depressing effects upon the general system, before the local consequences have had time to fully develop themselves, are very marked. It is not always necessary that there should be an actual breach of surface, as cases are on record where the effect has been produced without any observable wound. The following case, related by Dr. Williams, will give a general idea of the symptoms:—

“ Dr. Pett examined the body of a patient that had died of puerperal peritonitis at eight o'clock in the morning of Saturday, the 28th of December. On the evening of the same day he complained of feeling some heat and uneasiness on the outer side of the last phalanx of the middle finger of the hand, and suggested he might have pricked himself in the morning. Mr. Toulmin examined the finger cursorily but found no wound. There was, however, a slight blush upon it, and by a stronger light and

by the aid of a lens a minute opening was discovered in the cuticle and in the centre of the redness before observed, and this spot was touched with a pointed piece of nitrate of silver and subsequently with a very small quantity of nitric acid. In the course of the night Dr. Pett again touched the part with the nitrate of silver, and the pain produced, he said, increased to agony. He got into bed and was shortly after seized with violent shivering, followed by some degree of heat.

“About one o’clock,” says Mr. Toulmin, “I accompanied my father. On entering the room it was impossible not to be struck with his altered appearance. His countenance was rather suffused with redness, his eyes were hollow and ferrety, and there was a peculiarity in his breathing which never forsook him during his illness. It was a suddenness and irregularity in the act of breathing amounting almost to sighing. His manner, usually gay and playful, was now marked by excessive torpor. Indeed his whole appearance reminded me of a person who had taken an excessive dose of opium. His pulse was between ninety and a hundred, and rather soft. Dr. Pett described himself as having suffered intensely. He said he was completely knocked down, and that he had not the strength of a child.

“Upon removing the poultice, the finger and hand appeared more swollen than in the morning,

the skin of the last and middle phalanges was very tight and of a bluish livid appearance, with effusion under the cuticle surrounding the crust where the poultice had been applied. A lancet was passed freely into this and down to the bone; some bloody serum escaped, but it gave no pain. The last and middle phalanges were indeed quite gangrenous. The inflamed absorbents could be traced to the elbow, and there was no uneasiness in the axilla.

“On Tuesday morning we found things looking worse than before. Dr. Pett’s general appearance and powers were more sunk, the pulse was quick and more irregular, the breathing more oppressed, and that torpor of manner which was present at first was converted into restless anxiety. There was, however, no increase of inflammation or extension of gangrene on the hand, but the same kind of virulent inflammation which had existed in the finger seemed to be proceeding to the axilla and the parts adjacent. The skin over the axilla and side of the chest was marked by an erysipelalous blush and pitted on pressure. Dr. Pett died in the evening of the following day, or on the fifth from the examination of the body.”

There are two circumstances worthy of note in these cases—firstly, that they arise generally from wounds received in the examination of bodies that have recently died. In an advanced stage of putrefaction the danger is much less. Secondly, that

most of the recorded cases are of wounds received during the examination of bodies where death has been caused by an acute disease such as puerperal peritonitis.

The remarks that have been made on the post-mortem appearances observed in cases of phlegmonous erysipelas apply to cases where death has resulted as a consequence of dissection wounds. When sufficient time has elapsed for the local symptoms to develop and spread, the same evidence of structural disintegration in the various stages of inflammation, suppuration, and gangrene is to be found.

CHAPTER V.

PATHOLOGICAL SUMMARY.

WHEN we pass in review the pathology of these various forms of zymotic disease we cannot fail to be struck by the similarity of the changes that occur. Whatever may be the cause (and the etiology will be discussed in a future chapter), the disease being once set up, the effect, varying in accordance with the different structures primarily affected, resolves itself into a uniform process of structural irritation, structural inflammation, and structural death.

In typhus, where the blood is primarily affected, we can trace the disintegrating blood imparting its death action to all the various tissues until every organ and structure of the body becomes affected and every function disturbed. The post-mortem examination plainly records the intimate nature of the disease; the blood is seen to be in a state of disintegration, its corpuscles misshapen, partially dissolved, the muscles softened, blackened; the viscera have taken on the action and give evidence of structural change tending to structural death;

the most minute chemical and microscopical examination fails to discover any adventitious diseased product.

In cholera, where the disease appears to strike upon the nerves that preside over organic life, all vital functions seem to be at once arrested, and the patient dies, in the majority of cases, before the secondary effects can be developed.

Cases of enteric fever show the train of symptoms that ensues when zymotic action is set up in the glandular structure of the intestinal canal. We can trace the gradual extension of the diseased action by the functional disturbances that are exhibited during the course of the disease, and after death the dead-house tells us in language that it is impossible to misinterpret the nature of the diseased action. Glands in every stage of disintegration: mesenteric glands, reddened, swollen, gorged with blood; Peyer's patches showing all the various stages of destructive inflammation, from simple engorgement to ulcerative disintegration; and if we test the débris of the structures by the microscope we obtain additional evidence that structural death has ensued as a consequence of inflammatory disintegration.

If there be one form of zymotic disease that might appear to have established a claim to specificity it is small-pox. The constant and well-defined character of the eruption, the many cen-

turies over which its history extends, and the difficulty of tracing its occurrence otherwise than from contact with a person similarly affected, moreover, the undoubted prophylactic power of vaccination, might appear to mark it as a specific disease; but a close examination shows that this claim cannot be maintained. The phenomena exhibited by the skin differ in no manner from those we witness when the dermic covering is submitted to ordinary causes of inflammation proceeding to suppuration. The pustules that are observed in the vicinity of a dissection wound, the pustules consequent on irritation by croton oil or tartarized antimony, cannot be distinguished from the pustules of small-pox. If the contents be placed under the microscope, no observer could possibly distinguish between them, and the most minute chemical analysis has failed to detect any product peculiar to small-pox pustulation. Post-mortem examination proves that the diseased action is generally confined to the tegumentary envelope of the body, and when change can be traced beyond the skin it appears to be merely an extension of inflammation to contiguous structures.

The rapid molecular changes that we know to be constantly going on in epithelial structures are intensified in scarlatina, the destructive process far outbalancing the reparative, and the result is evidenced in the great functional disturbance

during life, and the structural destruction that can be traced after death. Both the functional disturbance and the structural changes can be clearly traced as consequent on an inflammatory process.

The exceptional condition of the generative organs in parturient women renders them specially prone to take on inflammatory action, and whatever septic influence they are submitted to, whether it be contact with a scarlatina patient, or contact with a hand recently engaged in post-mortem examination, or putrid lochial discharge, or unfavourable hygienic surroundings, that septic influence is translated into the symptoms of the fatal and much dreaded form of inflammation known as puerperal fever. The symptoms do not vary in accordance with the varying cause. The disease is ever identical, inflammation of the lining of the uterus extending by contiguity to the peritoneum and other structures.

It would be tiresome and needless iteration to proceed with this analysis into other varieties of zymotic action. The key-note is ever the same; the symptoms and appearances vary in accordance not with the producing cause, but with the structure primarily affected. The irritated structures respond to the irritating cause in the only language they can use—exaggerated or perverted function; and as the irritation increases to inflammation, and the inflammation becomes destructive, we find each

structure exhibiting the inflammatory phenomena peculiar to its kind: blood, cutis, epithelium, gland, as they pass through the process of destructive disintegration, give rise to those symptoms that we have been hitherto used to recognise as distinct forms of disease produced by distinct and special causes.

CHAPTER VI.

CONTAGION—IMMUNITY FROM FUTURE ATTACKS.

CONTAGION.

THE property that many of the forms of zymotic disease possess of communicating, by mediate or immediate contact, the same form of disease to individuals hitherto unaffected, has always excited much attention, and has had more influence than any other fact in inducing a widespread belief in the specificity of these forms of disease. Various hypotheses have been advanced as to the exact mode in which the diseases are communicated, and observers are even now diligently searching for some material and specific form of contagium.

This important subject will be best treated in detail under the section on Etiology.

IMMUNITY FROM FUTURE ATTACKS.

Another fact observed in many of the forms of zymotic disease, is the property they possess of exhausting future susceptibility in the constitution of the person affected to any similar action of the same disease. We must, however, bear in mind that the immunity is not absolute, that although second attacks of variola, rubeola, and scarlatina are rare, they do occasionally occur;* and we must further bear in mind that this property is not peculiar to some forms of zymotic disease, but is in accordance with a well-known physiological law.

A bunch of violets, after being held to the nose twice or thrice, ceases to convey the impression of sweet perfume. Persistence in the habit of tobacco smoking overcomes the nauseating effect of the herb. The system becomes so accustomed to opium that doses may, after a time, be taken with impunity that would previously have produced a poisonous effect.

Iodide of potassium is by some constitutions only tolerated after repeated doses. Purgative drugs have to be administered in constantly in-

* We frequently see sore-throat without eruption in persons submitted to the contagion of scarlatina, such persons having previously had the disease.

creasing doses to insure a continuance of their special action. The system becomes gradually able to bear very considerable quantities of arsenious acid.

Numerous authentic observations have been published that tend to show that, in certain cases, organs that have been induced, by artificial means, to take on an inflammatory action, are capable of offering a successful resistance to the contagion of disease. Thus Dr. Tweedie says,* speaking of the prophylactic action of belladonna in scarlatina:—
“From the result of trials made by Bernat, of Custrin, we find that in an epidemic of scarlatina which prevailed in Custrin in 1818–19 he employed the belladonna as a preservative in 195 children under fifteen years of age. Though they were freely and constantly exposed to the contagion of scarlatina, only fourteen took the disease, and that after employing a stronger preparation of the drug every individual escaped. Professor Koreff, of Berlin, after extensive and long continued trials, asserts that if the belladonna be taken in proper doses for eight or nine days before exposure, and be continued to the period of desquamation, there is little danger to be apprehended from free intercourse with persons affected with scarlatina. Hufeland, Kunzman, and others have given similar testimony.”†

* Cyclop. Prac. Med., Art. “Scarlatina.”

† Many physicians doubt the fact of the prophylactic power of belladonna on scarlatina, and recent experiments appear not to have altogether fulfilled the hopes of the early advocates of its use.

It is well known that belladonna given in small doses produces heat and dryness in the throat, swelling of the submaxillary glands, and sometimes a rash upon the skin.

Dr. Clapton, in a paper read before the Clinical Society, Oct. 8th, 1869, "On the Effects of Copper upon the System," said:—"One very remarkable circumstance, of which I was first informed by Benham and Froud, Chandos Street, was mentioned at each of the works—viz., the absolute freedom of the workmen from cholera or even choleraic diarrhœa. During each of the great cholera outbreaks there were terrible ravages in one or other of their neighbourhoods, but not one of these men was in the slightest degree affected."

As examples of immunity induced by repeated contact with decaying animal matter we may cite the following. Dr. Tweedie says that butchers enjoy a special immunity from typhus, and Dr. Murchison writes that this is probably correct.

Sir James Paget, in the interesting account of his recent illness, dwells especially on the fact that during the time he was constantly exposed to the contact of dead animal matter he enjoyed immunity from ill effects.*

* Short, in his description of the Black Death in England in 1348, says, "Tanners, curriers, such as cleansed bog houses, servants in hospitals, and others employed in other nasty, stinking businesses, all escaped infection."

During the violent epidemic of cholera that raged with especial

Dr. Walshe relates, in the *Lancet* of March 4th, 1871, the following very remarkable instance of special immunity:—“Mr. Ambrose Blacklock, Surgeon-major of the Madras Army, believes ‘that persons who have had well-marked cow-pox at no distant date, say within five years, are fully protected from Asiatic cholera, as well as from small-pox.’ Again he says that after twenty-three years’ residence in India, ‘where vaccination makes but slow progress among the people, and pock-pitted persons are to be seen in abundance, and where I have had a large number of cholera cases to attend to from year to year, I cannot remember ever having seen or heard of any person marked by pits of small-pox being affected with Asiatic cholera.’”

It is to this physiological law we must refer for an explanation of those anomalous facts in surgery that have repeatedly excited the observation of those who have seen much of hospital practice, but which have not been hitherto explained.

What hospital surgeon can have failed to notice the favourable results of amputation performed upon persons who have been the subjects of chronic disease of joints, compared with amputations performed in consequence of recent injury?

We know the grave consequences that are feared

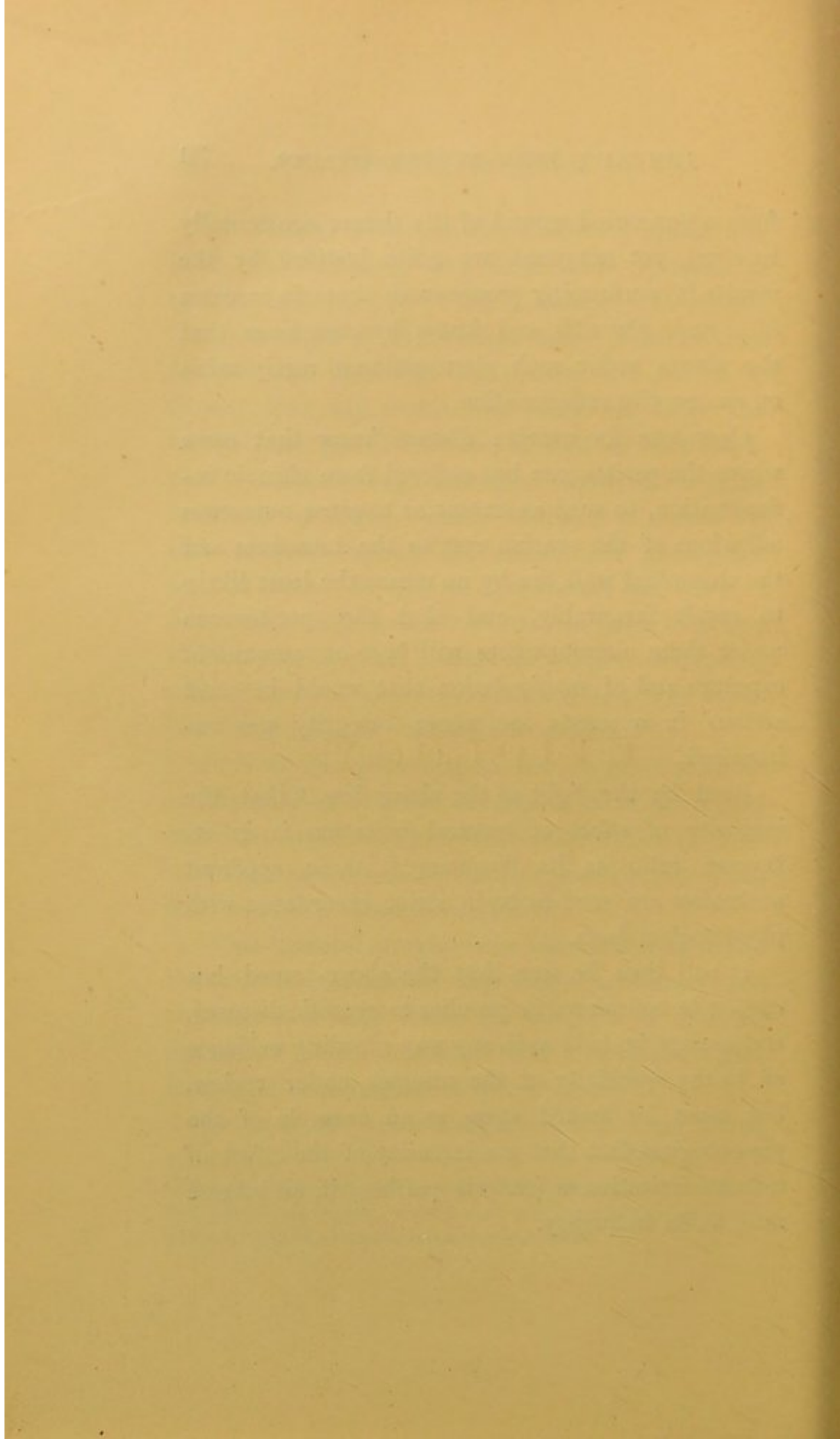
violence in Shoreditch and its immediate neighbourhood, the workmen, sifters and others, employed by two large firms of dust contractors, enjoyed a singular immunity.—AUTHOR.

from a punctured wound of the thorax accidentally received, yet surgeons are quite justified by the results in performing paracentesis thoracis in cases of chronic pleuritis, and know by experience that the pleura under such circumstances rarely takes on destructive inflammation.

Operators for ovarian disease know that cases where the peritoneum has suffered from chronic inflammation, to such an extent as to cause numerous adhesions of the ovarian cyst to the intestines and the abdominal wall, are by no means the least likely to result favourably, and that the peritoneum under these circumstances will bear an amount of exposure and of manipulation that would be most serious in a serous sac whose integrity was unimpaired.

Read by the light of the above law, "that the intensity of effect of textural irritation is in an inverse ratio as its frequency," these apparent anomalies are seen to be in strict accordance with physiological facts.

It will thus be seen that the above-named law cannot be considered as peculiar to zymotic diseases, and cannot be held as in any way affording evidence as to the specificity of the diseases under review, but must be looked upon as an example of the physiological fact that the intensity of the effect of textural irritation is (*cæteris paribus*) in an inverse ratio as its frequency.



SECTION II.

ETIOLOGY.

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CHAPTER I.

THEORIES OF CAUSATION.

Morbid Poisons—Zymotic Theory—Germ Theory.

THE opinions that have been held respecting the nature of zymotic disease have largely influenced the various hypotheses that have been brought forward to explain their causation. Physicians, seeing the great variety of phenomena that the different forms of zymotic disease present, have looked upon each form as a distinct and special disease; they have noted that certain symptoms are common to all, and that they have a degree of affinity in their external causation sufficient to entitle them to be considered a distinct class of diseases, but the great majority of writers have advocated and taught the doctrine of specificity, considering each disease as a distinct entity, having a separate and specific cause.

Dr. Williams, the learned and accomplished author of *Elements of Medicine*, 1836, enunciated the prevailing doctrine current in his day, that the

remote causes of the different forms of zymotic disease were certain morbid poisons always present in the atmosphere, and attacking persons who, from weakness or other unexplained cause, were specially predisposed to be affected by such poisons. The persons so affected communicating the disease to others with whom they might be brought into contact, such contact being either mediate or through the medium of the atmosphere or of fomites. Dr. Williams explains the laws that govern these poisons, and points out where those laws agree with, and where they differ from, those regulating the action of other poisons. He does not, however, attempt to explain the actual nature of these so-called morbid poisons, nor to account in any manner for their origin.

The zymotic or fermentation theory carries the explanation a step further than had been attempted by Dr. Williams, and whilst accepting the hypothesis of morbid poisons, undertakes to explain the manner in which these poisons act when they have gained access to the body. It is supposed, according to this theory, that a particle, however small, of the poison, whether that particle be in the atmosphere or derived from a person already affected with the disease, having contact with a healthy but predisposed person, acts as a ferment to certain textural waste products that are lingering in the body, and transforms them into the likeness of itself.

According to this theory therefore, not only is the atmosphere laden with a large variety of morbid poisons whose nature is unexplained, and whose origin is unaccounted for, entities that elude our senses, that defy the inquisition of our test-tubes and of our microscopes, but we must take for granted the special affinity that these atoms have for certain loosely defined portions of our frame, and are to suppose that there are certain materials necessary to be eliminated from our bodies, and waiting for such extrusion until they chance to come into contact with one of these morbid poisons. Moreover, when these morbid poisons have, haply and opportunely, had access to the body, and have effected their purpose (not seldom at the sacrifice of life), when measles and scarlatina, and small-pox and erysipelas, perhaps cholera and typhus fever, have (each in its turn) exercised their salutary purgation—the body so freed, differs in no appreciable manner from another body that has enjoyed no such advantage.

The most widely prevailing doctrine of the present day respecting the origin and communication of zymotic disease is that known as the germ theory.

Certain industrious and distinguished observers think that they have discovered the actual form that morbid poisons assume, that they are germs, living organisms, with enormous power of repro-

duction under certain favouring circumstances, capable of development into new forms within a certain limit, and indestructible by ordinary processes of nature. They hold that these germs having access to the body, excite, under certain favouring conditions, changes in such body of a fermentative or putrefactive nature, that the germs are the poisons, and that the diseases are the results of the changes induced in the body by such poisons.

The advocates of this theory have not explained the source whence these germs originate. Whilst noticing that they are in some manner connected with putrefactive change, they have not clearly made out whether they are the cause or the consequence of putrefaction. The most learned and industrious observers differ as to the facts. Thus Dr. Sanderson in the appendix to the last report of the Medical Officer of the Privy Council, says, "It will be shown that so long as the germinal matter of microzymes is excluded, animal fluids or tissues withstand decomposition for very long periods, while the slightest contact with media containing this material at once determines septic changes." On the other hand, Professor Hoppe-Seyler "treated the serum of pus, filtered and perfectly clear, and hydrocele fluid in various ways, some of the experimental tubes being hermetically sealed with almost entire exclusion of air, others

being loosely corked, and they were exposed to varying temperatures up to 58° C. for various periods of time. The results show that the fluids underwent putrefaction, whether living organisms were present or not, and that they exercised apparently no influence on the rapidity of the process, but that this was solely dependent on temperature. Two similar fluids were kept, the one loosely corked at a temperature of 68° F., the other in an hermetically sealed tube at 108° F., for equal lengths of time. At the end of the experiment the former, which swarmed with monads and vibriones, had undergone much less putrefaction than the latter, in which no sign of life or organization could be detected."—*Med. Chem. Unters.*, 1871, pp. 557–581.

Professor Hallier states that micrococcus is constantly observed and very abundant in the blood of patients affected with scarlatina, at all stages of the disease, whilst Dr. Sanderson says that according to his researches no such bodies exist in it. There is a diversity of opinion whether these germs are to be sought for on the confines of the animal or of the vegetable kingdom, for some contend that they are the germs of minute fungi floating in the air, others endeavour to prove that they are bacteria or microzymes, and that water is their habitat.

Whatever may be the part that these low forms

of organized matter perform in the process of decay, no plausible explanation has hitherto been offered for connecting their presence with the genesis of zymotic disease.* Their presence will account for no symptoms peculiar to these diseases. If always present in the atmosphere, why is the consequence exceptional; if ever present in water, why do any of us escape repeated attacks of zymotic disease in various forms? Are there several distinct species of them, each species producing its own special affection? If their immense multiplication within the body cause and constitute the disease, why does spontaneous cure occur in the majority of cases? Every fresh patient must be a focus of indefinite increase of these poisonous particles, and the disease once commenced should go on continually increasing in intensity and diffusion.

The Report of the Committee for Scientific Inquiries in relation to the Cholera Epidemic of 1854 (Spottiswoode, 1855), contains the following passage:—"With respect to the living animal and vegetable forms traced by Dr. Hassall through the whole series of waters, there seems no evidence that they, by their own action on the human body could be productive of choleraic symptoms. There are

* Their existence as accompaniments of putrefactive change is explicable quite apart from any possible connexion with the causation of diseases.

indeed many instances, human and brute, of disease engendered in the living body through the tenantry of parasitic organisms, animal and vegetable: and for aught we know to the contrary many of the creatures described by Dr. Hassall may be capable of sustenance and multiplication within the bowels of those who swallow them. But in every known case where it can fairly be presumed that parasites are the causes of disease, they exist as a palpable morbid product, occupying some considerable share of the affected body. The silkworm destroyed by muscardine dies because its whole body is riddled with parasitic vegetation, so dense that at last a mere heap of mould remains in place of the absorbed and disorganized animal, and every molecule of that mould makes evident the nature of the destructive process. What we know of parasitic disease in the human subject, of hydatids and porrigo for instance, tends all to the same point; in whatever way the foreign occupant have proved hurtful, whether it have starved the proper substance of the body on which it was grafted, or have provoked particular textures to acts of inflammation, or have choked their functions by pressure, itself the causative thing remains as a material shaped body, susceptible of ocular demonstration, side by side with its effects, and having bulk proportionate to them. Analogy therefore would lead us to infer that parasites could produce

no attack of exhaustive purging and vomiting except by having first along the digestive canal multiplied to such swarms that they would be obvious to the most casual observer both in the discharges of living patients and in the subsequent examination of the dead."

The argument so conclusively urged in the above passage respecting the causation of cholera applies with equal force to the other varieties of zymotic disease: there is not one of them in which the symptoms can be traced to, or accounted for by the presence of foreign living organisms, and in the dead-house, the fatal result is to be traced in the textural destruction that has taken place; the putrefactive changes that follow death differ in no manner from the ordinary process of decay after death from other than zymotic disease.

The chain of argument by which it is sought to prove that zymotic diseases are dependent on the presence of organic poisons, that there is a specific poison for every specific disease, and that such poisons consist of certain living organisms, animal or vegetable, is weak in every link.* It has been by no means proved that these diseases are caused by

* "The germ-theory is an assumption of causes of the existence of which we have no distinct proof, to account for effects which they do not explain."—*Beckington*.

poisons in the strict sense of the term. There is strong reason for believing that their specificity has been much overstated; and moreover, there have hitherto been no distinct observations recorded that connect the occurrence of communicable diseases with the presence of foreign living organisms.

CHAPTER II.

MOLECULAR THEORY.

Physical Theory—Molecular Motion—Physical Law of Molecular Motion—Examples—Physical Law of Transmission of Molecular Motion—Examples—In Inorganic Bodies—In Organic Bodies—Application of the Law to the Causation of Zymotic Disease—Predisposition.

THE great physical laws that govern all forms of matter, organic and inorganic, have perhaps been scarcely sufficiently studied with reference to the bearing they may possibly have on diseased action; two of these laws deserve especially to be carefully considered.

1st. That the molecules of all bodies can, under the influence of some form of force, be made to move upon one another, and that by such alteration of arrangement of molecules, the appearance and functions of a body may be altered.

2nd. That a molecule set in motion by any

force can impart its own motion to another molecule with which it may be in contact.

Examples of the first law abound in Physics and Chemistry—*e.g.*,

The change of bulk under the influence of heat.

The facts of crystallization and of solution.

The change that occurs when phosphorus is heated to 240° C. in an atmosphere incapable of acting upon it, is a striking illustration of this law; from a yellow waxy substance readily inflammable at a temperature of 46° C., readily soluble in carbonic disulphide, it is converted into a hard red substance which may be heated to 260° C. without inflaming, and which is perfectly insoluble in carbonic disulphide.

This change can be explained only by the supposition of a fresh arrangement of molecules; nothing has been added or subtracted. The weight of phosphorus is the same, and when burned it yields the same weight of phosphorous anhydride as would have been obtained from the yellow phosphorus employed.

Another striking example is the change that is produced when melted sulphur is thrown into cold water.

The changes of colour produced in certain substances under the influence of heat also illustrate this law.

The most familiar example of the second law may be found in the transmission of the molecular

motion of a heated body to the molecules of another body of a lower temperature with which it may be in contact.

The motion imparted successively to the molecules of a solid body, and by them transmitted to the molecules of the atmosphere, is well known as the cause of sound.

Water may be cooled down to several degrees below 32° F. without freezing, but if motion be communicated to some of the molecules, this motion is rapidly transmitted through the whole body, which becomes ice.

If fluid carbolic acid be even slightly touched with a solid body, the part so touched immediately crystallizes, and the change from the liquid to the solid form may be watched gradually spreading until the whole becomes a solid mass.

Platinum does not dissolve in nitric acid—it may even be boiled in nitric acid without change; but an alloy of platinum and silver dissolves rapidly in nitric acid; the motion in the molecules of silver is transmitted, through contact, to the molecules of platinum.

These physical laws of molecular motion and of the transmission of such motion to contiguous atoms is even more evident in organic than in inorganic bodies. Physiology presents us with abundant evidence of the active molecular motion that is constantly going on in all living bodies,

and the phenomena of growth and of decay show how the molecular movement is transmitted from molecule to molecule. The modern operation of transplantation of skin is a striking instance of the transmission of molecular motion. The living particles of skin transmit their action to the organized molecules with which they have contact, and induce in them a similar action. All observers are agreed that one of the most active agents in inducing and intensifying putrefactive action is the contact of matter already in a state of decay.

It is to the second of these natural laws that we must look for the real explanation of zymotic disease. We have seen in the preceding section that all the forms of zymotic disease are actions of destructive disintegration—actions tending to structural death, and it will be shown that in those cases where we can trace the cause of these diseases, that cause is the contact of decaying organic matter. Organic matter in a condition of decay has had contact with living structure; the healthy actions of growth and of repair are replaced by the diseased actions of structural disintegration. The disease manifests itself in the form dictated by the structure that has taken on the dying action. Typhus, Variola, Scarlatina, Enteric fever, develop their pathognomonic symptoms accordingly as the blood, the skin, the epithelial structures, or the bowels, are respectively the recipients of the morbid cause.

That animal matter in an active state of decay has the power of causing disease to be set up in a healthy body is beyond dispute.

“Sir B. Brodie shows that the application of various kinds of offal to wounds, and especially pricks of the finger with spiculæ of bone from the hare, may cause an obstinate chronic erysipelas of the hand.”—*London Medical and Physical Journal*, lvii. 342.

Gaspard injected into the inguinal abdominal cellular tissue of a dog about three ounces of water rendered putrid by steeping in it raw and tainted meat.* This animal exhibited signs of great pain during the injection, then refused all food, and soon became agitated, growled, and rolled himself on the ground. Some hours after this his abdomen seemed to give him pain when touched, a hard painful tumour formed in the groin, and the inflammation which extended on every side, terminated in gangrene.

M. Dupuy shows that the “pustules malignes” of cattle may be caused by applying to a wound the blood of an animal killed by gangrene of the lungs.—*Revue Médicale*, 1827, ii. 488.

“From the cases described by Morand, it follows that the flesh of overdriven animals is wholesome

* “M. Gaspard performed many similar experiments with analogous results.”—*Magendie, Journ. de Phys.*, vol. ii.

enough when cooked and eaten, but that if the blood or raw flesh be applied to a wound or scratch, nay, even to the unbroken skin, a dangerous and often fatal inflammation is excited, which at times differs little from diffuse cellular inflammation, and at other times consists of a general eruption of gangrenous boils, the 'pustules malignes' of the French."—*Christison on Poisons*, p. 631.

Accounts are also to be found of poisoning from bacon, sausages, and many other kinds of food when commencing to decay. — *Christison on Poisons*, p. 637 *et postea*.

"Some drops of putrid matter from a fish injected into the veins of a dog produce in less than an hour symptoms which have the greatest analogy to typhus and yellow fever. Death occurs usually in twenty-four hours, and on examining the body one finds all the traces of a chemical alteration of the blood."—*Magendie, Journal de Physiologie*, tom. iii.

"From the preceding facts it may be seen that putrefying liquids injected into the veins of animals cause death, or excite symptoms which have the greatest analogy with those of typhus or yellow fever; that the prolonged respiration of putrid miasms also causes death, but in a much longer time, and with symptoms that do not at all resemble those of the diseases I have named."—*Ibid.*, p. 87.

The peculiar form of zymotic disease caused by

dissection wounds, the "cellulitis venenata" of Williams, is another example. Here there can be no possibility of doubt as to the exciting cause; animal matter in a state of active molecular disintegration has contact with healthy structure, overcomes its resistance, and induces in it a process of textural disintegration.

Dr. Salisbury relates several cases where an eruptive disease, not to be distinguished from measles, was induced in several children from sleeping upon mouldy straw.

Dr. Kennedy, in Ireland, found that a disease exactly like measles was produced by blowing musty flax into a child's face.

"Mrs. ———, primipara, healthy, had a natural but rather quick delivery. On the third day she was attacked by rigors, succeeded by feverishness and delirium. The next day the symptoms increased in severity, the pulse reaching 130. She was wandering in mind and had a hurried tremulous manner. The lochia had ceased. She rapidly sank into the typhoid state, and died about the sixth day after delivery.

"This case occurred completely in the country, in a healthy spot. The medical attendant informed me that he had, on the day of her labour, and for a few days before, been in attendance on a man whose hand was sloughing, and which he had dressed twice each day, and that just before going to her

he had been engaged in dressing the slough.”—Dr. Hicks on “Puerperal Diseases:” *Trans. of Obstetrical Society*, vol. xii.

“Mrs. M——, primipara. Was very long in labour, altogether three days, from rigid os uteri. The child was dead and decomposed, putrid gas escaping with the fœtus and placenta. About the third day she began to vomit, had rapid pulse, and hot skin without delirium. The fourth day she had slightly improved, but on the fifth the symptoms returned with greater severity, the vomiting being very trying to her; the pulse then was 120 per minute, small and variable, hot skin, flushed face, glazed tongue, without diarrhœa, no rash observable; the urine free. She again made a slight improvement, but again became worse, incessantly vomiting, and died on the seventh day after delivery, and on the fourth of the attack.”—*Ibid.*

These examples prove that decaying organic matter possesses the power of inducing an analogous action in bodies with which it has contact. We must, however, bear in mind that the extension of the action is in the majority of cases successfully resisted; the healthy actions of growth and repair are generally able to overcome the diseased action of textural decay, and that it is in those persons in whom these healthy actions are impaired, whether by faulty hygienic surroundings, deficiency of

nourishment or otherwise, that the victims of zymotic disease are to be found.

When writers endeavour to explain why of many persons subjected to the cause of disease some only are affected whilst the majority escape, they tell us that the affected persons were predisposed, but they do not tell us in what this predisposition consists. Mr. Simon writes: "The inhaled 'something,' which after a fortnight's lingering in the body covers the skin and mucous membranes with the pustules of small-pox, or develops the cutaneous blotches and respiratory irritation of measles, or sets the skin and kidneys desquamating and the tonsils sloughing with scarlatina, is as real an exciting cause of inflammation as any stab, or bruise, or burn. But between the two cases there is this difference; with regard to the latter causes of inflammation, no bodily predisposition is necessary; but in all persons alike, on a certain quantity of stab, or bruise, or burn, inflammation assuredly results. Whereas, on the other hand, the morbid poisons are inoperative as exciting causes of inflammation, except where there is a definite bodily predisposition."

This argument is by no means conclusive as regards the specificity of the so-called morbid poisons in requiring predisposition in those subjected to their influence. The cases are much more analogous than appears at first. In both it is a question

of power of attack, and of successful or unsuccessful power of resistance. The heated iron that would cause the delicate hand of a lady to inflame and perhaps to mortify, may be handled with impunity by the blacksmith. The blow that would bruise a delicate face and cause inflammation, has no effect on the prizefighter. The knife that would penetrate a thin and delicate skin, would be successfully opposed by a tough hide. The predisposition that Mr. Simon speaks of as necessary to set up the symptoms of small-pox, measles, and scarlatina, is nothing more than diminished power of resistance in the textures attacked. The badly fed, scantily clothed, and foully lodged bodies of the extremely poor offer less resistance (*cæteris paribus*) to disintegrating action than the well-nourished and carefully tended structures of their more fortunate fellow-citizens.

CHAPTER III.

EPIDEMICS.

*Epidemics—Traces to be found in Early Records—
Black Death—Sweating Sickness—Plague—Cholera
—Typhus and Relapsing Fever—Enteric Fever—
Small-pox—Scarlatina—Conditions common to all
Epidemics—Determining Cause.*

WHEN from any cause—such as abnormal atmospheric condition (of which we have no precise knowledge), or the unusual prevalence of droughts or floods, or failure of crops causing famine, or devastation of large tracts of country by war, or unusually large aggregation of individuals, under faulty hygienic conditions, as in pilgrimages, or camps, or overcrowded prisons—the normal resistance of healthy action to the incursion of disease is diminished in a community, zymotic disease assumes the form of epidemic; disease that under ordinary conditions is usually met with in sporadic cases, spreads with sometimes fearful rapidity; the ordinary symptoms become aggravated; the minds of men are excited by

the unusual phenomena, and failing in their endeavour to account for the fearful visitation, they believe they are the victims of a hitherto unknown form of disease.

The earliest records we possess give instances of the prevalence of epidemics; several examples are related in the Pentateuch. The succession of events immediately preceding the Exodus from Egypt is very striking. First, an unusually foetid state of the Nile, "And the river stank, and the Egyptians could not drink of the water of the river," Ex. vii. 21. This was followed by unusual abundance of the lower forms of animal and of insect life: frogs, lice, flies. After this followed a cattle plague, succeeded by what is described as a plague of boils amongst the people; after this locusts in such quantities that "they covered the face of the whole earth, so that the land was darkened; and they did eat every herb of the land, and all the fruit of the trees which the hail had left: and there remained not any green thing in the trees, or in the herbs of the field, through all the land of Egypt" (x. 15). Then followed a terrible mortality, "And there was a great cry in Egypt, for there was not one house in which there was not one dead" (xii. 30).

Hecker, in his interesting work, has given us a graphic account of the Epidemics of the Middle Ages. The Black Death, that caused an unexampled amount of mortality in Europe, appears to have

commenced in China in 1333. China had been previously visited with drought, floods, swarms of locusts that devoured the growing crops and produced a great famine; this was followed by pestilence. In 1348 the disease reached Europe. In Cyprus, where it raged with great violence, there was observed, what was noticed in many cities and countries afterwards, "a peculiarly offensive state of the air, sometimes spoken of as a stinking mist, possibly due to the dead locusts which had never perhaps darkened the air in thicker swarms."—*Hecker*.

It appeared in England in August, 1348. "It was an Oriental plague, marked by inflammatory boils and tumours of the glands."—*Ibid*.

"It is probable that the atmosphere contained foreign and sensibly perceptible admixtures to a great extent."—*Ibid*.

In England the advent was preceded by great floods. "It rained from Christmas to Midsummer without one fair day."—*Short*.

"When the rains left there followed a great dearth of cattle, after that a dearth and scarcity of corn."—*Short*. "We read of 5000 cattle dying in one pasture, of beasts dying in holes, furrows, and ditches, in innumerable multitudes, over the whole kingdom."—*Ibid*.

The cities were with few exceptions narrowly built, filthily kept, and surrounded by stagnant ditches.

In the record of those times we have all the

conditions likely to induce a wide-spread epidemic, floods, droughts, failure of crops, cattle plague, and the presence of an enormous and unusual amount of decaying organic matter. These influences acting upon a population living under unfavourable hygienic conditions, naturally induced a large amount of disease; the usual sporadic outbreaks of plague and other forms of fever became epidemic, the aggravated symptoms leading observers to think that they had to deal with a new form of disease.

Epidemics of sweating sickness visited this country in 1485, 1506, 1517, 1528, 1551. If we look into the European records of the years preceding the last of these attacks, we find that from 1528 to 1534 had been years of scarcity. "In these years malignant fevers, especially the plague and petechial fever, had been constantly occurring."—*John Lange*.

"It was a century of putrid malignant fevers."—*Ibid*.

"In 1538 an epidemic flux spread over a great part of Europe."—*Spongerburg*.

1540 to 1543 was a period remarkable for plague.

"1542 was remarkable for swarms of locusts: they came from the interior of Asia, and travelled in dense masses over Europe, passing northward over the Elbe, and southward as far as Spain."—*Ibid*.

1545-1546.—There was an outbreak of trousse-galant in France.

1549.—There was a great outbreak of malignant

fever in Germany ; and in 1551 the last epidemic of sweating sickness occurred in England.

The hygienic condition of the inhabitants of London at that period has been described by Erasmus :—

“ The streets were generally covered with clay and rushes, which sometimes remained undisturbed for twenty years, concealing a mass of filth not fit to mention, and exhaling a vapour not wholesome for the human body. The floors of the houses generally were made of loam, strewed with rushes, constantly put on fresh without disturbing the old, lying there, in some cases for years, concealing fish-bones, broken victuals, and other filth, and impregnated with the urine of dogs and men.”

EPIDEMICS—PLAGUE.

The intense form of malignant fever known as the Plague visited the different cities of Europe, in repeated epidemics, from the sixth to the middle of the seventeenth century.

So frequent were these epidemics that Sir Gilbert Blane has calculated no less than forty-five outbreaks between 1602 and 1665. This form of zymotic disease is indigenous in Egypt, and is still annually epidemic in that country. The cause is no doubt to be sought in the enormous amount of decaying matter washed down by the Nile, and spread over a large tract of country by the annual overflow of that river; on the subsidence of the inundation, the organic decomposing material is left, and serves as a natural manure.

“The plague is more rare in Upper than in Central Egypt, and in Central than in Lower Egypt.”
—*Clot Bey*.

“The plague has never been known to pass the first cataract.”—*Williams*.

The social condition of the people is such as to offer a feeble resistance to disease.

“The crowded state of the population of Egypt, their misery and insufficient nourishment, are men-

tioned by authors as the probable cause of plague. They point to the mud-built huts, the narrow and tortuous streets of their cities, and their habitations, whether isolated in villages, or in the towns, surrounded in every direction with heaps of dung and other immundities. In the midst of these the Arab lives in his wretched cabin with his wives and children, his servants and domestic animals, all huddled together."—*Williams*.

"Unheard of filth reigns in their infected 'taudis.'"—*Clot Bey*.

"The lower classes are often reduced to live on the leaves of the thistle or on bread made from the seeds of the cotton tree."—*Williams*.

The streets are encumbered by the dead bodies of animals lying unburied; human burial is performed so carelessly that the corpse is scarcely covered with earth.

The history of the successive outbreaks of plague affords a fair opportunity for studying the mode in which epidemics arise and spread. Nearly all the epidemic outbreaks of plague can be clearly traced back to Egypt as their birthplace, and so clearly defined is the locality, that we can map out the portion of Egypt where plague is even to the present day endemic, and whence as from a focus it spreads under certain favouring conditions to surrounding countries. The Delta of the Nile has always been and still remains the cradle of the plague. Volney

tells us that the plague never comes from the interior of Egypt, but always appears first on the sea coast at Alexandria, passes from Alexandria to Rosetta, and from Rosetta to Cairo. Williams tells us that it has never been known to pass the first cataract.

What then is the special condition peculiar to the region known as the Delta of the Nile that should make it, rather than any other region, the focus of a malignant fever?

The one great fact that constitutes the specificity of this tract of country is the annual overflow of the waters of the Nile. The stream of the Nile carries down an enormous and incalculable amount of decaying organic matter, the inundation spreads this over a large tract of country, and the subsidence of the river to its bed leaves this decaying organic matter deposited on the surface of the ground. How highly charged this deposit must be with organic matter is proved by the effect it produces on the land to which it serves the purpose of a manure.

Here, then, we have the first condition necessary for the origin and diffusion of an epidemic of malignant fever.

But in order that this cause of disease should produce its full effect, a second condition is necessary—that the persons subjected to its influence should from some cause, habitual or occasional, be ill able to offer to the disease-producing cause the

successful resistance of healthy action. How are the inhabitants of the countries at the mouth of the Nile situated in this respect? Sunk in abject poverty, reduced at times to subsist on food that can barely suffice to sustain life, lodged in filthy huts, and those dwellings surrounded by filth of every description, forced to labour continuously for the means to keep up their wretched existence, all the causes that can be conceived favourable to the genesis and spread of disease exist combined in the inhabitants of those regions.

There is no lack of evidence as to the effect produced by these conditions on those subjected to the cause of plague.

“It attacks the poor rather than the rich—patients labouring under disease rather than healthy persons—individuals constitutionally feeble rather than the robust.”—*Williams*.

“Low and humid places, ill-ventilated houses—the quarters of the indigent—and populous cities with narrow, obstructed streets pay the largest tribute to this disease.”—*Clot Bey*.

“The poorer classes suffered more than the rich. In Alexandria the quarters of the poor were horribly decimated; two villages situated on the seashore—Ras-el-Tsin and Abattoir—were almost depopulated; but in no other place was there such an accumulation of filth, misery so general, or houses so ill-built and unwholesome.”—*Aubert, On the Epidemic of 1834-5*.

“Whilst the plague was spreading amongst the natives, British-born soldiers escaped, although holding intimate communication with the diseased.”—*Sir R. Wilson.*

We see, then, plainly demonstrated in the case of the Egyptian plague, all the conditions necessary for the production, and favourable to the diffusion of a malignant disease; the continuous history of eleven centuries shows how this form of fever spread repeatedly over all the countries of Europe, and if in later years its ravages have been confined to the neighbourhood of its birthplace, we may fairly attribute the change to the improved social condition of the inhabitants of Western Europe.

EPIDEMICS—CHOLERA.

India is as certainly the birthplace of cholera as Egypt is of the plague. The first distinct history of this disease assuming the epidemic form is the great outbreak, which commencing in Bengal in the autumn of 1817, gradually made its way westward, reaching England in 1831; but there can be little doubt that although it was looked upon as an entirely new disease, it had been always known in India even from the most remote periods. The Brahminical medical writings refer to a disease like cholera prevailing in India as far back as the annals of that country extend; and writers in the seventeenth and eighteenth centuries describe cases that can scarcely be mistaken for any other form of disease. The description of the disease as witnessed by Mr. Curtis in Ceylon in 1782 proves that cholera was prevalent in India in the eighteenth century, and that it sometimes spread in the form of epidemic far from the region where it originated.

The great epidemic of 1817 commenced in Jessore, a dirty, crowded town surrounded by jungle, situated in the Delta of the Ganges. The Ganges bursts its boundaries in the rainy season and overflows the country to a great extent. The water gradually sub-

siding as it trickles in innumerable channels through the Delta leaves an immense tract of country “neither perfectly overflowed nor yet quite dry, covered with animal and vegetable matter in every stage of decomposition, and combining all the other circumstances for giving miasmata their fullest influence over the human body—heat, moisture, calm, &c.—as any spot of equal extent on the surface of the globe.”—*Williams*.

In the region of the Delta of the Ganges we have thus periodically occurring, a condition that observation and experience combine in pointing out as eminently favourable to the production of zymotic disease.

The condition of the inhabitants of those parts renders them peculiarly liable to suffer from the effects of disease. Badly fed, scantily clothed, ill-lodged, the lower classes oppose but a feeble resistance to the destructive agent, and wherever the cholera extended to, we find that this law obtained. “In India the city of palaces forms only one-half (the English) of the city of Calcutta; the other is the native town, which contains, in connexion with the suburbs, at least 500,000 inhabitants. The native town is composed chiefly of miserable lanes, narrow, dirty, and unpaved, and the majority of dwellings are low huts, with side walls built of mud, mats, and bamboos, and covered with small tiles. Amongst the swarming population of these filthy

receptacles, in which all descriptions of disgusting animal and vegetable odours abound, the distemper ran a wild career of destruction.”—*Kennedy*.

In Moscow the mortality was almost confined to the poorer class. In Paris the largest mortality was in the eleventh arrondissement—the poorest. In London also the chief mortality has always been amongst the class that is poorly fed and badly housed.

In the army Mr. Orton tells us “that the officer suffered less than the soldier, the cavalry less than the infantry, and these again less than the hard-labouring, ill-fed camp followers.”

In the present state of medical science we cannot explain the reason why at irregular intervals, certain forms of disease, endemic in some countries, and generally met with in the sporadic form, assume an epidemic character, and extend their action over a large portion of the earth’s surface—why cholera starting from Jessore in 1817 should have extended from country to country, until in the course of sixteen years it had encircled the earth with a girdle of disease. We cannot say if there be any special atmospheric condition that renders the human body liable to manifest the action of zymotic disease in the shape known to us as cholera. Mr. Glaisher, in his summary to the report of the “Commission on Cholera Epidemic of 1854,” gives the following particulars:—“The three epidemics

(1832, 1849, 1854) were attended with a particular state of the atmosphere, characterized by a prevalent mist, thin in high places, dense in low. During the height of the epidemic in all cases the reading of the barometer was remarkably high, the atmosphere thick, and in 1849 and 1854 the temperature above the average. A total absence of rain and a stillness of air amounting almost to calm accompanied the progress of the disease on each occasion. In places near the river the night temperatures were high, with small diurnal range, with a dense torpid mist, and air charged with many impurities arising from the exhalations of the Thames and adjoining marshes, a deficiency of electricity, and, as shown in 1854, a total absence of ozone, most probably destroyed by the decomposition of the organic matter with which the air in these situations is so strongly charged."—*Mr. Glaisher's Summary, Report of Commission on Cholera Epidemic of 1854.*

A study of the items of mortality, as reported by the Registrar-General, shows a gradual increase in the deaths from diarrhœa for several years previous to the epidemic of 1849.

Thus the deaths from diarrhœa per million of persons living in 1840 were 246 ; 1841, 248 ; 1842, 369 ; 1843, 428 ; 1844, 348 ; 1845, 407 ; 1846, 1022 ; 1847, 886 ; 1848, 857 ; and in 1849 diarrhœa 1522, and cholera 6209.

If we refer to the accounts we possess of the state

of the populations previous to the outbreak of each of the great epidemics of the Middle Ages, we meet with analogous facts.

Each outbreak of sweating sickness was preceded by several seasons in which petechial and other fevers are said to have been unusually prevalent.

Sydenham tells us that autumnal intermittents broke out in 1661 in London, did fearful mischief, "causing excessive mortality, and carrying off whole families, and continued to prevail until 1665, the year of the Plague."

These facts tend to show that whatever be the condition (meteorologic or other) that determines the character that the prevailing form of zymotic disease shall assume, and favours its diffusion in the form of epidemic, such condition is gradually induced, and extends over a longer period than we have been accustomed to believe.

Difficult as it is to investigate the laws that regulate the outbreak and spread of cholera, such data as we possess are in accordance with the known laws that regulate the origin and diffusion of other forms of zymotic disease. It commences in a region whose geographical position causes periodically the exposure (under a tropical sun) of enormous quantities of decaying organic matter, animal and vegetable; the inhabitants of the districts, from their mode of life and hygienic sur-

roundings, are in the worst possible condition for resisting the disease-producing cause; and when the disease spreads to other countries, we find that it especially affects those localities where the same condition of the presence of disintegrating organic matter favours its advent, and whose inhabitants, by their social position and hygienic surroundings, are least able to offer an efficient resistance.

EPIDEMICS—TYPHUS.

Typhus being one of the most usual forms in which zymotic disease manifests itself in Great Britain and Ireland, being, as it is termed, endemic in these islands, ample opportunity has been afforded to study the circumstances under which it assumes the epidemic type.

All observers are agreed in tracing the origin of this disease to the presence of decaying organic matter, and generally to the decaying animal matter thrown off from the bodies of human beings under favouring conditions—disintegrating tissue waste.

“Typhus is a disease attacking persons of all ages, generated by contagion or by overcrowding of human beings with deficient ventilation, and prevailing in an epidemic form in periods or under circumstances of famine and destitution.”—*Murchison*.

“Typhus arises from the effluvium of human live bodies.”—*Dr. Brown Langrish*.

“The hospitals of an army, when crowded with sick or at any time when the air is confined, produce a fever of a malignant kind, and very mortal. I have seen the same sort arise in foul and crowded barracks, and in transport ships when filled beyond

a due number, or when the men were kept at sea under close hatches in stormy weather.”—*Pringle*.

“Typhus arises from the concentrated emanations from living bodies or from contagion.”—*Dr. William Grant*.

“That typhus arises from the putrefaction of the perspirable matter admits of every species of evidence applicable to a matter of fact and observation.”—*Hildenbrand*.

“Typhus is generated by air too much charged with exhalations from human beings.”—*Ibid*.

Thus all observers agree, that the originating cause of typhus is the being subjected under favouring circumstances to the influence of disintegrating organic matter. It may be called “emanations from the bodies of persons kept in an unchanged atmosphere,” or “human exhalations,” or “the effluvium of live bodies,” but the fact is ever the same. Even those who believe that typhus depends upon a special poison, state in their writings that such poison may be generated *de novo* under the conditions of “overcrowding, deficient food, and dirt.”*

As might have been *à priori* expected, typhus is a very usual addition to the horrors of war. Mur-

* Dr. Murchison gives the detailed history of several local outbreaks of typhus that have come under his personal observation, in which there was no evidence of the disease having been engendered by contact with persons already affected.

chison tells us that in many parts of Europe, where typhus rarely occurs in time of peace, it becomes epidemic in time of war. The two conditions essential for the origin and diffusion of zymotic disease are rarely absent from camps and besieged cities:— 1st. The overcrowding of men and horses under necessarily unfavourable hygienic conditions, subjects soldiers to the influence of the disease-producing cause, and the scarcity and mental and bodily depression engendered amongst the inhabitants of the war-ravaged country favour the diffusion of the disease in the form of epidemic.*

“ During the whole period of the revolutionary wars, typhus followed in the march of the French armies, and spread along their route. In the campaigns of 1804–9 typhus did not spare a single village on the route from Strasbourg to Vienna; and in 1812 the long line from Mayence to Vienna was in like manner infected.”—*Williams*.

The dreadful sufferings of the French army from typhus during its retreat from Russia in 1812 are well known.

“ In 1813 there perished by typhus in Dantzic

* “ The soldiers of the French army during their retreat from Italy in 1799 communicated fever to the inhabitants of fifteen towns and villages where they halted on their route. The soldiers suffered from privations of every kind. They were ill-fed, their clothes were in tatters, their bodies were covered with filth and exhaled a noxious smell, and their shirts, unchanged for many months, were glued to their skin.”—*Williams*.

two-thirds of the French garrison and one-fourth of the population.”

Typhus also prevailed largely amongst the British troops that returned from Corunna.

“Our army in the Crimea suffered dreadfully from typhus in the winter of 1854-5, when the commissariat was very bad and the hygienic surroundings such as to favour the development of zymotic disease. At that time the condition of the French troops as regards food and lodgment was superior to ours, and they consequently suffered much less; but when in the succeeding winter the relative positions were altered as regards food and lodging, it became the turn of the French to suffer, and it is computed that 12,000 out of 120,000 were affected with typhus, of whom 6000 died.”

“The disease was not endemic in the Crimea, and no evidence has been adduced to show that it was imported; but its origin was universally attributed to overcrowding and deficient food.”—*Murchison.*

After reading the description of prisons by John Howard, as they existed in his time, we can have no difficulty in understanding how typhus was repeatedly generated in them, and the description of the well-known Black Assizes must excite horror, but not surprise.

“Scantily supplied with water, air, and light, the

prisoners literally rotted in close rooms, cells, and subterranean dungeons.”—*Howard*.

“Some jails have no privies.”—*Ibid*.

“Add to this that the prisoners were half-starved.”—*Ibid*.

So foul was the air of the jails, that Howard’s clothes in his first journeys were so offensive, that he could not close the windows of his post-chaise, and was therefore often obliged to travel on horseback.

Dartford County Bridewell.—“No chimneys, offensive sewers; the rooms dirty. No water; no straw.”—*Howard*.

Cambridge Town Bridewell.—“Seventeen women were shut up in the daytime in a room nineteen feet square, which has no fire-place or sewer.”—*Ibid*.

It would be tedious to repeat in detail the history so often told of the various Black Assizes, when typhus, always more or less rife in the prisons, was spread through the medium of the prisoners amongst the general population. The first occurred at Cambridge, 1522; the second at Oxford, 1577; the third, 1586, at Exeter.

“Some time before, thirty-eight Portuguese seamen had been cast into ‘a deep pit and stinking dungeon’ in Exeter Castle. They had no change of raiment, and were left to lie upon the bare ground; many of them were sick during their trial, and by them the disease was communicated to those present in court.”—*Holinshed*. Fourth, Taunton, 1736;

fifth, at Launceston, 1742; sixth, Old Bailey, 1750.

When we consider the condition of the cells in which the prisoners had been lying previous to being brought into court, we cannot be surprised that typhus was communicated to many who were present, and as each of these persons must, when in his home, have become a fresh and separate nucleus from which the disease would be communicated to others, it would naturally assume the appearance of an epidemic. But if we inquire into the particulars of these outbreaks, we shall find that the range was limited, and that typhus did not spread over large tracts of country, that the number attacked was limited, and that attention was excited by the social rank of the victims and the peculiar circumstances under which the disease was engendered amongst them. That these outbreaks did not assume the dimensions of widespread epidemic, must be attributed to the absence of the second condition that has been stated as essential to a general epidemic of zymotic disease—viz., some cause affecting the general population of a country, and overcoming the normal resistance of healthy action to the incursion of disease.* Where such condition is present,

* As illustrations of this law the following may be quoted:—"A large number of Spanish prisoners were confined in Forton prison in 1780. Typhus broke out among them with great severity. During seventeen weeks 785 cases were admitted into hospital, of whom

the disease loses its sporadic character, and becomes truly epidemic. The medical history of Ireland furnishes us with several melancholy illustrations.

“The winter of 1815–16 was unusually severe, and was followed by a cold and wet summer and autumn, and in Ireland there was a complete failure of the harvest and of the potato crop. The harvest of the following year was no better. In September, 1817, the thermometer in Ireland fell suddenly from 75° to 30°, and the cold completely destroyed the potato crop and the late oats. In the month of December sheaves of corn might be seen rotting on the ground. Owing to the wet seasons, the turf or peat, the chief fuel of the poor, could not be cut. Many of the working classes were thrown out of employment. Extreme distress ensued. Towards the end of 1816 fever became very prevalent in Cork. In the spring of 1817 it spread extensively in Ulster, Munster, and Connaught, and in the autumn to Leinster. In Dublin it commenced in September, 1817. It raged in Ireland until the end of 1819, having prevailed over all Ireland and extended to England and Scotland.”—*Murchison*.

156 died. At the same time 229 Americans were confined in another part of the same prison. Not one of the Americans was attacked with fever.

When the London Fever Hospital was one of a row of houses in Gray's Inn Lane, typhus never spread to the inmates of the neighbouring houses.

The number of sick was estimated at 737,000, out of a population of 6,000,000. In Dublin a third of the population was affected.

The failure of the potato crop in 1846 was followed by an epidemic of fever that spread widely over England, Ireland, and Scotland, and did not entirely subside until the end of 1848. It is estimated that the number of cases in Ireland alone exceeded a million.

The foregoing facts show that continued fever is one of the usual forms in which zymotic disease manifests itself in Western Europe, when persons are submitted to the action of decaying organic matter, and that it assumes the epidemic type when masses of the population are subjected to such influences, natural or accidental, as diminish the resistance of healthy action to the incursion of disease.

ENTERIC FEVER.

The etiology of Enteric Fever has been so carefully studied and so clearly shown to be caused by decaying organic matter, that no further argument is necessary. The investigations of Pringle, Jenner, Murchison, and of many Continental observers, have fixed the etiology of this disease as one of the least questioned facts of medical science.

The histories of the various local outbreaks detailed by Dr. Murchison in his treatise on Continued Fevers would be sufficient to prove the causation, and in some of them the disintegrating matter can be clearly traced to the alimentary canal through the medium of drinking waters.

The outbreak that occurred in 1859 at Bedford was caused by faecal matter from cesspools soaking into the wells: the water was found to contain a large quantity of decaying animal matter.

In 1847 a local outbreak of enteric fever occurred in Richmond Terrace, Clifton. The inmates of thirteen houses out of the thirty-four constituting the terrace were affected. The inhabitants of those houses drew their water supply from a well that betrayed by its taste and smell contamination from decaying animal matter.

The outbreak in Windsor in 1858,
That of Cowbridge in Wales in 1853,
The Croydon fever in 1852,
The fever that broke out in Westminster School
in 1848,

The attack at the Clapham School in 1829,
have all been clearly traced by Dr. Murchison to the
presence of decaying organic matter.

More recently, in 1870, the local outbreak of
fever at Islington was proved by Dr. Ballard to
be caused by decaying organic matter mediately
contaminating the milk that was supplied to the
persons attacked.

When this disease assumes the form of epidemic,
the outbreak is generally in a great measure con-
fined to those who are subjected to the direct
action of the disease-producing cause ; and so limited
is the power of communication, that many physicians
have doubted or even denied its contagiousness.
The following very suggestive passages, in their
bearing on the correlation of different varieties of
fever, occur in Dr. Murchison's treatise :—

“ The opinion has been long prevalent in America
that enteric fever has a tendency to take the place
of intermittents and remittents, as these diseases
from the effects of cultivation and other causes
decrease or disappear. This opinion has been to
some extent corroborated by the investigations of
M. Boudin.”

In connexion with this subject is a remarkable communication made to the French Academy of Sciences in 1845, by M. Ançelon :—

“Many years before, enteric fever had been constantly endemic in the Commune of Guermange in Lorraine, making its appearance every year during the hot season ; but for twenty-five years it had entirely disappeared from the northern part of the commune, its disappearance having been simultaneous with the suppression of a stagnant pond in that locality. At the southern part of the commune, however, there had been epidemics of intermittent fever every third year, alternating with epidemics of enteric fever and of furuncular diseases. At this part of the commune was a large lake called the ‘Indre basse,’ which every third year was emptied and cultivated, and afterwards the water was allowed to collect again for two years. The intermittent fevers appeared during the first year that the pond was full of water. The epidemics of enteric fever coincided with the second year.” In this year the pond began to dry, and M. Ançelon attributed the fever to the action of heat and moisture upon an immense quantity of animal and vegetable débris which during the two years had been collecting upon the banks of the lake.

THE EXANTHEMATA.

The three varieties of zymotic disease known as the Exanthemata—Small-pox, Measles, and Scarlatina—have been so generally looked upon as specific diseases caused by special morbid poisons, that few observations have been recorded respecting their causation.

It is so entirely assumed as a fact that small-pox does not exist independently of human contagion, that no accurate or careful observations appear to have been made on the subject; no rational explanation is offered as to the origin of sporadic cases, although every physician must occasionally meet with a case where there is no history of contagion, where no trace can be discovered of any case having occurred in the neighbourhood; but in such a case the fact of contagion is assumed, and in the absence of evidence, supposititious modes of contagion are accepted in explanation. "It must have been caught by riding in a cab that must have previously carried a small-pox patient;" or the disease is supposed to have been caught by "passing an infected child on the opposite side of the street."—*Williams*. Such an explanation as the last being accepted notwithstanding the experiment of Dr.

O’Ryan of Lyons, who placed several children round an oval table whose least diameter was three feet, and in the centre he placed dossils of lint and of silk strongly impregnated with variolous matter taken from persons labouring under either the natural or the inoculated small-pox. This experiment was repeated every morning for a week—sometimes in the open air, and sometimes in an apartment—without any of the children being affected, since they all remained free from the disease nine months afterwards.

There are examples on record of small-pox having broken out in a regiment marching in India where the men, previously healthy, were many miles from any probable source of contagion.

When we refer back to the pathology of small-pox, we find that the symptoms during life are in strict analogy with the phenomena presented by other varieties of zymotic disease that are known to be produced by decaying organic matter—when small-pox assumes the form of epidemic, such epidemic observes the same laws of diffusion and of decline as regulate the epidemics of cholera and of typhus, and in the dead-house the body gives evidence of structural disintegration in accordance with the texture affected.

Little doubt can be entertained that when the minds of physicians are directed to the possible occurrence of small-pox otherwise than by contagion,

there will be no difficulty in tracing it, like the other varieties of zymotic disease, to the action of decaying organic matter. The connexion of small-pox with the other varieties of zymotic disease will also be perceived by referring to the tables showing the distribution of epidemics.

The occurrence of scarlatina otherwise than by contagion with an individual already affected by the disease, appears to take place under analogous circumstances to those that mark the genesis of other forms of zymotic disease. When due care is taken to investigate the cause that has led to an outbreak of this disease, it will be invariably found that the persons who were first attacked were exposed to the influence of decaying organic matter. It has been frequently noticed how commonly the early occupants of newly built houses are attacked by scarlatina, and upon investigating these cases it has been found that frequently the houses have been built upon soil not duly prepared, and consisting in a great measure of disintegrating organic matter, as where houses are built in suburban districts upon garden ground; and in other cases, where the soil has been gravel, the gravel dug out has been sold, and the foundation filled in with miscellaneous refuse, a large portion of which has consisted of organic matter subject to and perhaps in the very process of disintegration.

If we inquire into the season when scarlatina is

most prevalent, we find that it is in the later summer and autumn months, when decaying organic matter abounds, and a high temperature lends unusual activity to the molecular motion. And if we glance at the circumstances under which scarlatina assumes the epidemic form, we have revealed to us the picture so repeatedly drawn in these pages—decaying organic matter affecting a special structure setting up the well recognised structural lesions and spreading as an epidemic amongst those who by their hygienic surroundings or other cause have failed to offer efficient resistance. The ill-fed, the scantily clothed, the foully housed, afford to this, as to other forms of zymotic disease, the greater proportion of victims.

In a paper read before the Medical Society of London, in January, 1871, Dr. Carpenter of Croydon has proved how much even a single able and unprejudiced observer can do towards elucidating the causes of disease. Refusing to be bound by the prevailing doctrine of special germs pervading the atmosphere, Dr. Carpenter carefully watched the circumstances under which several local outbreaks of scarlatina occurred in and about Croydon during a period of eight years, and in every instance succeeded in tracing its origin to the presence of decaying organic matter; and further showed that where a recurrence of such cause was prevented, the disease did not return, whereas repeated

attacks were the penalty of ignorance as to the cause, or carelessness as to the removal of such cause.

The cases related by Dr. Carpenter are very instructive, and appear to trace most conclusively the originating cause to decaying organic matter.

“The children of B. B., living in a well ventilated house on a hill-side, were all seized with scarlatina in the month of September, 1864. For three or four days previously the house had been pervaded with a most nauseous smell, which proceeded from some market-gardens to windward of the house. The wind had blown very quietly from the same quarter all that time. The weather had been very hot, and was moderately dry. The miasms from the gardens below seemed to hang upon the crest of the hill. There was no scarlatina prevalent in the place, and there was no known communication with any other house in which scarlatina had occurred. The servants denied they had visited any infected houses, and the children had not been away from the hill for some time previously. Six cases occurred in that house, all commencing within forty-eight hours of each other. They were all mild in character, they ran the usual course, and each recovered in the ordinary time. The infection did not spread to others, the ordinary means of isolation and disinfection being at once employed.

“At the same time, on the same hill and within the same forty-eight hours, two other families had

children affected with the disease of the like mild character, and with a similar result. The inmates of these houses were not known to each other, the children had not played together, the servants were not acquainted, they did not employ the same laundresses, the same milkmen, and scarcely the same tradesmen. Everything seemed arranged so as to make it conclusive that the miasms from the market-gardens were the cause of the disease. These miasms were produced by slaughter-house refuse, with which the market-gardens had been manured. The manure had been spread upon the ground as a top-dressing in dry weather, and had not been ploughed in, and at a time when there was marked absence of ozone. Complaint was made to the local authority on the subject. The epidemic ceased immediately the nuisance was removed by the manure being ploughed in, and no other cases as far as could be ascertained occurred at that place at that time."

"In the autumn of 1865 a nauseous sickening odour pervaded the neighbourhood of Park Hill and Croham near to Croydon. This odour was traced to the application of slaughter-house manure to a field at Croham. This manure had been brought by railway from London, and spread upon the soil for some time before it was ploughed into it. Four or five days afterwards cases of scarlatina occurred simultaneously in three large schools in

that neighbourhood, several persons being attacked in each establishment. The first cases commenced in each school within three days of each other, whilst no known intercommunication, whether by milkmen, laundress, or butcher, existed between them. One school consisted of about one hundred and twenty children belonging to the Society of Friends. The second was a girls' school about two hundred yards from the first, and the third a boys' school about a hundred yards further on. They were all situated on different lines of sewers, and there did not appear to be anything in common to account for the attacks of coincident illness.

“A large school existed in Croydon which on several occasions had been broken-up by reason of outbreaks of scarlatina; eventually the master gave up the house and it became disused for a time. It was then discovered that a very large cesspool under the children's playground had not been filled up when the privy was changed into a water-closet some years before. This cesspool seemed at times to receive the washings from a neighbouring slaughter-house, and doubtless some blood by the same means. This cesspool was filled in, and the slaughter-house had its connexion made more correctly with the sewer. The house has been inhabited again for several years by a large family; the gentleman head of the establishment taking pupils; but scarlatina has not appeared there since.”

“The village of S—— C——, near Sevenoaks, was in a similar manner severely visited by scarlatina in the autumn of 1870. The writer has no personal knowledge of this case, but he is informed on reliable authority that the public well is in close proximity to the slaughter-house of the district, and it was proved by analysis that the water of that well was contaminated with blood-products.”

Other cases equally interesting and convincing are related by Dr. Carpenter.

When outbreaks of the other varieties of exanthemata shall be as carefully investigated as these of scarlatina, there can be little doubt that they also will be traced to the presence of decaying organic matter. Even in the present state of our knowledge all epidemics that we have been enabled to trace to their origin, have as their one constant condition, the exposure of the persons in whom the attack commenced to the influence of organic matter in a state of disintegration; the disease subsequently extending by mediate or immediate contact to other persons, and the extent of such diffusion being regulated by the greater or less predisposition to the disease of those submitted to the contagion, such predisposition consisting exclusively in a lessened power of resistance.

This loss of resisting power may depend on causes special to the individual, as weakness caused by previous illness or otherwise; or congenital

want of power. In this manner we can explain why of several members of one household exposed to the disease-producing cause some are affected and others escape; and why some have the disease in a more aggravated form than others. Or the cause may be common to larger or smaller groups exposed to some special noxious influence, such as unfavourable hygienic surroundings. Thus we see that the parts of cities inhabited by the poor suffer in inordinate proportion during the prevalence of any form of epidemic disease. Or large masses of population may have their normal powers of resistance lessened by famine consequent on failure of crops, when varieties of disease usually met with in the sporadic form become epidemic. Nor may we omit to notice as a probable cause, certain meteorologic conditions of which we have no precise knowledge.

CHAPTER IV.

DISTRIBUTION OF EPIDEMICS.

*Distribution of Zymotic Diseases—Statistical Tables
—Correlation of Zymotic Diseases confirmed by
Statistics.*

THE originating cause of zymotic disease appears to have always been, and still remains endemic, wherever human beings are aggregated. The determining cause that regulates what especial form the disease shall assume, in other words, what special structure the disease-producing cause shall primarily affect, appears to depend upon a variety of circumstances. The hygienic conditions under which a community is living have, as might have been predicated, a very powerful influence in determining the form of the disease. Thus Dr. Murchison gives evidence for supposing that the modern typhus, always more or less endemic in these islands, is the analogue of the plague that used formerly to commit such ravages in this country; that the milder form of the zymotic has taken the place of the former terrible disease, and that the modification is due to improved hygiene. Some observers noting that notwithstand-

ing the undoubted prophylactic value of vaccination the annual mortality from zymotic disease is not materially lessened since the introduction of the practice, have endeavoured in various ways to account for this fact; and it is argued by some that the zymotic action has assumed other forms, and that the same mortality is caused by zymotic disease exhibited in those other forms. M. Carnot appears to have paid much attention to this subject, and in his work published in 1856, argues that enteric fever that now causes so great a mortality, has in France assumed the place of the much dreaded small-pox, and that the zymotic action affects the lining of the intestinal canal instead of expending its force on the dermic envelope.

Dr. Bostock writes: "It was about the period when the western part of the old Continent was in its lowest state of degradation that we hear of the ravages of those varieties of fever emphatically styled 'Black death' and Plague, which were described in the thirteenth, fourteenth, and sixteenth centuries as invading various parts of Europe and Asia, and sweeping away a large proportion of the inhabitants. The account which we have of these epidemics would indicate that they were not absolutely new diseases, but that the symptoms were modified and aggravated by the peculiar condition of the great bulk of the people."

Climate has undoubtedly a powerful influence in

determining the form that zymotic action shall assume. Thus in India, the especial home of cholera, scarlatina is comparatively unknown, and typhus is so rare that it is even now a subject of debate whether it ever occurs in those climes. Egypt appears to be the cradle and favoured site of the plague. Ireland is known to be never free from typhus, whilst enteric fever is well known to be endemic in England and is the prevailing type of fever in France.

Certain meteorologic conditions, the precise nature of which cannot in the present state of science be explained, must be admitted as an additional determining cause that has immense influence in fixing the prevailing form that zymotic disease shall assume.

Thus, for several successive years the intestinal canal is the favourite structure affected by zymotic disease, and we see year by year the numbers of deaths from diarrhœa increasing until the cycle culminates in an epidemic of cholera. In other years the exanthemata become the marked feature in zymotic mortality, and sporadic cases of scarlatina or of small-pox increase to the proportions of a pestilence.

This, again, may be followed by a series of years during which the above diseases shrink into their usual restricted channel, and malignant fevers claim the majority of the victims of zymotic disease.

Observers have not failed to note the strong proof of the correlation of the various forms of zymotic disease that is afforded by a study of the death register.

It has been remarked that the sum of deaths from all forms of zymotic disease is a tolerably constant quantity, whilst the distribution of the sum amongst the various forms is a quantity constantly varying. "Each year seems to have its prevailing epidemic, but the total number of deaths from zymotic disease remains much the same."—*Letheby, Report of Health of City of London, 1870.*

Deaths from Zymotic Disease in every 1,000,000 Persons Living, collected from the Reports of the Registrar-General, by Dr. Guy, and published by him in the Statistical Journal for 1855.

<i>Causes of Death in .</i>	1840.	1841.	1842.
From all Causes. .	25,206	24,197	23,699
„ Zymotic Diseases	4,575	4,226	4,046
„ Small-pox . .	673	563	188
„ Measles . .	617	520	677
„ Scarlatina . .	1,064	354	641
„ Diarrhœa . .	246	248	369
„ Cholera . .	33	15	62
„ Typhus . .	687	615	615
<i>Causes of Death in .</i>	1843.	1844.	1845.
From all Causes. .	24,947	24,878	23,392
„ Zymotic Diseases	5,160	5,520	4,636
„ Small-pox . .	225	890	440
„ Measles . .	741	583	1,122
„ Scarlatina . .	959	1,494	525
„ Diarrhœa . .	428	348	407
„ Cholera . .	44	32	21
„ Typhus . .	1,070	837	630

<i>Causes of Death in .</i>	1846.	1847.	1848.
From all Causes.	23,306	27,105	25,831
„ Zymotic Diseases	4,549	6,285	7,938
„ Small-pox	122	428	725
„ Measles	355	797	512
„ Scarlatina	441	643	2,132
„ Diarrhœa	1,022	886	857
„ Cholera	108	52	292
„ Typhus	853	1,428	1,600

<i>Causes of Death in .</i>	1849.	1850.	1851.
From all Causes	30,080	20,940	23,399
„ Zymotic Diseases	12,302	4,135	5,234
„ Small-pox	228	215	451
„ Measles	507	421	559
„ Scarlatina	943	507	536
„ Diarrhœa	1,522	812	960
„ Cholera	6,209	55	90
„ Typhus	1,090	829	992

<i>Causes of Death in</i>	1852.
From all Causes	22,474
„ Zymotic Diseases	4,904
„ Small-pox	483
„ Measles	249
„ Scarlatina	1,057
„ Diarrhœa	897
„ Cholera	67
„ Typhus	897

Registrar-General's Tables of Deaths from Different Causes per Million Persons Living.

<i>Causes of Death in .</i>	1853.	1854.	1855.
From Zymotic Diseases	5,315	10,541	4,567
„ Small-pox . .	87	269	136
„ Measles . .	402	558	397
„ Scarlatina . .	825	1,371	935
„ Diarrhœa . .	921	1,290	689
„ Cholera . .	351	4,269	45
„ Typhus . .	1,057	1,064	865
<i>Causes of Death in .</i>	1856.	1857.	1858.
From Zymotic Diseases	4,148	4,739	5,757
„ Small-pox . .	121	206	335
„ Measles . .	379	313	481
„ Scarlatina . .	752	746	1,572
„ Diarrhœa . .	734	1,111	719
„ Cholera . .	46	60	35
„ Typhus . .	818	957	928
<i>Causes of Death in .</i>	1859.	1860.	1861.
From Zymotic Diseases	5,469	3,863	4,424
„ Small-pox . .	197	140	66
„ Measles . .	490	487	455
„ Scarlatina . .	1,021	493	456
„ Diarrhœa . .	940	494	944
„ Cholera . .	45	17	42
„ Typhus . .	814	663	776

<i>Causes of Death in .</i>	1862.	1863.	1864.
From Zymotic Diseases	4,551	5,877	5,770
„ Small-pox . .	81	293	373
„ Measles . .	487	558	404
„ Scarlatina . .	738	1,498	1,443
„ Diarrhœa . .	552	735	798
„ Cholera . .	25	40	45
„ Typhus . .	931	886	977
<i>Causes of Death in .</i>	1865.	1866.	1867.
From Zymotic Diseases	5,489	5,522	4,288
„ Small-pox . .	309	144	118
„ Measles . .	412	521	310
„ Scarlatina . .	852	656	580
„ Diarrhœa . .	1,132	818	937
„ Cholera . .	62	685	43
„ Typhus . .	1,109	1,005	795

Mortality from Zymotic Disease in London during the first Six Months of the Year 1871 (Weekly Returns of Registrar-General).

<i>Week ending</i>	<i>Zymotic Disease.</i>	<i>Small-pox.</i>	<i>Measles.</i>	<i>Scarlatina.</i>	<i>Fever (Typhus, Typhoid, and Simple Continued).</i>
January 7	401	79	34	112	37
„ 14	398	135	27	77	38
„ 21	453	188	17	68	34
„ 28	396	157	12	66	30
Total	1648	559	90	323	139
February 4	396	196	12	49	39
„ 11	434	211	9	46	43
„ 18	433	218	11	48	35
„ 25	428	227	15	47	34
Total	1691	852	47	190	151
March 4	450	213	15	53	36
„ 11	445	194	14	54	27
„ 18	370	185	13	33	43
„ 25	390	205	10	36	32
Total	1655	797	52	176	138

Mortality from Zymotic Disease, &c.—continued.

<i>Week ending</i>	<i>Zymotic Disease.</i>	<i>Small-pox.</i>	<i>Measles.</i>	<i>Scarlatina.</i>	<i>Fever (Typhus, Typhoid, and Simple Continued).</i>
April 1	368	192	13	33	31
„ 8	402	214	15	36	29
„ 15	452	265	6	40	31
„ 22	453	276	13	27	33
„ 29	445	261	12	30	37
Total	2120*	1208	59	166	161
May 6	447	288	17	29	34
„ 13	380	232	16	35	24
„ 20	461	267	11	28	34
„ 27	426	257	23	28	23
Total	1714	1044	67	120	115
June 3	400	229	16	25	43
„ 10	418	245	23	34	30
„ 17	384	240	21	17	22
„ 24	393	232	27	24	22
Total	1595	946	87	100	117

* It should be observed that the returns for *five* weeks are included in this month.

Compare the death-rate from the exanthemata for the years 1841 and 1842. In the former year the deaths from variola were 563 per million, in the latter 188, but in 1842 there was an increase in the deaths from measles and scarlatina that went far towards restoring the balance.

In 1844 scarlatina destroyed 1494 per million, a number that in 1845 was reduced to 525; but in the latter year the mortality from measles was 1122 per million, against 583 in 1844.

In 1845 the exanthemata destroyed 2087 per million, whilst 428 per million died of cholera and diarrhœa.

In the succeeding year the deaths per million from the exanthemata were only 918, whilst, on the other hand, the deaths from cholera and diarrhœa had increased to 1130.

In 1849 there was a severe and widely spread epidemic of cholera, and the deaths per million from cholera and diarrhœa were 7721. In that year the deaths from the exanthemata were less per million than in 1848 by 1691; and from continued fever by 510.

In 1852 scarlatina increased from 536 per million in 1851 to 1057; but this increase was exactly balanced by the decrease in measles, diarrhœa, cholera, and fever, which decreased from 2601 in 1851 to 2110 in 1852.

In 1853 the deaths from variola and scarlatina

were less than in 1852 by 628 per million, whilst the deaths per million from measles, diarrhœa, cholera, and fever were more than in 1852 by 621 per million.

In 1862 the deaths per million from diarrhœa and cholera were less by 409 than in 1861, whilst the deaths per million from the exanthem and fever were more by 484 per million.

In 1865 the deaths per million from scarlatina were less by 591 than in 1864, but the deaths from diarrhœa and cholera had increased by 309 per million.

In 1866 the deaths per million from scarlatina were less than in 1865 by 296, whilst the deaths per million from cholera and diarrhœa had increased by 309.

The weekly reports of the deaths in London from zymotic disease during the prevalence of the epidemic of small-pox in 1871 show the deaths from scarlatina steadily diminishing as those from small-pox increase. This can be traced in the tables week by week, the total of deaths from zymotic disease remaining during the entire period at a nearly uniform figure.

We may here again refer to the gradual increase of deaths from diarrhœa year by year from 1841 until the epidemic of cholera in 1849. On referring to the tables the numbers will be found as follows :—

In 1841, 248 ; 1842, 369 ; 1843, 428 ; 1844, 348 ; 1845, 407 ; 1846, 1022 ; 1847, 886 ; 1848, 857 ; 1849 (1522 from diarrhœa, exclusive of the deaths from cholera).

The facts revealed by a study of these statistics are too uniform to be looked upon as fortuitous, and point to a much closer relation between the various forms of zymotic disease than has been hitherto supposed to exist. Upon no theory of special poisons giving rise to special and specific diseases can we explain the reason why the different factors constituting the sum of mortality from the various forms of zymotic disease are mutually complementary ; this can only be explained on the hypothesis of a correlation so intimate as to indicate a great probability that these diseases may claim an intimate relation as regards causation.

CHAPTER V.

CONTAGION.

Contagion—Diffusion of Disease by Contagion observed by the Ancients—Ancient Sanitary Laws—Contagion a Physical Law of Universal Application—Examples.

As zymotic disease must have necessarily occurred whenever and wherever human beings were sufficiently aggregated to give rise to the condition necessary to its genesis, attention must have been at a remote period directed to the mode in which the different forms of the disease are communicated from individual to individual by mediate or immediate contact. That such was the fact is proved by the minute directions given in Leviticus for the careful isolation of the sick, and the directions ordained by the oral law and still extant in the Talmud, show that the ancient Jews were aware of the mode in which certain diseases are capable of being communicated by mediate contagion. One of the Talmudic laws still observed in many parts of the world, directs that when a death occurs in

any Jewish dwelling, the stored water in three houses on each side of the dwelling where the death has occurred, shall be poured away. Another law is that all the passages and staircases along which a dead body has been carried, shall immediately undergo a thorough cleansing and purification.

We see therefore that the sanitary legislation of the present day was foreshadowed at a very remote period, and that the importance of studying the laws that regulate the diffusion of zymotic disease was recognised long before Dr. Williams expressed in words the great truth that "contagion is as important an element in medicine as gravity in mechanics, or electricity in chemistry."

Daily observation and universal experience having proved beyond a doubt that certain forms of disease are communicated to healthy individuals by contact with those affected by such disease, great efforts have been made to discover the exact manner in which this contagion acts, and to detect the form that the contagium assumes.

In a preceding page of this section the various hypotheses that have been brought forward have been examined, and an opinion has been ventured as to how far they are borne out by evidence.

Great importance has been attached to contagion, as evidence of the specificity of each form of zymotic disease, as though the principle of contagion were a special quality peculiarly appertaining to

these particular varieties of diseased action. If, however, we allow ourselves to take a somewhat wider view of this property, we shall find that it is by no means confined to zymotic disease, but is in fact a property enjoyed in common by all organized beings, healthy or diseased, and that its range is not even confined to the organic world, but that it can be traced to non-organized matter.

Bearing always in mind that the true explanation of contagion is the action of the physical law that "a molecule set in motion by any power is capable of communicating its own motion to another molecule with which it may have contact," let us examine some of the instances of the working of this law.* The contagion of motion as witnessed when a stone is dropped into water, the diffusion of heat from molecule to molecule, the phenomena of crystallization, are examples of contagion in inorganic bodies. The solution of an alloy of platinum and silver in nitric acid, when the platinum, that under ordinary circumstances is insoluble in nitric acid, takes on the action that is transmitted through the atoms of silver, is a striking instance of contagion.

In organic substances the action of contagion is very obvious. We know that putrefaction is much hastened by placing in contiguity with a substance prone to undergo that change a portion, however

* Refer to examples already given.

small, of any substance that is already undergoing putrefactive disintegration.

Assimilation is very likely due in a great measure to contagion. By the action of this law we may explain how matter in the very process of putrefaction, and about to be disintegrated and transmuted into its inorganic elements, is caught back, as it were, into the stream of life. We can thus understand the action and the purpose of the countless swarms of the lower forms of insect life. The entire process of digestion, from the first mingling of the food with the saliva until, after a prolonged contact with living matter, the once dead organic material mingles in a highly vitalized state with the general blood current, is strikingly suggestive. This revitalization of dead organic matter and the building up into living tissue appear to be a gradual process. No physiologist has been enabled to point out the special stage in the digestive process where vitality commences; but we do know that during the whole process, the organized material is everywhere in contact with living tissue. It would perhaps be not too speculative to claim for contagion the rationale of the process by which each organ, taking as its pabulum a portion of the general blood current, imparts to the molecules of that fluid its own action, and thus repairs its functional waste.

It has been already stated (page 95) how the extension of molecular motion by contact has been

made available in modern surgery, and explains the mode in which the small oases of skin tissue gradually cover over the desert of ulcer.

When we commence to inquire into the part that contagion may perform in the processes of disease, its action meets us on the very threshold.

Mr. Simon writes—"What is the meaning of the ordinary 'sympathetic' diffusion of inflammatory excitement in the body? When, for instance, in order to make an issue, we burn (say with moxa) a piece of skin the size of a sixpence, why do the surrounding textures to the size of half-a-crown show that they have been disturbed? Why, when inflammation is produced, say in the skin and cellular membrane of a finger by the impaction of a splinter of wood, does not this inflammation confine itself to the particles of texture in contact with the wood? Why does it go on spreading hour after hour in wider and wider circles, dying away as vaguely in the distance as the undulations of ruffled water? And why, three feet off, do the lymph glands in the axilla swell and grow painful and tender and not unfrequently suppurate? Here are two striking facts of inflammatory contagion; on the one hand, as regards the spread of inflammation according to a continuity of tissue, the fact can scarcely be stated in any other etiological form than this. That such particles as are directly affected by causes of inflammation, become capable

of exciting inflammation in particles with which they have contact.”*

Cases are familiar to physicians in which tubercles deposited in the lungs remain quiescent for an indefinite time, and the lung tissues may remain uninjured by their presence for a long period of years. But when from any cause the tubercles take on a disintegrative action, such action is extended by contagion to those portions of lung with which they have contact, and continues spreading until life is destroyed.

That the extension of disease by contagion is an extension of molecular disintegration, is further proved by the experiments of Ricord, who affirms “that syphilitic pus taken from a primary sore is not contagious in its latter stages, but only when the ulcer is extending or stationary; for during the process of reparation or cicatrization he found inoculation produce none of its usual effects.”†

Although contagion forms so striking a feature in the phenomena of zymotic diseases, we are forced to admit that its action is strictly in accordance with a physical law common to all matter in a state of motion, and that the attempt to prove the specificity of zymotic diseases will not be strengthened by a classification that isolates them under the term of “communicable diseases.”

* Holmes's Surgery: Simon, Art. "Inflammation."

† Williams's Elements of Medicine.

CHAPTER VI.

CONTAGION AS A DETERMINING CAUSE.

Causes that determine the Form of Zymotic Disease in the Individual—General Determining Cause, the Source from which the Contagion was Incurred—Modifying Causes—Cases.

WE must accept as a general law (not without exceptions), in zymotic disease, that when the disease is communicated from individual to individual, the diseased action is continued in the same direction as that from which it was received, and this law applies whether the contact be mediate or immediate. As was long since pointed out by Dr. Budd in the case of the transmission of enteric fever by mediate contact, the cases must be looked upon as indirect continuation of intestinal canal, and so in the case of the other varieties of zymotic disease, the transmission of the disease in the same form from individual to individual must be explained in a similar manner. The various individuals subjected to the diseased action must be looked upon,

as regards the texture attacked, as mere extensions of surface or of substance, and the diseased action is continued from the texture of one individual to the corresponding texture of another, by the same law of contagion as obtains when the disease spreads from portion to portion of texture in the body of the person first attacked.

The general law that individuals not subjected to the original cause of disease, but incurring it by communication with others, manifest the disease in the same form as it exhibited in those from whom it was received, is frequently modified by causes special to the individual; and cases may be cited that show this fact in a very marked manner, thus proving that amongst the causes that determine the type of zymotic disease the idiosyncrasy of individuals must be allowed a place. The mode in which the exceptional condition of a patient shapes the exhibition of zymotic disease is forcibly illustrated in the history of puerperal fever. Dr. Braxton Hicks, in his masterly summary of the causes of puerperal fever, published in the twelfth volume of the Transactions of the Obstetrical Society, has brought forward many valuable illustrations of this fact.

Dr. Hicks gives many cases of parturient women who, subjected to the contagion of scarlatina, manifested the disease in a manner more or less modified by their special condition.

CASE I.

“ Mrs. H., æt. thirty-two, seventh confinement.

“ Scarlatina had been constantly in the house (a new one) for two months before her labour. She would not leave it, contrary to the advice of her medical attendant. The fever was mild in the children. Her delivery was very rapid, being over before her medical man could reach her. She went on well till the third day, when severe rigors occurred with intense headache, and rapid (140 per minute) fluttering pulse. From this she somewhat rallied, but the pulse never fell below 110, generally was 120 for the remainder of her life. On the fourth day there was diarrhœa and headache, and the scarlatina rash began to appear on face and neck: the lochia scanty—no vomiting, or pain in the uterus, &c. There was a tremor and nervousness in her manner, but no delirium. She continued fluctuating somewhat in this state, better or worse, for ten days, when the tongue was becoming browner, the diarrhœa still being troublesome, with a tendency to vomit at times. The abdomen was tympanitic. There was then tenderness in lower abdomen, especially in left groin, where a distinct thickening was felt (probably cellulitis). There was evident tendency to sinking, she was irritable and restless, but dozing. The pulse was rapid and fluttering,

which condition was relieved by large doses of etherial stimulants. There were occasional sweatings. She died on the fifteenth day after delivery. The rash behaved as in an ordinary case of scarlet fever." In this case the proximate cause of the disease being scarlatina, the symptoms were modified by the puerperal condition.

CASE II.

"Mrs. ———, multipara, an excitable woman, was delivered naturally. On the third day pain commenced in the uterine region, particularly in the right groin. Some excitement of mind was also present, and sleeplessness; the pulse was at 120, and she was thirsty. The lochia was scanty and offensive, and the milk was scanty throughout the case. She continued much the same, though with less heat, for two days more; when, on the sixth morning, she was attacked with most violent diarrhoea, the pulse rising to 134 per minute and very feeble. She felt great exhaustion and refused food; there was however no sickness. The pain was severe in the right groin, and it was very tender on pressure; in the other part of the abdomen she could bear pressure fairly. The diarrhoea ceased towards the end of the sixth day, and she rapidly sank during the night; she had no rash. The day after her death two of her children were attacked by malignant scarlatina and shortly died."

In the above case the subsequent family history indicates scarlatina as the proximate cause, but the special condition of the patient so altered the character of the disease that the symptoms peculiarly appertaining to scarlatina were altogether absent. There was no rash, and no sore-throat.

CASE III.

“Mrs. ——, multipara, had been confined three days, when rigors succeeded by feverish symptoms came on. She was quickly delirious and suffered from vomiting and diarrhœa; furred brown tongue. The symptoms rapidly became typhoid, and she died on the fourth day of the attack. No rash was discernible on her.

“There were three sources from which she could have been infected by scarlet fever:—

“1st. Her medical man was attending cases on the same day. 2nd. Her husband was a ragged-school teacher, and was at the time visiting all sorts of contagious cases. He attended upon her as more or less nurse through her confinement. 3rd. Her medical man afterwards recognised the nurse. She had been attending scarlatina cases before she went to the above-reported labour.”

Here scarlatina is indicated from three distinct sources as the probable proximate cause, yet there was no rash, no sore-throat; the tongue not red,

but brown, and it is distinctly stated that the symptoms were typhoid.

CASE IV.

The following is a case where a parturient woman submitted to the contagion of scarlatina showed pyæmic symptoms that ultimately proved fatal.

“ Mrs. B——, multipara, in poor circumstances. For some time before her labour her children had been ill with scarlet fever, who were in her bed at the time of her labour, both before and after. She had an easy labour, but shortly after was attacked by feverishness and delirium. Pulse 120. The tongue was white and furred, and there was severe thirst; no diarrhœa, but irritable stomach. She remained in this state for five days, when they subsided; but at this time there was a deep swelling above the right elbow. I did not see her for some days, when I found her much worse: delirious, deaf, vomiting, tongue red and glazed, very thirsty, but unable to retain the fluid; the arm much more inflamed. She was removed into Guy’s Hospital for better nursing, and shortly began to improve; the redness and swelling in arm subsided, but another over left forearm was noticed, but the skin over was not inflamed. However, the deafness went off, her appetite returned, and at the end of a month after delivery she was rapidly improving, when a severe loss of blood occurred from the

uterus at her first menstrual period, reducing her excessively. She appeared, however, to be slowly recovering, when she suddenly died. On the third week of her illness she was covered universally by a rash like erythema. It lasted three days and was unattended by any additional symptoms."

CASE V.

In the following cases, where the proximate cause was respectively erysipelas and decomposing animal matter, the form of the zymotic was determined by the peculiar condition of the patient.

"Mrs. W., æt. forty. In her fifth confinement; easy delivery. Seized with shivering on the third day; succeeded by pyrexia, and pulse 120 per minute, which continued with slight confusion of mind till the sixth day; when tympanitis, delirium, and typhoid symptoms supervened, accompanied by diarrhœa and vomiting. Saw her on this day and found her pulse very feeble and rapid, temperature low; hands and feet cold. In fact she was sinking. The tongue was put out tremblingly and uncertainly, and she was partially wandering in mind. She died soon after. *

"On inquiry as to the health of the other inmates, I found that one of the daughters, about fourteen years old, had on that day been attacked by erysipelas of the face, extending over more than one-half already. She had been frequently in her mother's room."

CASE VI.

“Mrs. S., multipara.—Was delivered naturally in a house into which the stable and cattle shed entered as a part. The house was very badly drained, the bedroom smelling strongly of ammonia. She went on well for three days, when diarrhœa set in with cough and rapid pulse (120 per minute). A measles-like rash accompanied this condition without any other symptoms of measles or scarlatina. She gradually lost these symptoms, but was much depressed by the attacks; but in a few weeks she was well again.

“I could not find any account of measles or scarlatina having occurred in the house or neighbourhood previously.”

In the course of the remarks with which Dr. Hicks introduced his valuable series of 89 cases he makes this significant statement:—

“The consideration of these and similar facts leads me to take this opportunity of dwelling on the importance of recognising the variability of diseases. There is a natural tendency when engaged in defining disease to notice and adhere to the more distinct and typical forms, while we ignore all the less marked or anomalous kinds. But there are many diseases, particularly the exanthemata, which are not rigidly defined. Every practitioner has seen many cases which either from possessing symptoms

common to others or failing in some of those proper to them, cannot be strictly placed under any absolutely limited head; these we dismiss from our minds as anomalous cases and then forget them."

Cases of zymotic disease are frequently met with in which the proximate cause can be distinctly traced, and in which the manifestations of the zymotic action differ in the individuals exposed to the same influence.

In the third volume of the "Transactions of the Edinburgh Medical and Chirurgical Society," Dr. Gibson gives the following case:—

CASE VII.

"An infant was attacked with erysipelas of one foot. The mother was soon after seized with the disease in the head and face. The wet nurse of the infant was taken with pneumonia and was removed home, a distance of four miles. Her father, who had had an injury to the head, was soon after seized with erysipelas of the scalp and died. Her sister had low fever with sore-throat, and two children in the same house were attacked with croup and died."

CASE VIII.

Mr. Erichsen, in his "Art and Science of

Surgery," mentions a case of six students who were infected by the body of a patient who had died of peritonitis. Two had suppuration of the areolar tissue under the pectoral muscles and in the axilla, one a kind of maniacal delirium, a fourth had typhoid fever, and the remaining two were seriously indisposed.*

CASE IX.

In the month of December, 1870, five of the clerks in a Bread Street warehouse were simultaneously seized with feverish symptoms and obliged to leave their employment. These men all slept in the same building and worked in the same room. Three of them, after suffering for a week with mild fever symptoms, were enabled to resume their occupation. A fourth had a severe attack of small-pox and was taken to the Small-Pox Hospital. The fifth lay for a month in St. Thomas's Hospital with symptoms of enteric fever. All these men had been vaccinated during infancy.

"A very close relationship appears to exist between scarlatina and diphtheria and one variety at least of enteric fever. All are for the most part autumnal diseases, and they may be observed to

* The case may be cited of the late Mr. Potter, of U. C. H., who died of dissection wound fever. The nurses who attended upon him subsequently suffered from erysipelas.—*Erichsen's Surgery*.

increase and decrease together, and all appear to arise spontaneously out of the same conditions. Stöber, Löschner, and Friedleben maintain that scarlatina and enteric fever prevail epidemically in an inverse ratio to each other, the one prevailing in proportion as the other declines."

"I have known several instances of scarlatina affecting one member of a family and enteric fever another simultaneously."

"The day before C. B.* came into the hospital her brother, aged fourteen, was admitted with scarlatina in its most marked form."—*Dr. John Harley, Russell Reynolds' System of Medicine*, Art. "Enteric Fever."

CASE X.

Mr. Paget, in his lectures on surgical pathology, p. 373, gives the following case, on the authority of Mr. Huxley.

"One of the crew of H.M.S. *Rattlesnake*, after slightly wounding his hand with a beef-bone, had suppuration of the axillary lymphatic glands, with which typhoid symptoms and delirium were associated, and proved fatal. His illness began the day after the ship left Sydney, where all the crew had been remarkably healthy. A few days after his death the sailor who washed his clothes had similar

* A typical case of enteric fever.

symptoms of disease in the axilla, and for four or five months he suffered with sloughings of portions of the areolar tissue of the axilla, arm, and trunk on the same side. Near the same time a third sailor had diffuse inflammation and sloughing in the axilla, and after this the disease ran, in *various forms*, through the ship's company, between thirty and forty of whom were sometimes on the sick list at once. Some had diffused cellular inflammation, some had inflammation of the lymphatic glands of the head, axilla, or lower extremities; one had severe idiopathic erysipelas of the head and neck, another had phlegmonous erysipelas of the hand and arm, after an accidental wound; others had low fever, with or without enlargement of glands. Finally the disease took the form of mumps, which affected almost everybody on board. The epidemic lasted from May to July. The ship was at sea the whole time, and in the greater part of it in the intense cold of a southern winter."

The following cases, related by Mr. Metcalfe Johnson (*Medical Times and Gazette*, December 10th, 1870), are instances where of several persons subjected to the contagion of one form of zymotic disease, some manifest disease in another form:—

"A. Z., æt. three. Rash distinct, raised vesiculæ; tongue slightly furred; catarrhal symptoms; no sore-throat; fever slight. Temperature 98°."

"B. Z., æt. two. Rash uniform red; swelling of

submaxillary glands. Temperature $101^{\circ}4$. Throat became ulcerated; skin peeled off."

These two children occupied the same bed, and were ill at the same time.

"Miss A. W., æt. five. Had measles when a baby. Vomiting, diarrhœa, delirium in sleep; submaxillary glands swollen; tonsils inflamed, afterwards ulcerated. Rash on legs in patches; more diffuse and scarlet on arms."

"Master J. W. sleeps in same bed with the above. Rash distinct, prominent, crescentic patches. Evidently measles. No swelling of submaxillary glands."

"A. B., æt. nine months. Distinct measles."

"R. B. Ulcerated throat, tongue red, enlarged papillæ. The whole family had a severe attack of scarlatina."

These cases scarcely need comment; their significance must be at once apparent. If, as is generally held, each form of zymotic disease is a specific disease caused by a special poison, the occurrence of such deviation from this law as is shown in the cases recited above would be quite incapable of explanation, and we cannot doubt that when attention shall be directed to this subject it will be found that similar cases are by no means uncommon. In the present state of medical science, if of many persons subjected to the contagion of a particular variety of

zymotic disease some individuals manifest the disease in a different form, the fact would probably pass unnoticed, and the occurrence of these different diseases would be looked upon as mere coincidences, standing in no relation of cause and effect.

CHAPTER VII.

CONCLUSION.

IN the foregoing review of zymotic diseases, the salient point that cannot fail to strike us is the strict correlation that exists between the different varieties. They all present in their symptoms the unvarying characteristics of inflammatory destruction of tissue. Varying in accordance with the special texture primarily affected, the great fact remains, that the phenomena presented during the whole course of the disease are always the phenomena that inflammation of such structure would have evolved under ordinary circumstances. The redness, heat, and swelling of the mucous membrane during an attack of measles differ, in no manner, from the redness, heat, and swelling of the same membrane under an attack of ordinary catarrh. The disintegrated blood circulating through the various organs during an attack of typhus produces the same symptoms as when the disintegration is the result of pyæmic poisoning. So strictly analogous are the symptoms, that it would be difficult for even an

accurate observer to diagnose the disease with certainty, unless furnished with the previous history of the case.

The pustules of small-pox do not differ in any essential particular from the pustules produced when any irritant is applied to the cutis sufficiently powerful to set up inflammation that proceeds to pustulation.

The general symptoms that usher in and accompany all the varieties of zymotic disease are further evidence of the strict correlation that exists between them. At a certain period after submission to the noxious influence, the normal resistance of healthy action is overcome, and a train of symptoms sets in that afford proof of a great increase of tissue change, heightened temperature, quickened circulation, general functional derangement. In short, those symptoms to the sum of which we apply the term fever; and so analogous are the phenomena of this stage that it is most difficult to predicate which is the texture that will ultimately prove to have been the point attacked. The divergence that occurs at a later stage, when the characteristic symptoms are set up with which we are all familiar and which enable us to give a name to the disease, is due to the special structure exhibiting during its disintegration the symptoms that are pathognomonic.

The examination after death of the bodies of those who have died of zymotic disease might be expected

to afford some clear indication of the mode of action of the death-producing cause. If the disease have been the result of some special poison introduced from without and indefinitely multiplied within the body, we should expect to find amongst the tissues abundant evidence of heterologous products as the necessary result of the exotic importation. But however minute our examination, we seek in vain for the trace of any organisms, animal or vegetable, that can account for the origin of the disease or for its fatal termination. The dead-house fully confirms the experience gained at the bedside. We find the organs especially affected, softened, disintegrated, showing evidence of having undergone a process of destructive inflammation. The diseased products that we find amongst the affected tissues are such as we know are to be found as the natural result of the disintegration of such tissues—fatty matter, oil globules, granular matter, spoilt blood corpuscles—just such exuvia as would result from the disintegration of the same tissues under the ordinary processes of inflammation. We find also at the post-mortem examination of the various forms of zymotic disease the same strict correlation as is evidenced in the symptoms—in all we find that the diseased process has been an act of textural inflammation resulting in textural disintegration and death, varying in appearance in accordance with the texture primarily affected. The intestinal gland ulcerated

and disintegrated by enteric fever cannot be distinguished from a similar gland in which ulceration has resulted from ordinary inflammation.

The correlation that has been contended for is still more evident if we consider that the various forms of zymotic disease are (as has been shown in the statistics) in a great degree mutually complementary; that the sum of zymotic diseases is in some measure a fixed quantity; and that the prevalence of any one form depends upon causes that may be looked upon as in a certain degree accidental; and the argument is further strengthened by the cases cited to prove that it not very unfrequently occurs that a person subjected to the contagion of one form of zymotic disease exhibits in his own person a different variety of the disease as a consequence of such contagion.

There are two features in zymotic disease that have always attracted much attention and would appear at first sight to be peculiar to those forms of disease, and to separate them from other forms of diseased action; these are—1st. The comparative immunity conferred by one attack of zymotic disease against a renewed attack of the same disease. 2nd. The mode in which zymotic diseases are communicated from individual to individual by contagion.

An endeavour has been made in the text to examine the laws that govern immunity and contagion, and to explain their real significance in

relation to zymotic diseases. It has been shown that the immunity is not absolute; that it is not confined to zymotic disease; that it is accordant with a well-recognised physiological law; and that it applies to all forms of textural irritation, zymotic and other.

No argument can be necessary to prove that the principle of contagion is not confined to zymotic disease; as we have seen, it is a law of such large and extensive application that its influence is almost as general as gravitation; it applies to all forms of motion, to all forms of matter, organic and inorganic; the processes of growth and of repair are influenced by it quite as much as the actions of disease; its influence in disease is not confined to those diseases classed as zymotic, and if its effects are more commonly noticed in those diseases it is only because they offer, from the structures affected, more occasions for the exercise of this widely prevailing influence. If hearts and livers and spleens were as easily brought into contact as skins and bowels, it would be seen that contagion is in medicine as in physics a principle of universal application.

With a view to determine the causation of zymotic diseases an examination has been made into the histories of the epidemics that have at various periods prevailed, and the facts these histories disclose teach us that epidemics have prevailed from

time to time from the earliest period when men were aggregated in large numbers; that the condition necessary for their genesis was the presence of large quantities of decaying organic matter; that in those countries whose physical conformation necessitated the periodic accumulation of these large masses of organic refuse some form of zymotic disease becomes endemic; and that occasionally, from causes known or unknown, such endemic diseases assume the form of epidemic, spreading especially amongst those whose power of resistance was lessened by previous disease or famine or unfavourable hygienic surroundings; that under these circumstances the epidemic spread over enormous tracts of land; and that when meeting with conditions analogous to those in which it had its origin it became endemic in its foreign home, like the Egyptian plague formerly with us, and the Asiatic cholera recently in Russia.

When we examine into the origin of those more limited outbreaks of the forms of zymotic disease that are common in this country, and with which we are more familiar, we find a great analogy in the circumstances: these diseases are most common in the autumn months, when there is necessarily a large quantity of decaying organic matter. The outbreak can in many cases be traced to actual contact with such decaying refuse, and it spreads with the greatest rapidity and acts with the greatest virulence amongst

those whose natural power of resistance is lessened by privation or otherwise.

The actual effect of decaying organic matter in producing certain forms of zymotic disease is constantly seen in those cases where the point invaded is more than usually obvious, and the subsequent phenomena can be carefully watched. Such are the cases arising from dissection wounds, cases of puerperal fever, and erysipelas.

The conclusions that we have endeavoured to establish are the following:—

That the various forms of zymotic disease are strictly correlated, that they are essentially processes of textural inflammation, tending to textural disintegration.

That they vary in their phenomena in accordance with the special texture primarily affected.

That in their causation the correlation is as evident as in their symptoms.

That the cause is invariably the presence of decaying organic matter.

And that the mode in which this decaying matter acts, is by overcoming the resistance of the natural actions of repair and growth, and inducing in their stead a process of inflammatory disintegration.



The first part of the chapter is devoted to a discussion of the general principles of the theory of the firm. It begins with a definition of the firm as a collection of individuals who are organized to produce goods and services. The author then discusses the various forms of the firm, such as the sole proprietorship, the partnership, and the corporation. He also discusses the role of the firm in the economy and the importance of the firm's internal structure.

The second part of the chapter is devoted to a discussion of the theory of the firm. It begins with a discussion of the firm's production function, which is a relationship between the inputs used by the firm and the output produced. The author then discusses the firm's cost function, which is a relationship between the inputs used by the firm and the total cost of production. He also discusses the firm's profit function, which is a relationship between the inputs used by the firm and the profit earned by the firm.

The third part of the chapter is devoted to a discussion of the firm's behavior. It begins with a discussion of the firm's production decisions, which are decisions about the level of output to produce. The author then discusses the firm's cost decisions, which are decisions about the level of inputs to use. He also discusses the firm's profit decisions, which are decisions about the level of inputs to use and the level of output to produce.

