On some points in the pathology and treatment of typhoid fever : delivered at the Royal College of Physicians of London / by Wm. Cayley.

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Cayley, W. 1836-1916.

Publication/Creation

London : J. & A. Churchill, 1880.

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CAYLEY

ON TYPHOID FEVER

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With the author's compliments.

CROONIAN LECTURES

ON SOME POINTS IN THE PATHOLOGY AND TREATMENT OF TYPHOID FEVER

Delivered at the Royal College of Physicians of London

BY

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OF MEDICINE

LONDON
J. & A. CHURCHILL, NEW BURLINGTON STREET

1880

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CROONIAN LECTURES.

ON SOME POINTS IN THE PATHOLOGY AND TREATMENT OF TYPHOID FEVER.

LECTURE I.

Etiology.

MR. PRESIDENT AND GENTLEMEN,—

In taking for the subject of these lectures the Pathology and Treatment of Typhoid Fever, I fear I may seem to have chosen one which has been completely exhausted, and about which, therefore, little or nothing new remains to be said; its general pathology, its mode of origin and dissemination, its distinction from other diseases, have indeed been thoroughly investigated, owing in no small degree to the researches of eminent Fellows of this College, among whom I need only enumerate the names of Stewart, Jenner, and Murchison. Of late years, too, there has been a pretty general agreement as to the principles on which the disease should be treated; but, notwithstanding, upwards of 73,000 persons

died during the past nine years in England alone of typhoid fever.

In the face, then, of this great mortality, I think it may not be without some degree of interest and advantage if we again pass in review some of the received opinions as to its nature and treatment. I do not, however, propose again to discuss the distinction between typhoid and other fevers, or to give any clinical account of the disease, but to confine myself to the consideration of some points about which more or less difference of opinion and practice still prevails. And I shall first consider the causation and mode of dissemination; secondly, some points in the pathology; and, lastly, the treatment.

In proceeding to consider the mode of origin and propagation of typhoid fever, we are at once arrested by the question, What are the nature and properties of the poison which is supposed to give rise to the disease?

Now, the typhoid poison has up to the present time eluded all attempts to isolate it or to demonstrate its nature either by microscopical examination or chemical analysis; we are only conscious of its existence by the effects which it produces on the human organism. Nevertheless, some of its properties are known with tolerable certainty: First, when introduced into the systen it multiplies; secondly, it is contained in the alvine discharges of persons suffering from the disease; thirdly, it

retains its activity for an indefinite time after it has passed out of the body, when placed under favourable conditions, these conditions being the presence of decaying animal matter and moisture. Hence its usual habitats out of the body are drains, sewers, cesspools, dung-heaps, wet manured soils. And there are some grounds for supposing that in these situations also it may possess the power of multiplying. Lastly, in all probability it is particulate, and not either liquid or gaseous.

The actual nature of the poison,—whether it be, according to the hypothesis most generally accepted, some kind of fungus, or microzyme, or protoplasm, in a word, a *contagium vivum*, or whether, as maintained by others, it is some derivative of albumen, capable of exercising a catalytic action on other albumen,—I do not propose to discuss, as it is a question at present rather of theoretical than practical interest, and it is one, moreover, for whose final determination the data are hardly yet sufficient.

A subsidiary question to this, but one of considerable practical importance, is whether the poison can be generated *de novo* from decaying organic matter, whether it be pythogenic as was so ably maintained by Dr. Murchison, or whether it can only arise by continuous propagation, as was maintained no less ably by Dr. William Budd.

The arguments on both sides may be very briefly stated.

In favour of its origin *de novo*, it is asserted that

typhoid fever has often broken out in isolated situations—as solitary farm-houses, far removed from, and holding no communication with, places where the disease exists; and many such instances are given by Dr. Murchison. On the other hand, it has been proved incontestably by numerous examples, both in this country and on the Continent, that all the conditions supposed to be required for its generation may be present for an indefinite time—as percolation of sewage into wells supplying drinking-water,—and yet the disease does not show itself till the poison is introduced by the arrival of an infected person, when an outbreak at once takes place.

Now, I would submit that this latter argument far outweighs the former; for otherwise, if it be proved that all the conditions necessary for the origination of the poison are present, as shown by its subsequent development when the germs are introduced, and yet it does not develop, we should have to admit that the same causes are not always followed by the same effects.

The instances in which persons in the latent stage of typhoid fever, or actually ill with it, have carried the disease to distant places, and caused it to spread, are so numerous, that I believe that this mode of propagation is now universally admitted; the communication of the disease taking place not by direct contagion from the sick to those brought into immediate contact with them, but by the ordinary mode of sewage contamination.

I will quote in illustration three instances which have occurred in this country.

One is the famous epidemic at Over Darwen. The water-supply pipes of the town were leaky, and the soil through which they passed was soaked at one spot by the sewage from a particular house. No harm resulted till a young lady suffering from typhoid fever was brought to this house from a distant place: within three weeks of her arrival the disease broke out, and 1,500 persons were attacked.

Another well-marked instance occurred at Calne. A laundress occupied the middle one of a row of three houses supplied by one well, into which the slop of the laundress's house leaked. She, on one occasion, received the linen soiled by the discharges of a case of typhoid fever, and after fourteen days cases occurred in all three houses.

At Nunney a number of houses received their water-supply from a foul brook contaminated by the leakage of the cesspool of one of the houses, but no fever showed itself till a man ill with typhoid came from a distance to this house. In about fourteen days an outbreak of fever took place in all the houses.

Many equally striking influences might be quoted from foreign sources; I will, however, only adduce the well-known one of Lausen, as it illustrates some other points of great importance in the etiology and prophylaxis of the disease.

Lausen is a village through which I have no doubt

most of my hearers have passed, as it lies on the railway between Basle and Olten, shortly before coming to the great Hauenstein Tunnel. It is situated in the Jura, in the valley of the Ergolz, and consists of 103 houses, with 819 inhabitants; it was remarkably healthy, and resorted to on that account as a place of summer residence. With the exception of six houses, it is supplied with water by a spring with two heads which rises above the village at the southern foot of a mountain called the Stockhalder, composed of oolite. The water is received into a well-built covered reservoir, and is distributed by wooden pipes to four public fountains, whence it is drawn by the inhabitants. Six houses had an independent supply—five from wells, one from the mill-dam of a paper factory.

On August 7, 1872, ten inhabitants of Lausen, living in different houses, were seized by typhoid fever, and during the next nine days fifty-seven other cases occurred, the only houses escaping being those six which were not supplied by the public fountains. The disease continued to spread, and in all 130 persons were attacked, and several children who had been sent to Lausen for the benefit of the fresh air fell ill after their return home. A careful investigation was made into the cause of this epidemic, and a complete explanation was given. Separated from the valley of the Ergolz, in which Lausen lies, by the Stockhalder, the mountain at the foot of which the spring supplying Lausen rises, is a side

valley called the Furlenthal, traversed by a stream, the Furlenbach, which joins the Ergolz just below Lausen, the Stockhalder occupying the fork of the valleys. The Furlenthal contained six farm-houses, which were supplied with drinking-water, not from the Furlenbach, but by a spring rising on the opposite side of the valley to the Stockhalder.

Now, there was reason to believe that, under certain circumstances, water from the Furlenbach found its way under the Stockhalder into one of the heads of the fountain supplying Lausen. It was noticed that when the meadows on one side of the Furlenthal were irrigated, which was done periodically, the flow of water in the Lausen spring was increased, rendering it probable that the irrigation water percolated through the superficial strata, and found its way under the Stockhalder by subterranean channels in the limestone rock. Moreover, some years before, a hole on one occasion formed close to the Furlenbach by the sinking-in of the superficial strata, and the stream became diverted into it and disappeared, while shortly after the spring at Lausen began to flow much more abundantly. The hole was filled up, and the Furlenbach resumed its usual course.

The Furlenbach was unquestionably contaminated by the privies of the adjacent farm-houses, the soil-pits of which communicated with it. Thus, from time immemorial, whenever the meadows of the Furlenthal were irrigated, the contaminated water of the Furlenbach, after percolation through the superficial

strata and a long underground course, helped to feed one of the two heads of the fountain supplying Lausen. The natural filtration, however, which it underwent rendered it perfectly bright and clear, and chemical examination showed it to be remarkably free from organic impurities; and Lausen was extremely healthy and exempt from fever.

On June 10 one of the peasants of the Furlenthal fell ill with typhoid fever, the source of which was not clearly made out, and passed through a severe attack, with relapses, so that he remained ill all the summer; and on July 10 a girl in the same house, and in August a boy, were attacked. Their dejections were certainly, in part, thrown into the Furlenbach; and, moreover, the soil-pit of the privy communicated with the brook. In the middle of July the meadows of the Furlenthal were irrigated as usual for the second hay crop, and within three weeks this was followed by the outbreak of the epidemic at Lausen.

In order to demonstrate the connexion between the water supply of Lausen and the Furlenbach, the following experiments were performed:—The hole I mentioned above as having on one occasion diverted the Furlenbach into the presumed subterranean channels under the Stockhalder was cleared out, and 18 cwt. of salt were dissolved in water and poured in, and the stream again diverted into it. The next day salt was found in the spring at Lausen. Fifty-six pounds of wheat flour were then poured into the hole,

and the Furlenbach again diverted into it; but the spring at Lausen continued quite clear, and no reaction of starch could be obtained, showing that the water must have found its way under the Stockhalder in part by percolation through the porous strata, and not by distinct channels.

Besides showing the necessity of the introduction of the specific poison in order to render sewage contamination capable of giving rise to typhoid fever, this case is remarkable as an instance of the extreme dilution to which the poison may be subjected without losing its potency, and also the uselessness of irrigation and any ordinary filtration in separating it or rendering it inert. Here the dejections of two cases of typhoid are thrown into a stream; the water of this stream is used to irrigate extensive meadows; a portion of it sinks through the superficial strata, and probably finds its way into subterraneous channels, and passes through a distance of many thousand feet under a mountain, partly, no doubt, by mere percolation; it then takes an insignificant part in feeding one head of a copious spring, which has another head that is not contaminated, and, nevertheless, it gives typhoid fever to 130 persons out of a population of 800. The dilution must have been almost infinitesimal, unless we assume that a multiplication of the poison took place after its discharge from the intestinal canal of the first two cases—possibly in the reservoir at Lausen.

The apparently spontaneous origin of the disease

in isolated places may, doubtless, be in part explained by the very long time the poison may lie dormant, still retaining its essential properties, and capable under favourable conditions of developing its activity.

The poison, too, may be introduced in quite unsuspected modes. It is well known that the typhoid poison by no means always produces what is usually recognised as typhoid fever. In very many cases its only effect is to cause some malaise, together with slight intestinal catarrh, which is not necessarily attended by diarrhœa. These cases are only recognised as typhoid when they occur during an epidemic, and among persons who have been exposed to infection; but if we may judge by the analogy of scarlatina, diphtheria, and other contagious fevers, where similar slight unrecognisable forms are not uncommon, these apparently trivial cases should be as potent in communicating the disease as the severe well-marked forms. Hence it is quite possible that the typhoid poison may be introduced into a locality by a person whom no one would suspect of being infected with the disease.

It has been ingeniously suggested by my colleague, Dr. Robert King, that the typhoid poison is derived solely from decomposing albumen, which is not present in healthy stools, and that it can therefore only arise from morbid stools which contain albumen, as is the case in some forms of catarrhal diarrhœa, and he has published a case where the poison seemed

to be generated in this manner. This theory might certainly account for long-continued sewage contamination of water without the production of typhoid fever, as the special material from which the typhoid poison is generated might be absent. But it is hardly likely that any such derivative of albumen would remain undecomposed in a drain or cesspool for so long a period as we have reason to believe is the case with the typhoid poison.

We have next to consider how long the typhoid poison can thus retain its activity out of the body in a suitable locality. One of the clearest pieces of evidence on this point is the well-known instance related by Dr. von Gietl. To a village free from typhoid an inhabitant returned suffering from the disease which he had acquired at a distant place. His evacuations were buried in a dunghill. Some weeks later five persons who were employed in removing dung from this heap were attacked by typhoid fever: their alvine discharges were again buried deeply in the same heap, and nine months later one of two men who were employed in the complete removal of the dung was attacked and died. Here we have distinct evidence that the poison retained its powers for nine months.

Dr. Murchison, in his work on Fever, gives an instance in which six cases were spread over a period of eight years. I have recently seen an instance in which an interval of two years occurred without apparently any fresh importation of the poison.

Supposing the germ theory to be correct, there is, of course, no reason why the poison should not preserve its vitality, by continuous propagation, for an indefinite time.

On the whole, though the point cannot be regarded as finally determined, I think the weight of evidence is against the *de novo* origin of the disease.

We have now to consider a question about which much difference of opinion still prevails, viz., the contagiousness of typhoid fever. That the disease is communicable from the sick to the healthy is, I believe, universally acknowledged, the point in dispute being the mode in which the transmission takes place, whether directly by emanations from the patient, or from his fresh evacuations, or indirectly from eating or drinking, or inhaling the emanations from the stools, modified by their having undergone some kind of decomposition or fermentation outside the organism. Although the point in dispute is a narrow one, it is of considerable importance, and erroneous views may lead, on the one hand, to the adoption of unnecessary restrictions, which may seriously incommode the patient and his attendants, while at the same time it is only too likely to cause a neglect of the really essential precautions.

I have myself witnessed an outbreak of typhoid where the belief in its contagiousness—shared, I may say, by the medical practitioners—was so strong as to excite quite a panic, so that difficulty was found in procuring attendants for the sick. In consequence, I

had on one occasion myself to carry a patient from one room to another, and a man who volunteered to help me was regarded as having done something rather heroic. I need hardly say that, at the same time, the utmost recklessness is commonly shown with regard to the real causes of its dissemination.

On the other hand, a disbelief in the direct contagiousness of the disease might, if not well founded, lead to unnecessary exposure of the patient's friends.

The arguments against the direct contagiousness of the disease are in the main these :—First. In hospitals we rarely find that typhoid fever spreads either to the attendants on the sick or to the other patients. When it does thus spread, persons in other parts of the building, who have never been brought in contact with the typhoid cases, are attacked about as frequently as the immediate attendants themselves.

Dr. Murchison has published the evidence on this point afforded by the London Fever Hospital for a period of twenty-three years, up to 1870. During this period 5,988 cases of typhoid were admitted, and seventeen of the resident staff took the disease, but of these seventeen only five were in communication with the typhoid cases, and twelve occurred at a time when there were serious defects in the drains ; twelve patients also admitted for other diseases became infected. But since 1861, when the patients have been so classified that the typhoid cases and the patients suffering from other acute diseases, not fever, are placed in the same wards, not a single instance has

occurred in which the infection has spread to the non-typhoid cases, though the same night-stools and water-closets have been used by both classes of patients, and the use of disinfectants has been exceptional. This shows pretty conclusively that the emanations from the recent stools are not capable of communicating the disease.

Since 1871, 1,447 cases of typhoid have been admitted and treated in the same wards with 692 patients suffering from other diseases, and during this time only three nurses and no patients have acquired the disease. On several occasions, however, cases of scarlet fever lying in other wards have become infected. So far as the London Fever Hospital is concerned, it would seem that the risk of becoming infected with typhoid is rather less in the typhoid wards than in other parts of the building.

The experience of other hospitals is for the most part in accordance with that of the London Fever Hospital.

Liebermeister states that up to 1865, neither at Greifswald, nor Tübingen, nor Berlin, had a single instance been known of the disease spreading to other patients in the hospital, or to the attendants on the sick. The Hôtel-Dieu and La Pitié at Paris, the City of Glasgow Fever Hospital, and the general hospitals of London show much the same result.

During the past year sixty cases of typhoid fever were admitted into the Middlesex Hospital, and six nurses took the disease; but of these six, five were on

duty in the surgical wards and never came into contact, either directly or indirectly, with the typhoid cases. I need hardly say how unlike this is to the behaviour of a contagious disease. I have no doubt but that in all these six cases the disease was due to the condition of the drains of the dormitory.

It is also found that when persons suffering from typhoid fever are removed to distant places, it is the exception, and not the rule, for the disease to spread; and when it does spread, it quite as commonly attacks those who have not been brought into immediate contact with the sick, as it does the attendants themselves. It may, I think, be laid down as absolutely certain, that an epidemic of typhoid is never caused by the disease spreading by direct contagion, as epidemics of small-pox, scarlatina, and typhus are.

On the other hand, it is argued that sometimes in hospital practice, typhoid does spread to the attendants on the sick or the other patients; and, not at all uncommonly, persons suffering from typhoid, when removed to a distance, do communicate the disease to those brought into contact with them.

Thus, in the hospital at Basle, 1,900 cases were admitted in six years; and during this period forty-five residents in the hospital became infected, and a large number in addition suffered from a non-febrile intestinal catarrh which in all probability was due to the typhoid poison. But of these forty-five cases many had never been brought into contact with the patients, and of the nurses it was found that those

were especially liable to be attacked who occupied a particular room where they were exposed to the emanations from a leaky choked soil-pipe. Probably there is no city in Europe, not even Munich, and no hospital, so saturated with the poison of typhoid as Basle and its hospital; and, moreover, the hospital drains are, or were, in a very unsatisfactory condition; and thus there is every reason for believing that the contagion in all these forty-five cases was indirect.

The same argument applies to the spread of the disease in private houses. In the great majority of instances where the fever is imported from a distance it does not spread; where it does, some defect in the sanitary conditions favouring indirect contagion can generally be detected: and it is therefore almost certain that the same is the case in the other instances; otherwise we should have to admit that typhoid fever is sometimes contagious and sometimes not—nay, that during the same epidemic the disease is contagious in one house or institution, and not contagious in another house or institution, though they may be situated close together.

I have seen many instances of supposed direct contagion, but have generally succeeded in tracing them to an indirect source.

A boy was admitted into the Middlesex Hospital under my care, on March 27th of last year, suffering from a very severe attack of typhoid. For several days he lay in an unconscious condition, and during

this time he had very profuse diarrhœa—twelve to twenty liquid motions daily, which were for the most part passed in the bed. In the next bed was a boy aged six, who had been admitted on April 16, with acute renal dropsy and bloody urine. He was kept strictly confined to bed, and never got up to go to the water-closet down which the motions of the typhoid case were thrown. On May 11, when he was convalescing, the dropsy having disappeared and the albumen much diminished, he was seized by typhoid fever, and passed through a moderately severe attack, with a well-marked rash and characteristic symptoms. This at first sight appeared to be a case of direct contagion, but I have no doubt that the true explanation is this:—The bedding of the first patient was constantly kept saturated by his liquid motions, and, though every care was taken to change the linen frequently, it was obvious, from a distinctly fæcal smell which was always present, that the bedding or mattress remained contaminated, and thus time was given for the poison to develop its infectious properties. Another patient in the same ward, admitted for acute rheumatism, was also attacked by the fever. He occupied a bed on the opposite side, and never came near the first case, but, being convalescent, he used the water-closet down which the motions of the typhoid case were thrown; and it so happened that at this time the closet was out of order, the contents were retained, and an offensive smell was constantly present. Hence, I think, there can

be no doubt but that he was infected by the emanations from the evacuations of the first case.

The first case affords an example of a very common mode of infection; and many of the cases attributed to direct contagion are really produced in this way, especially among the lower orders or where skilled nurses are not employed. The bed-linen or mattress, or the patient's own person, become soiled by the liquid alvine discharges. These soon dry, and are left undisturbed, and in a short time the poison develops its infectious properties. Soiled linen is well known to possess very active contagious powers, and to retain them for a long time.

If these views are correct, it becomes an important subject of inquiry as to how soon the stools acquire contagious properties. I am not aware of any observations which would enable us to decide this point with certainty, but the time is probably a very short one. The frequency with which washerwomen have been infected by soiled linen points to a very short duration, as such linen is seldom retained for any length of time. Some facts which have come under my own observation, though not conclusive, are also in favour of the period being very short.

At the Middlesex Hospital it was formerly the custom to preserve the stools of cases of typhoid, which the physician wished to inspect, in pans which were placed in the water-closets of the wards. Now, the time during which such stools would be so kept would hardly ever exceed twelve hours; neverthe-

less, several instances have occurred in which other patients using these closets have become infected. Supposing the cause of the infection to have been these reserved stools, we should have a period of not longer than twelve hours for the contagious properties to become developed. The experience of the London Fever Hospital pretty conclusively proves that the fresh stools are innocuous.

The practical conclusion to be drawn is, I think, this: that we have in all cases a few hours during which it lies in our power to render the poison innocuous, and to prevent the spread of the disease by direct infection.

Admitting, then, that in all probability typhoid fever never arises *de novo* from decomposing sewage, and that whenever it spreads from the sick to the healthy it is by a process of indirect infection, it still remains to be considered whether this is the only way in which the disease may originate or become disseminated. To this question I think we are compelled to give a negative answer. Several outbreaks have now been recorded which have been ascribed to eating diseased meat. And although in some of these considerable doubt has been expressed as to the real nature of the disease, in others I think the evidence is quite decisive.

The first of these outbreaks on record is the well-known one which occurred at Andelfingen, in the canton of Zurich, in 1839.

On July 10 of this year the Local Choral Society

held a festival meeting, after which 513 persons of all ages sat down to a cold collation, consisting chiefly of veal and ham. It was noticed at the time that neither the veal nor the ham were perfectly good. Some portions of the former had a greenish colour and a disagreeable smell; the ham also is said not to have tasted well. But most of the guests observed nothing amiss, and ate heartily. Of the 513 persons who partook of this collation, 421 were subsequently attacked by an acute febrile disease, which was regarded at the time as typhoid. Thirty-four inhabitants of Andelfingen were also attacked who had taken no part in the choral festival, but all of whom, it was ascertained, had been supplied by the same butcher who had furnished the veal and ham for the festival.

The day after the festival there was a wedding in the neighbourhood of Andelfingen, at which 15 persons were present, only one of whom had attended the choral meeting. The meat—veal and beef—for the wedding breakfast was supplied by the same butcher. Of these 15 persons 11 were attacked.

The period of incubation of this epidemic was very variable. A few were seized with nausea and vomiting on their way home, but this was ascribed to their having drunk too much wine. Out of 230 cases in which the incubation period was ascertained, 43 were taken ill during the first five days, 123 during the second five days, 48 during the third five days, and 16 during the fourth five days, 6 being attacked on the nineteenth day.

The symptoms were those of severe gastro-intestinal irritation, with high fever, delirium, stupor, congestion of the lungs, and great prostration. No rose rash was observed, but in some cases there were petechiæ. The duration of the milder cases was about eight days; of the severer ones, three to four weeks. Convalescence was slow, and often the hair fell out. The mortality was slight, and on post-mortem examination in some cases there were infiltration and ulcerations in the lower part of the ileum, with enlargement of the spleen; in others these changes were not observed.

There can be no doubt as to the meat having been the cause of the epidemic, as only those persons who had partaken of it were attacked, while a very large number of persons from all parts of the canton were present as singers or spectators who did not share in the collation, and they all escaped. But great doubts have been expressed as to really whether it was typhoid fever, or a form of poisoning resembling sausage poisoning.

In 1845 a similar epidemic occurred at Thalweil, though on a much smaller scale. The particulars of this I have failed to obtain, as the journals containing it are out of print. But 8 out of 10 persons were attacked with what was believed to be typhoid fever after eating bad veal.

A third epidemic due to the same cause, and which was undoubtedly typhoid fever, occurred at Kloten, a place about seven miles north of Zurich, in 1878.

On Ascension Day, June 30, a festival was held of the united choral societies of the district, together with choirs from Zurich and Winterthur. The festival collation was furnished by the landlord of one of the inns, who himself was attacked by the epidemic. The food supplied consisted of ragout of veal, roast veal, and veal sausages.

The meat, which came from various sources, was hung up in the meat room of the inn, and the day before the festival was partly roasted, partly minced up for sausages, and the fragments used for the ragout were cooked on the following day.

Nothing amiss was observed with the ragout, but the cold roast veal was in part decomposed, and the sausages were manifestly bad. In consequence of this they were largely distributed among the spectators, the children, and persons who could not afford to pay. Out of 690 persons who sat down to the collation, 290 were attacked. In all, 668 persons were infected who had partaken of the meat provided for the festival, either at the collation or at the inn, or who had been supplied with it at home; besides which 49 secondary cases occurred—*i.e.*, persons who subsequently became affected by contagion without having eaten of the meat. All other sources of infection could be almost certainly excluded, and Kloten was quite free from typhoid at the time. A very large number of the visitors to the festival ate no meat, but only drank wine; none of these were attacked. And it was clearly shown that the water

was not the cause of the outbreak. Many persons who had drunk no water were attacked, and others who had drunk freely escaped. Several persons who drank wine to excess, and consequently vomited in the evening, afterwards escaped.

The incubation period, as in the Andelfingen epidemic, was for the most part very short. Some persons were taken ill on the second day, with loss of appetite, nausea, headache, pain and swelling of the belly, and slight fever. These early cases were the mildest, and many patients recovered in a few days. The greater number fell ill between the fifth and the ninth days. The symptoms were chills, fever, diarrhœa, great prostration, in many cases early and violent delirium. Epistaxis frequently occurred, and also profuse intestinal hæmorrhage. The roseolous rash of typhoid was present in almost all the cases, and in many was remarkable for its extensive development, sometimes leading to little infiltrations forming distinct elevations, and leaving behind slight pigmentations.

Post-mortem examination showed the characteristic appearances of typhoid fever, infiltration, and sloughing of Peyer's patches and the solitary glands, with characteristic ulcers where the sloughs were detached; not unfrequently also infiltration and sloughing of the solitary glands of the large intestine, great enlargement of the mesenteric glands and spleen.

With regard to the meat supplied, the following

facts were ascertained:—Forty-two pounds of veal were furnished by a butcher at Seebach, taken from a calf which appears to have been at the point of death from some disease, when it received the *coup-de-grâce* from the hands of the butcher. All the flesh of this animal was sent to supply the festival at Kloten, but the liver was eaten by an inhabitant of Seebach, and he was attacked by typhoid fever; and the brain was sent to the parsonage at Seebach, and all the household became affected by the same disease.

It was also ascertained that another of the calves which supplied the veal was suffering from umbilical phlebitis and peritonitis, and was at the point of death when it was slaughtered. The veal from this calf had been kept fourteen days, and was in a decomposed condition. All the meat was placed together in the meat receptacle of the inn, which was in a horribly filthy state, and no doubt the putrefying flesh of this last calf, together with the state of the receptacle, would rapidly excite decomposition in the whole supply.

According to Professor Huguenin, the meat had two injurious qualities—First, it was putrid; secondly, it was infected with the specific typhoid poison. And the resulting epidemic showed two corresponding groups of symptoms—First, a putrid intoxication, which caused an acute catarrh of the gastro-intestinal mucous membrane, coming on early; and secondly, typhoid fever, which sometimes fol-

lowed on the former, and sometimes occurred independently. The mortality was extremely small, this being probably due to the fact that many of the affected persons only suffered from the former symptoms. The peculiarities of the Andelfingen epidemic are no doubt to be explained in the same manner.

The question now arises, How did the meat acquire the specific typhoid infection? There can be little doubt but that this was derived from the calf supplied by the butcher at Seebach, the liver and brain of which gave typhoid fever to persons residing there who had not visited Kloten. This calf was very ill; and Professor Huguenin thinks there can be no doubt but that the animal was itself suffering from typhoid fever. He has convinced himself that typhoid fever is not uncommon among cattle in Switzerland; and that the flesh of such animals is often eaten without any injurious results, some putrefactive changes being necessary to develop the infectious properties. A remarkable fact bearing on this question was observed in this epidemic. In the house of one of the persons attacked, while he was still laid up, two calves were taken ill and slaughtered, and a post-mortem examination showed the characteristic intestinal lesion of typhoid fever, the presumption being that these amimals became infected by the alvine discharges of their master before he took to his bed.

Shortly after this outbreak another small one took place at Kronau. A butcher there refused to buy a

calf because it was ill; the family therefore to whom it belonged ate it themselves, and six members were attacked by typhoid fever.

A similar outbreak affecting fifteen persons occurred near Winterthur in 1869, the particulars of which I have been unable to obtain; and one has been recorded from Iceland.

I think, therefore, the evidence that typhoid fever may be caused or disseminated by diseased meat is sufficiently conclusive. But whether this is due to the animals being themselves affected with the disease requires further investigation. The experiment of feeding pigs with typhoid stools in this country has always failed to produce the disease: whether the attempt has been made with calves I do not know.

Seeing, then, that the flesh of calves suffering from typhoid fever, when in a state of decomposition, appears to be capable of communicating the disease, it is an interesting inquiry as to whether the milk of cows similarly affected would also possess contagious properties. I believe that in this country, wherever epidemics have been caused by contaminated milk, the poison has been traced to the water which has been mixed with the milk; but, judging by analogy, there is great probability that the milk itself might be capable of communicating the disease. It has been demonstrated by repeated experiments that not only will the flesh of tuberculous animals, when eaten by other animals, cause tubercular ulceration

of the intestines followed by general tuberculosis, but that also the milk of tuberculous cows possesses the same properties; in both cases the infection is prevented by thorough cooking or boiling. It is an uncomfortable reflection that cooking seems not to have destroyed the poison contained in this infected meat.

Doubtless these questions, which are of the utmost importance to the public health, will soon be determined by the researches of foreign experimenters; for in this country, I presume we must, in investigations of this kind, be satisfied now to play the part of hearers rather than of doers.

The usual modes in which typhoid fever becomes disseminated have been so thoroughly investigated, and are so universally recognised, that I do not propose again to repeat them. Widespread epidemics have always been traced to contamination of the water-supply, the water itself being the medium of the infection, or else the milk with which the contaminated water has been mixed. Local outbreaks confined to particular houses are sometimes due to the same cause, sometimes to inhaling emanations from cesspools, drains, and sewers, in consequence of some defective sanitary arrangements; and it must be confessed that great ingenuity has been shown by architects, builders, and plumbers to insure that the air we breathe or the water we drink shall be so contaminated. Sometimes this is due to dishonesty, sometimes to carelessness, more often to ignorance.

Several striking instances of each of these have come under my notice. Thus, in a new country house, where the drains had been properly planned and arranged, and it was supposed every precaution had been taken, the basement nevertheless became sodden with sewage. On taking up the drains it was found that the pipes had merely been laid end-to-end, and not cemented together. This was an example of dishonesty. In another case the drain-pipes had been made to slope the wrong way, insuring the perpetual accumulation of stagnating sewage. This was carelessness. A third case was that of large public institution in which several cases of typhoid had originated, and I was asked to examine the water-supply and closets. The main cistern supplying the building was placed on the roof, and was of course covered by a lid. On lifting this I found a distinct smell of sewer-gas, and I noticed a large pipe which opened a few inches above the surface of the water. On investigation this was found to be a ventilating shaft constructed for the purpose of ventilating the main sewer of the building—no doubt put up with the best intentions many years ago, when the necessity of ventilating drains first began to be insisted upon.

We may hope that such glaring examples are fast becoming things of the past, though I fear many of our architects are far behind the standard laid down 1,800 years ago by Vitruvius, who demands of an architect not only that he shall be acquainted with the technicalities of his art, but shall have studied

philosophy and acquired a knowledge of medicine,—"Philosophos diligenter audiverit, ... medicinæ non sit ignarus."

Probably one of the commonest causes of sporadic outbreaks of typhoid fever in large towns are water-closets, and the introduction into houses of this most necessary adjunct of civilisation has always been followed by an increase in its development. This has been notoriously the case in Edinburgh, where water-closets are of comparatively recent introduction; and I think we are justified in regarding Sir John Harrington, who, in the reign of Queen Elizabeth, invented water-closets, as being largely responsible for the spread of typhoid fever. The dangers of water-closets are twofold:—1. When the handle is pulled up the contents of the pan necessarily displace the air in the soil-pipe and drain, and unless (which is the exception) a proper ventilating apparatus has been adapted, some of this rushes up into the closet, carrying of course with it the typhoid poison, if this should happen to be present, and so very likely infecting the occupant. 2. The risk of contaminating the drinking-water. Even at the present time the great majority of water-closets are so constructed that the waste-pipe of the supplying cistern, from which the drinking-water is often taken, goes down directly into the soil-pipe, and so—as in the institution I have just described—though not intentionally, forms a ventilating shaft to the drain. I have no doubt but that this arrangement holds in two-thirds

of the London houses, and consequently the drinking-water is habitually contaminated with sewer-gases. Fortunately the system of drainage is now so admirably constructed that stagnation of sewage, which seems to be the most important factor in the development of the typhoid poison, does not often occur.

It is manifest that a system of periodical inspection of the water-cisterns and water-closets is urgently required. The present water companies have the right, which they occasionally exercise, of inspecting the cisterns, but only for the purpose of seeing that the water is not allowed to run to waste. But if the water-supply were in the hands of a public body nothing would be easier than to enlarge a little the scope of this inspection and to make it more regular; and I believe if this were done it would almost put a stop to the local and sporadic cases which occur in London,—I except, of course, the annual outbreak among the upper and middle classes, who bring the disease back with them from the various health-resorts in this country and abroad, where they have spent their autumn holidays.

Another point of great practical importance in the etiology of the disease is the extraordinary dilution under which the poison may retain its potency. One remarkable instance of this I have described in the epidemic at Lausen. A still more remarkable example is furnished by the recent epidemic at Caterham and Redhill. This outbreak was clearly shown by Dr. Thorne to have been caused by the

contamination of the water of the Caterham Water Company by the alvine discharges of a single workman suffering from ambulant typhoid, who was engaged in the construction of a new adit. If any necessity arose for the workmen to relieve themselves during their spell of work below, which lasted from eight to twelve hours, and there should be any difficulty or delay in their being drawn up to the surface, it was arranged that they should use the buckets which were employed in raising the chalk.

This man, it appears, had very copious diarrhœa, and had to relieve his bowels two or three times during each spell, but he positively denied ever having passed his motions in the adit without waiting for a bucket. But, nevertheless, there were undoubted means by which his evacuations could have found their way into the water; for, as the buckets were drawn up, their oscillations caused them to strike against the sides of the shaft, and some of the contents would so be shaken out and fall over a stage in the water below. And he also stated that his motions were so liquid that the buckets, which were also used to lower materials used in the construction of the adit, must have been stained with them. Here, then, we have in all probability only some splashings of typhoid stools mixed with a very large body of water—a proportion of the most extreme minuteness—and yet the water so contaminated gave typhoid fever to 305 persons.

I think these two instances are sufficient of them-

selves to serve as a warning against trusting to irrigation and downward filtration as a means of purifying water, and also against the dictum that water containing less than a certain proportion of organic impurity is practically wholesome and fit for drinking, irrespective of its original source. It ought, I think, to be laid down as a rule of hygiene, that human excrements should under no circumstances be mixed with drinking-water, however completely they may be subsequently removed by filtration or rendered innocuous by oxidation. Of course, in the case of a city like London, this can only be looked upon as an ideal to be realised in some distant future; but with less than this we ought not to rest satisfied.

It is now an interesting question, What are the causes to which our immunity in London from the spread of typhoid fever by water is due? The Thames, from which we derive our drinking-water, is unquestionably contaminated not only with excrement, but also with the specific poison of typhoid fever. Until quite recently many large towns, where typhoid fever is more or less endemic, discharged their sewage into it, and many places do so to this day, and yet I believe that since the intake of the water companies has been removed above Teddington Lock, the water, as delivered from their mains, has never caused an outbreak. Where in London typhoid fever has been traced to the water, it has been due to its subsequent contamination in the cisterns of the consumers.

With the evidence afforded by Lausen and Caterham before us, we can hardly ascribe this immunity to the mere dilution of the poison; neither can it be attributed to filtration, for we have seen that the very complete natural filtration of the Lausen water was inoperative, and it is notorious that the filtration of the London water companies is very defective. It is not sufficient even to remove the coarser particles in suspension; thus in times of flood the water is often more or less turbid, and moreover frequently contains living organisms. It is therefore quite impossible that the extremely subtle poison of typhoid would be separated. What degree of natural or artificial filtration is required to effect this is not known. It is probable that filters which act chemically on the water may do so. Dr. Gustav Bischoff, in a paper communicated last year to the Royal Society, showed that the germs of putrefaction were completely separated or rendered inert by filtration through spongy iron; and the same thing has been claimed for charcoal; but hitherto these modes of filtration have only been applied on a small scale, and it must be remembered that unless a filter is kept clean and in good order, it only increases the risk of contamination.

I think, then, that we must assume that when the typhoid poison enters a large running body of water, where it is freely exposed to the air and various oxydizing agencies, it is soon rendered inert; while in close, confined situations it retains its activity for an

indefinite time. This is, perhaps, a reason for regarding a large open body of water as a safer source of supply in a thickly populated country than wells and springs, in consequence of the ever increasing difficulty of preventing their accidental contamination.

A point of great interest in the pathology of typhoid fever, and one which may perhaps be most conveniently considered in connexion with the etiology, is the period of latency or incubation, the determination of which presents great difficulties.

If we are to accept as trustworthy the statements which have been made by the authorities who have written on this subject, we should have to confess that typhoid fever may have an incubation varying from a few hours to six weeks ; and though the evidence adduced in favour of these extremes is practically valueless, nevertheless it can hardly be doubted but that great variations do occur.

We may, I think, accept as well established that the usual period, especially when the disease is communicated by drinking-water, is about fourteen days. But considerable deviations, both real and apparent, are often met with. Often much difficulty is experienced in fixing the exact date of onset of the disease, and to this many of the apparent deviations from the usual period are due. Not only in many cases is the onset gradual, but frequently there are distinct premonitory symptoms, a *stadium prodromorum,* during which the patients complain of weariness, some pain and weight in the limbs, impairment

of appetite, often diarrhœa. These symptoms may pass gradually into those of the developed disease, and unless thermometrical observations have been made—which is rarely the case at this stage—it becomes impossible to fix the date of the true commencement of the attack, which should be reckoned from the time at which the temperature rises. In other cases the period of invasion is distinctly indicated by the occurrence of rigors, which may or may not have been preceded by these premonitory symptoms. I have recently seen a very severe case of typhoid, in which the premonitory symptoms lasted for a fortnight before the occurrence of rigors and the definite onset. In this case a very slight increase in the severity of these premonitory symptoms might easily have caused the incubation period to have been reckoned as one day instead of fourteen. In children, in whom the nervous system reacts strongly to slight causes of disturbance, I have no doubt but that the premonitory stage is often included in the fever; and many of the best authenticated cases of unusually short incubation periods have occurred among children.

Another difficulty is that of excluding in a disease like typhoid, of which the contagion is indirect, other sources of infection than that which is supposed to have caused the attack.

The following may be cited as instances of unusually short incubation:—

In the milk epidemic in Marylebone a child was

taken ill five days after drinking the contaminated milk, but it is possible that here the premonitory stage was included in the fever.

Griesinger relates three cases in which the period was less than twenty-four hours. He states that one day, while sitting by the bedside of a case of typhoid, he suddenly felt unwell, and thought he had caught the fever, and the next day he was taken ill.

I only mention this case, as it is not unfrequently quoted as an evidence of the occasional occurrence of very short periods. His other two cases are almost equally valueless.

Dr. Clifford Allbutt published a case in which the period of incubation was exactly four days, but further investigation showed that the disease was acquired, in all probability, in quite a different manner from that at first supposed, and that consequently no conclusion could be drawn from it as to the incubation period.

One of the most striking instances of a supposed very short incubation period is the well-known outbreak at a school at Clapham, in 1829. Here, within four days of exposure to the emanations from the contents of a choked drain, twenty boys out of twenty-two were attacked by vomiting, purging, fever, and extreme prostration, and two died comatose within twenty-five hours. In these two cases the solitary glands and Peyer's patches were found swollen and infiltrated. Here, however, the nature of the disease is open to doubt. Some very high authorities, as Sir

Thomas Watson, do not regard it as having been typhoid; and certainly the phenomena were very unlike those of ordinary typhoid. The contagion must have been of extreme virulence, as two cases died comatose in twenty-four hours, and yet the other twenty recovered, giving a general mortality below the average. There can be no doubt but that decomposing sewage may contain other poisons besides that of typhoid fever, and considerable swelling of the glandular apparatus of the intestine may be caused by many kinds of irritation.

Professor Quincke, of Bern, cites the following example of unusually short periods. Three boys played on successive days, from March 13 to 16, with some straw from a mattress soiled with the discharge of a fatal case of typhoid. All three were infected; the first was taken ill on March 22. Here the maximum limit was nine, the minimum three days. Another case was that of a woman who came from a distance to an infected house, where she stayed two or three days. She felt ill on her way home, and after a few days took to her bed, and died on the fourteenth day. Here the maximum limit was six days.

More common than very short are protracted periods, and an incubation of twenty-one days is by no means infrequent, especially when the cause has been contaminated water or milk. But periods of five or six weeks may, I think, in all cases be ascribed to errors of observation.

The causes of this variability of the incubation period of the contagious fevers are very obscure, and cannot be satisfactorily accounted for on any of the theories which have been put forward as to the nature of these diseases. In some the period is much more variable than in others. Thus in morbilli, rubeola, variola, varicella, and for the most part typhus, it is pretty constant, and is much the same in all; scarlatina with a short, and typhoid with a long period, are much more variable, and in some fevers in which poison is fixed, and can only be communicated by inoculation, as syphilis and hydrophobia, the period may be protracted to an extraordinary degree.

Among the causes which may influence the incubation period is, first, the mode in which infection takes place. It seems probable that when the poison directly enters blood, as by inoculation, it is likely to take effect more rapidly than when it is absorbed through the mucous surfaces. Thus there is reason to believe that when the poison of scarlatina is introduced through a wound or raw surface, as after a surgical operation or delivery, the incubation may be very much curtailed.

So too we might expect that when the lymphatics are the medium of absorption, and the poison is first conveyed to the glands, the incubation period would be longer than when it is absorbed by the blood-vessels. Thus in syphilis, which has a very long incubation, the poison is absorbed by the lymphatics.

According to the views of Liebermeister, the poison of typhoid is always swallowed and absorbed by the intestinal glands, where it exercises its primary influence. There can, however, I think, be little doubt but that it may also be absorbed by the respiratory and other mucous membranes, and so obtain a more direct entrance into the blood. Indeed, if, as is maintained by Friedreich, the spleen begins to enlarge during the incubation period, it is manifest that the poison does not lie dormant during this period in the intestinal and mesenteric glands, but has already passed into the blood. Now, it appears, on the whole, that the incubation period is more frequently unusually short where the disease has been acquired by exposure to emanations from decomposing sewage than when drinking-water has been the medium of contagion, whereas the unusually long periods have occurred in the latter condition. But to this there are exceptions, as in the milk case I quoted above; and further evidence is required before any positive conclusions can be drawn.

Another cause which may influence the incubation period is temperature. It is well known that vaccinia will often run a somewhat shorter course in hot weather; and by increasing the temperature of the skin, as by a blister, the eruption of small-pox may be hastened: it is therefore probable, if anything occur to cause febrile disturbance, and so raise the temperature during the period of incubation, that this will be shortened.

Lastly, certain constitutional conditions will influence the incubation period. Thus Obermeier has shown that the occurrence of menstruation shortens the latent period of small-pox.

That the variability of the incubation period is due in many cases to some peculiarity on the part of the patient, and not to any difference in the state of the poison or in the manner in which it is introduced, is manifest from the fact that it usually happens, when several persons are exposed at the same time and in the same manner, that they fall ill at very variable intervals. A very remarkable instance of this kind is narrated by Professor Quincke.

On June 22, 1873, the Federal Gymnastic Festival was held at Münsingen, a village situated about seven miles from Bern, and there was a large gathering of visitors from all parts of Switzerland, most of whom left Münsingen the same day. It so happened that the wife and son of the landlord of one of the inns, which was close to the ground where the gymnastic meeting was held, were ill with typhoid fever, and only ten feet from the leaky soil-pit of the privy of the inn was a well from which water was supplied to the persons taking part in the festival, as well as to those who took refreshments at the inn itself. An epidemic of typhoid fever broke out among persons who had attended this festival, and the particulars of fourteen cases occurring among visitors who came from places free from typhoid have been ascertained.

In one case the attack commenced eight days after the festival, in three cases on the twelfth day, in one case on the thirteenth day, in two cases on the fourteenth day, in two cases on the fifteenth day, in two cases on the sixteenth day, in two cases between the sixteenth and eighteenth days, and in one case some time between the fourteenth and twenty-second days, when he first came under medical observation. Here, then, we see incubation periods varying from eight to eighteen days where there is strong reason for believing that the conditions of infection were the same.

These cases are also interesting as many of them were attended by well-marked premonitory symptoms, the causes of which in all the contagious fevers are very obscure. One of these patients was attacked two days afterwards by headache, vomiting, and diarrhœa, from which he recovered, though he did not again feel well, and then on the sixteenth day he was again attacked by rigors and headache, and the disease now became developed. In a second case violent diarrhœa and vomiting came on a few days after, and again quite subsided before the commencement of the fever. Similar cases are not uncommon. I have recently had under my care a case in which the patient was attacked by diarrhœa, which only lasted a few days, though he continued to feel somewhat unwell; but it was not till after the lapse of a fortnight that he shivered and was definitely taken ill with typhoid. These cases would seem to

indicate that the poison exercises a primary irritating action on the intestinal mucous membrane before its absorption; or possibly, where contaminated water is the medium of infection, other irratating substances may be present which cause the primary diarrhœa. Most probably, however, the primary diarrhœa is due to the typhoid poison.

On the whole, we must, I think, come to the conclusion that the variability of the latent period of typhoid, though it may be influenced by the manner in which the infection takes place, depends in most cases upon some unknown peculiarities of the patient himself. It is not probable that the amount or intensity of the poison can have any influence, as we see both in this and in other fevers that the period of incubation of the mild and severe cases is the same.

LECTURE II.

Pathology.

WHEN we proceed to consider the clinical and pathological phenomena of typhoid fever, and to compare them with those of allied diseases, it is evident that we may divide them into two classes:—Firstly. Those which are peculiar to this form of fever; Secondly. Those which are common to it with many other febrile diseases;—the former being the characteristics of the species, the latter those of the genus.

To the former class belong the etiology and mode of dissemination, the period of incubation, the intestinal lesions, the roseola, and the type and duration of the fever; to the latter, the high temperature, the febrile consumption and wasting, the albuminous infiltration and softening of many organs and tissues, and various disturbances of the principal functions of the body.

I propose first of all to consider the pathology of some of these secondary phenomena, which are indeed of primary importance in the treatment, for our

control over the former is so restricted that when once the system has become infected with the typhoid poison it is out of our power to prevent it from producing the typical lesions.

If we examine carefully the organs of a fatal case of typhoid fever, or any of the infective fevers, or even of other febrile diseases which are not due to an infective process—though here the changes are less constant and less marked—we shall find some or all of the following morbid conditions :—

Firstly. A granular or albuminous infiltration of the cells of most of the glandular organs, especially the liver, the kidneys, the salivary glands, the pancreas, the gastric glands; and also a similar change in the striped muscular fibres, both of the heart and voluntary muscles.

The change consists in the appearance in the protoplasm of the cells and fibres of extremely minute granules, soluble in acetic acid, giving a clouded appearance which has been compared to that of ground glass; this obscures the nuclei, and causes, when advanced, some enlargement and irregularity of form. In extreme cases the cells thus affected disintegrate into a detritus, this being usually, though perhaps not necessarily, associated with fatty degeneration. Unless the change is very extensive the affected organs do not show any marked alteration to the naked eye; they may be somewhat enlarged, of diminished consistency, and, unless congested, paler than usual, with their normal shiny appear-

ance somewhat dulled. Virchow, as is well known, regarded this granular infiltration as an inflammatory parenchymatous exudation, and in many cases these changes are associated with processes which are unquestionably inflammatory, but they may occur independently, and must probably be looked upon as essentially of a degenerative nature; often, indeed, they are but the first stage towards fatty degeneration.

A change, which is probably of the same nature, occurs in the nerve cells of the central nervous system; they become granular, with an increased deposit of granular pigment.

It can hardly be doubted but that the result of a large number, if not of the majority, of fatal cases of typhoid, is due to these changes. If we exclude those deaths which are directly caused by the intestinal lesions, as perforation and hæmorrhage, which after all are the minority, the remainder may for the most part be ascribed to the failure of the heart and central nervous system. And of these, failure of the heart with its concomitant congestion of the lungs plays the most important part.

The affection of the heart, then, is of primary importance, and its pathology and symptoms have been most carefully investigated by many observers. Very often the heart presents no obvious changes to the naked eye; in other cases it is more or less softened and altered in colour; but without a microscopical examination it is impossible to pronounce with any

certainty on its condition. The changes may affect both the muscular fibres, the inter-fibrillar connective tissue, and the vessels. The fibres present to a greater or less degree the granular changes which I have described; the striæ are more or less obscured, and frequently with the albuminous granules oil globules are present. The muscle nuclei often show indications of proliferation, and a small-celled infiltration is frequently visible between the fibres, due either to an exudation or to proliferation of the connective tissue. M. Hayem also describes a proliferation of the cells of the tunica intima of the small arteries, which he ascribes to an endoarteritis. Not unfrequently there is a diffused sanguineous infiltration of the inter-muscular connective tissue. Side by side with the altered fibres we commonly find others which are quite normal. These changes are frequently scattered in patches through the walls of the heart, and are often well marked in the musculi papillares of the mitral valve. We have, then, here evident signs of an irritative lesion as well as of degeneration, and M. Hayem regards the process as essentially a myocarditis.

The symptoms of this degeneration of the heart are well known and had attracted the attention of all the great writers on fever before the intimate pathology of the affection was studied. They consist in feebleness of the cardiac impulse, which sometimes becomes almost imperceptible; an alteration in the character of the first sound, which be-

comes short and faint; and to this must be added the soft systolic apex-murmur which is so frequently present in severe cases of typhoid, and which disappears again during convalescence, and is probably due to want of tone in the musculi papillares of the mitral valve allowing some regurgitation. This systolic murmur is often present before the other signs are very pronounced.

Corresponding changes occur in the pulse; the usual febrile dicrotism becomes exaggerated, and although the pulse may remain for a time tolerably full, it becomes extremely compressible, undulating, and shows more or less irregularity, not so much of rhythm as of force.

When death is caused by this condition of the heart, it is usually preceded by a gradually increasing hypostatic congestion of the lungs, and takes place slowly; but occasionally it is quite sudden from syncope. Usually the symptoms of this degeneration of the heart in typhoid do not show themselves till the later stages of the disease. But there are reasons for believing that the affection may begin quite early, and run a very rapid course. Last year a woman in the London Fever Hospital died quite suddenly from syncope about the twelfth or fourteenth day of the disease, before any intestinal sloughing or ulceration had taken place; and the heart was found in an advanced stage of granular infiltration.

These cardiac changes are by no means peculiar to typhoid fever; they are met with in typhus, small-

pox, erysipelas, scarlatina, diphtheria, where they are usually extremely well-marked; and in this disease, as is well known, sudden death by syncope is by no means uncommon.

The next change which is common to typhoid and many other fevers is the so-called vitreous or waxy degeneration of the striped muscles, described by Zenker. The fibres swell, lose their striation, and the tube of sarcolemma becomes filled with a semi-transparent, colourless, homogeneous material, which has been compared to wax in appearance. These altered fibres present transverse cracks and fissures, and ultimately break up into irregular fragments, which become smaller and smaller, and at last disappear, leaving the sarcolemma empty. Then, if the patient recovers, the fibre is regenerated by a cell-growth within the sarcolemma. Most probably the process consists primarily in a coagulation of the contractile muscle-substance analogous to that which takes place in the rigor mortis. This change is common to many fevers, as typhus, small-pox, scarlatina, tuberculosis. In typhoid it is often very extensive, and may cause considerable temporary destruction of muscle. It may affect all the striped muscles, but is most frequently met with in the muscles of the abdominal wall, the adductors of the thigh, the diaphragm, and the tongue. Though not very often present in the heart, it does occasionally occur here also.

Another change which is common to all febrile diseases is the diminution of the red blood-cells, pro-

bably due to increased disintegration on the one hand, and to diminished formation on the other.

A fourth change is the wasting of the body. This is usually proportionate to the intensity and duration of the fever; and in typhoid the serious lesions of the digestive canal tend still further to increase it.

Leyden reckons the average daily loss of weight in high continued fever at about 7 per 1,000 of the total weight of the body, which is about half that which takes place in total deprivation of food; and Chossat has shown that death takes place from inanition when the body has lost 40 per cent. According to this calculation, it would take eight weeks of high continued fever to reduce the weight 40 per cent. and cause death from inanition; but in chronic fever a much greater loss can be supported, in consequence of the febrile process maintaining the temperature, and so preventing death from cold, inability to maintain the temperature being one of the causes of death in inanition.

It rarely happens that death takes place in typhoid merely from the febrile wasting, though this is often very great, and exceeds the daily average given by Leyden. I have recently had under my care a case where the weight was reduced in about four weeks from eleven stone to eight stone—a loss of nearly 30 per cent. Supposing the fever to have continued for eight weeks, this would have produced a loss of nearly 60 per cent. It would, however, seem that the amount of wasting is greater in persons who are

muscular and corpulent than in those who are spare and lean; the latter class, it is well known, bear an attack of fever better than the former. The causes of this wasting of the body in fever are twofold. First, simple inanition due to the diminished ingestion and assimilation of food, the result partly of failure in the appetite, partly of impaired digestive and assimilating power, and partly from restriction of the diet in consequence of certain theories as to the injurious effects of food in fever. The influence of this last cause varies, of course, with the mode of treatment which happens to be in vogue. In this country, in consequence of the teachings of Graves, Stokes, Todd, and other authorities, fever patients are for the most part given as much food as they can assimilate; but there have been times, especially on the Continent, when the routine fever-diet would barely suffice to ward off death from inanition. Under any circumstances, it is probable that the amount of food assimilated in fever does not amount to half that taken in health.

This wasting from inanition takes place chiefly at the expense of the adipose tissue and of the store albumen, *i.e.*, the albumen of the blood, lymph, and interstitial fluids; to a less degree, and chiefly in the later stages, at the expense of the tissue albumen.

The second cause of the wasting in fever is the increased consumption due to the febrile process. This in man affects both the fat and the albumen, though in the artificial septic fever of dogs it appears

that the consumption of the hydrocarbons is not increased.

The consumption of albumen is measured by the amount of the urea discharged, which in fever very much exceeds that which could be produced by the metabolism of the nitrogenous food, and, notwithstanding the restricted diet, often exceeds the amount passed in health on a full diet. This great increase in the discharge of urea is always most marked in the earlier stages of the fever, and in protracted fevers, as typhoid, becomes much diminished in the later stages.

There are strong reasons for believing that this increased formation of urea takes place at the expense of the tissue albumen, and not of the store albumen; for it is found that it is accompanied by an increase of the salts of potash in the urine, while the salts of soda are commonly diminished; hence it is probable that the urea is derived from albumen, which is associated with potash, and not with soda. Now, this is the case with most of the tissues, especially the muscles and red blood-cells; while in the liquor sanguinis and lymph the salts of soda predominate: hence in all probability the former, and not the latter, are the sources of the urea produced by the febrile consumption. The colouring matter of the urine appears also to be increased in fever, and this must necessarily be derived from the coloured tissues, especially the red blood-cells, which we have seen to undergo great diminution; and as extensive dege-

nerative changes are commonly found in the muscles, it is probable that these tissues especially suffer in the febrile process.

The consumption of hydrocarbons, as shown by the increase of carbonic acid in the expired air, does not admit of so exact a determination; the proportion in each expiration is always diminished, but owing to the increased frequency of the respirations, the total amount expired in twenty-four hours is always increased. Senator reckons this increase at 37 per cent., a far larger quantity than could be afforded by the carbon set free by the increased disintegration of the albumen.

We have now to consider what is perhaps the most important phenomenon of fever, the temperature.

The maintenance of the temperature of the body depends, of course, on a balance being kept up between the production and dispersion of heat, this balance being maintained by a regulating apparatus, in consequence of which, in a state of health, the production of heat on the one hand, or its dispersion on the other, may be very greatly modified without producing more than slight and temporary alterations of temperature. There can be no doubt but that this regulation is effected through the nervous system, though the exact mode of operation is still somewhat obscure.

When the influence of the intracranial part of the nervous system is removed, as by the crushing or division of the spinal cord below the origin of the

phrenics, the temperature of the body, as is well known, cannot be maintained; it has become *labile* instead of *constant*. Sometimes a great rise takes place, sometimes a fall. In man the former is the more common effect, though several cases have been recorded where the temperature has fallen to extremely low degrees. In small animals the effect seems to depend very greatly on external conditions. Under the influence of external warmth, the temperature will rise to extreme heights; if the external conditions favour cooling, the temperature will sink to such a degree that the animal dies of collapse. These alterations of temperature, due to injury of some part of the nervous system, are probably produced by alterations in the dispersion of heat, which is effected chiefly by the lungs and skin, and consequently regulated by the respiratory and vasomotor centres, together with the special nerves, if such there be, which control the sweat glands.

What part the nerve centres play in the regulation of the production of heat is a question of great difficulty, and one about which physiologists are by no means agreed. There can be no doubt but that the chemical processes by which heat is produced in the animal body are independent of the nerve centres. This is shown by the fact that heat continues to be produced after the complete destruction of the spinal cord; nay, the production goes on for some time after death has not only destroyed all nervous influence, but has brought the circulation itself to a standstill.

It has been proved by Valentin that the post-mortem production of heat is a constant phenomenon; and in consequence of the diminished dispersion from arrest of the cutaneous circulation and respiration, we often see an actual rise of temperature. Generally, however, this post-mortem formation of heat is only indicated by a retardation of the cooling of the body.

Nevertheless, it can hardly be doubted but that the production as well as the dispersion of heat can be greatly modified by nervous influence, though we are not, perhaps, justified in assuming the existence of a special heat-exciting and heat-depressing centre. The phenomena are most probably vaso-motor. It is evident that if more or less blood flow through the more active heat-producing organs more or less heat will be produced.

When we proceed to consider the cause of the high temperature in typhoid fever, where it often remains above normal for several weeks, we can hardly doubt but that there is some disturbance of the regulating function, for no mere alteration of the conditions either of the production or dispersion will cause more than slight and temporary changes in the temperature; but the nature of this disturbance is still very obscure. That the vaso-motor system is rendered more unstable in typhoid is shown by the phenomena of the so-called *tache cérébrale*, which are almost always well marked.

Now, in a disease like typhoid, in which the tem-

perature usually runs such a typical course, with regular diurnal variations, corresponding, though in an exaggerated manner, to the diurnal fluctuations in health,—with so steady a rise to the fastigium, and decline to the defervescence, so that from a mere inspection of the chart we can generally diagnose the disease,—it is quite certain that the regulating apparatus has not ceased to act. The view has been put forward, and widely accepted, that in typhoid fever the regulating point has been simply raised and adapted, as it were, to a temperature, say five degrees above the normal. If, however, we compare the behaviour of the temperature in this disease and in health, this view becomes untenable. In the first place, the temperature has become more labile; it can easily be raised and depressed by causes which would have no effect in health. Thus, changes in diet, slight exertion, mental emotion, will often cause great temporary alterations. The same point is shown by the effect of remedies. A dose of thirty or forty grains of quinine will hardly affect the temperature in health, but in typhoid fever will generally reduce it three or four degrees, and keep it down for several hours.

Secondly, we miss in fever that compensatory reaction which Jürgensen has shown to occur in health when the temperature is artificially reduced. According to his observations, if a healthy man be placed in a cold bath, the temperature may be reduced many degrees, in proportion to the coldness

and duration of the bath; and it continues to sink for some time after removal. Soon, however, it again rises, and continues to do so till it has gone above the normal point. If the bath be repeated, the same phenomena show themselves; but after a certain number of repetitions the reactive rise counteracts the chilling effect of the bath, so that further immersion no longer reduces the temperature of the rectum. In typhoid this compensatory rise does not take place, or at any rate to a much less degree, and after fifty or even a hundred and fifty repetitions the last bath reduces the temperature as effectually as the first. Hence in typhoid fever we regard the regulating influence as weakened, or at any rate rendered insufficient for the new conditions caused by the fever, but neither abolished nor set at a higher point.

We, however, not unfrequently meet with cases where the temperature appears to follow no law, but where the regulating function seems to be entirely suspended: where sometimes the temperature will rise to hyperpyretic heights, far exceeding those that are ordinarily met with in fever, and incompatible with the continuance of life; at other times it will fall far below the normal, and the patient sink into a state of collapse.

There can, I think, be little doubt but that these hyperpyretic temperatures are due to a paralysis of the heat-regulating functions of the nervous system, and are mostly an indication of a general failure or

paralysis of the nerve centres. Usually, therefore, their occurrence is a certain sign of approaching death; and in this form hyperpyrexia is very often seen at the termination of all febrile diseases, including typhoid. In these cases the patient does not die from hyperpyrexia, but he has hyperpyrexia because he is dying; and no lowering of the temperature by the abstraction of heat, as by a cold bath, has any effect in retarding the fatal event. Indeed, where there is complete paralysis of the regulating centres, it is not possible to lower the temperature except quite temporarily: it rises again immediately the abstraction of heat is desisted from. In other cases, however, hyperpyrexia may occur without any general or irremediable failure of the functions of the nervous system, though the high temperature itself will, if not reduced, soon lead to fatal coma. In these latter cases, if we can succeed not only in lowering the temperature, but also in stimulating the nerve centres to a resumption of their functions, the patient will recover. Such cases are most frequently seen in acute rheumatism. In the zymotic fevers, as typhoid, extreme hyperpyretic temperatures, as 108° to 110° Fahr., commonly indicate a general paralysis of the nervous centres and impending dissolution.

The regulating function being thus weakened relatively or absolutely, and the temperature rendered more labile, we have now to consider what is the factor which causes it to maintain itself above the normal point, mere lability being as likely to cause

depression as exaltation. The lability, indeed, often continues longer than the fever; and very commonly, for some time after complete defervescence has become established, extremely slight causes, which would have no effect whatever in health, will send up the temperature many degrees.

The opinion generally entertained is, that the cause of the fever heat is increased combustion of the tissues, which we have seen to be the most characteristic phenomenon of fever. But, although in fever the combustion is excessive in relation to the amount of fuel supplied by the food, it is not necessarily in excess of that which takes place in health, where the supply of food is abundant and the muscles are well exercised; and it has not been found possible to establish any definite relation between the discharge of urea and carbonic acid on the one hand, and the temperature on the other. Attempts have been made to clear up this point by estimating the amount of the dispersion of heat in fever: if this be lessened, the temperature may of course rise, though the production of heat be not increased, or even be diminished; if, on the other hand, the dispersion of heat be increased, and at the same time the temperature rise, it is evident that the production must be increased in a still greater degree. The results obtained by different experimenters are conflicting, but on the whole there can be little doubt but that for the most part the dissipation of heat is increased in fever. During the initial rigors, indeed, it is most probable that the dis-

sipation of heat is much diminished from contraction of the cutaneous capillaries; and in all cases there is reason for believing that the dissipation as well as the production of heat is subject to very great fluctuations. Senator considers that periodic contractions and dilatations of the cutaneous capillaries take place, favouring the retention or dissipation. It is, however, quite conceivable that the balance may be disturbed in the other direction; the combustion, though increased relatively to the amount of food taken, may be actually below that in health, the dissipation may not be diminished in proportion, the labile temperature may yield readily to external conditions which favour cooling, and so we may have the paradox of *febris sine febri.*

In the artificial septic fever produced by the injection of pyrogenetic substances, if small animals, as rabbits and guinea pigs, be experimented on, and if the temperature of the air be below 75°, and means be not taken to prevent the dissipation of heat, the temperature will fall below normal; if, on the other hand, the air be above 75°, or the animal be wrapped up in wadding, the temperature will rise to febrile heights. In larger animals, as dogs, the influence of the surrounding conditions is less marked, and under ordinary circumstances their temperature always rises; but if they be shaved and exposed to a cold atmosphere, then here also a fall takes place. Similar phenomena occur in the human subject, and many cases and even epidemics of typhoid have been met

with in which the temperature has been subnormal throughout the whole course of the disease.

One such epidemic has been reported by Dr. Strube. During the siege of Paris by the Germans in 1870 an epidemic of typhoid fever broke out among the troops, beginning to show itself during the march to Paris, and attaining its greatest height in October. In November a decline took place, which was followed by a fresh outbreak in December. These two outbreaks differed greatly in their characters: the later one resembled in all respects the ordinary form of typhoid; the earlier one presented very different features. In many of the cases the temperature throughout was subnormal, and in others never exceeded the normal point. The roseola was unusually profuse; the nerve symptoms were of marked severity, and were in inverse ratio to the temperature, consisting of violent delirium alternating with stupor; the duration of the fever was very short, defervescence usually taking place at the end of a fortnight. Of the twenty-three fatal cases, in twenty death took place during the first fourteen days. The abdominal symptoms were slight, but the characteristic lesions were found on post-mortem examination. All the cases were characterised by great prostration. These cases presented some features which were probably due to this peculiarity of the temperature: thus, the pulse was but little accelerated, seldom exceeding a hundred; the tongue did not become dry and brown, and the enlargement of the spleen was either absent or

much less marked than usual. Dr. Strube attributed the peculiar features of this epidemic to the depressed condition of the troops; they had been exposed to great hardships on the way to Paris, over-fatigued by forced marches, and very insufficiently supplied with food, and the supply continued deficient for some time after their arrival, owing to difficulties of transport. In the later outbreak these conditions were no longer present.

It now becomes an interesting subject of inquiry, whether the mode of production of heat is the same in fever as in health, *i.e.*, whether it is produced in the same organs and by the same kind of combustion.

The source of the animal heat is of course the combustion of the food and the tissues; wherever metabolism of food or protoplasm is going on, *i.e.*, wherever there is living tissue, heat is being set free. Hence every living cell of the body contributes to the production of heat; but heat is set free more abundantly in some organs than in others, and, taking the body as a whole, the principal sources of heat are the muscles and abdominal viscera, especially the liver, the temperature of the blood in the hepatic vein being the highest in the body. The heat set free by the muscles depends largely on their functional activity: when in exercise it is very great; when they are at rest it is small. The production of heat by the abdominal viscera is probably much more constant, but is necessarily dependent on the assimilation and metabolism of the food.

Now, in fever the production of heat by the abdominal viscera, so far as it is due to the metabolism of the food, must necessarily be diminished in consequence of the restricted diet, and the production by muscular contraction is also much less than in health, as the muscles are for the most part kept at rest. As to how far the heat-producing functions of the liver and other abdominal viscera are modified in fever, little is known. It is possible that an increased production takes place in the spleen, which is so constantly found enlarged and softened, and where it is probable great disintegration of red blood-cells takes place. It is remarkable that in the cases recorded of apyretic typhoid this enlargement of the spleen was either absent or less marked than usual.

With regard to the muscles, however, we have evidence that, notwithstanding the diminution of functional activity, the production of heat in them is very active. It has been found by direct measurements that while in health, when the muscles are at rest, their temperature is below that of the blood in the left ventricle of the heart, in fever it is higher, and both clinical and pathological observations show that the metabolism of the muscular tissue is greatly increased. Thus there is the great muscular weakness so characteristic of fever, the increased discharge of the salts of potash and of creatine and creatinine by the urine, and the various forms of degeneration found after death in fatal cases. We have, therefore,

strong reasons for believing that the muscles in fever, though for the most part in a state of rest, are an important source of the febrile heat, and that this production of heat takes place at the expense of the contractile tissue, and not, as in muscular contraction, from oxydation of hydrocarbons. In fever also, though the voluntary muscles are kept at rest, it must be remembered that the heart and respiratory muscles are more actively exercised.

We have next to consider what is the relation which these different processes bear to one another, and to the symptoms of the disease. According to the views of many pathologists the phenomena of typhoid fever are produced in the following manner. The poison is swallowed, and enters the system through the lymph follicles of the ileum, on which it produces its primary effects; it then passes into the mesenteric glands, where it goes through a stage of latency or incubation, and at last enters the circulation and gets deposited in other lymphatic organs and the spleen, and causes a rapid hyperplasia of their cells, and so produces the characteristic local lesions. The poison also acts on the heat-regulating centres, and produces the febrile temperature. The high temperature in its turn causes all the other morbid phenomena—the albuminous infiltration and softening of the heart and other organs, the vitreous degeneration of the muscles, the acceleration of the circulation and respiration, the febrile consumption of the body, and especially the disturbances of the

nervous system, the delirium and stupor of the typhoid condition.

The correctness or not of this view is, I need hardly say, of the utmost importance in the treatment of the disease. It is therefore very necessary that we should examine the arguments by which it is supported.

In the first place, it is urged that these phenomena occur in many febrile diseases which have nothing in common but the high temperature. Secondly, it is possible to produce many of them by artificially raising the temperature. Thus, the experiments of Dr. Wickham Legge show that by raising the temperature of animals to 112° Fahr. albuminous infiltration of the liver and other organs is produced. Max Schultze, by means of his microscopic warming apparatus, directly demonstrated the injurious effects of high temperature on living cells. We have, too, distinct evidence that raising the temperature of the body increases metabolism. By means of a vapour-bath it is quite easy to raise the temperature many degrees, as the evaporation from the skin, and so the compensatory dispersion of heat, is prevented. Bartels found that sufficiently prolonged vapour-baths raised the temperature, accelerated the pulse and respiration, increased the discharge of urea 16 per cent., and diminished the weight of the body. It is interesting to observe that the discharge of urea was greater during the twenty-four hours after the bath, when the temperature was again normal, than during

the time the temperature was raised. This is analogous to the epicritical discharge of urea which takes place in those fevers which terminate by crisis. In both cases it is probably due to its partial retention and accumulation during the febrile period in consequence of the unfavourable conditions for its elimination during this time. Similar results were obtained by Naunyn in experimenting on dogs.

Lastly, in support of this view may be adduced the effects of treatment. If a patient in the typhoid state, *i.e.*, in a condition of delirium or stupor, with a dry, brown tongue, tremulous muscles, greatly accelerated pulse, and other symptoms which are liable to occur in severe fever, be placed in a cold bath and the temperature reduced to below 100°, a remarkable improvement usually takes place in his symptoms: the stupor passes off, the delirium ceases, the tongue becomes moister, the pulse less frequent, and commonly he falls into a quiet sleep; the symptoms recurring in the course of a few hours when the temperature has again risen to its former height. The effects of a cold bath in cases of hyperpyrexia, as in rheumatic fever, are still more striking, and are well known to all who have employed this mode of treatment. Hence there can be little doubt but that the pathological changes and injurious effects of fever are largely due to the high temperature. But, notwithstanding these and other arguments which may be adduced to show that almost all the phenomena of fever are due to the high temperature, other facts I

think prove decisively that this view is only in part correct and that many of them may occur independently.

In the first place, there are strong reasons for believing that the increased metabolism, as shown by increase in the discharge of urea, is in part, at least, due to the direct action of the fever poison, and not solely to the high temperature. Thus, in the artificial septic fever of the lower animals produced by the injection of pyrogenetic substances, it is found that the increased excretion of urea precedes the rise of temperature. Dr. Ringer has shown that this also takes place in intermittent fever; and other observers have proved the same for relapsing fever. In typhoid fever we are unable to adduce similar proofs, as the attack cannot be predicted, but there can be no doubt that the same law holds good in this fever also.

We have next to consider how far the albuminous infiltration and softening of the heart and other organs is solely caused by the temperature. Against this view it may be urged that these degenerations occur in conditions not attended by fever, as after severe burns, before any febrile reaction has had time to take place; the immediate effect of a burn, as of all stimulations of the sensory nerves, being to depress the temperature. A case has been recorded where death took place six hours after a burn affecting nearly the whole surface of the body, and on post-mortem examination the kidneys and heart were

found in an extreme state of granular infiltration. These degenerations have, moreover, been met with in cases of typhoid fever where the temperature has been but little raised; so that on the whole we must conclude that they may be in part produced by the direct action of the poison, though it cannot be doubted but that a prolonged high temperature not only greatly conduces to them, but is capable itself of their production.

The vitreous degeneration of the striped muscles is probably entirely due to the high temperature.

The next question is how far the characteristic symptoms of fever are due to the temperature, especially the nerve symptoms, the restlessness, delirium, and stupor. I think we must answer that these symptoms, especially the delirium and stupor, are not necessarily dependent on the temperature, though they are largely influenced by it. Dr. Murchison regarded them as caused by the accumulation in the blood of the urea and other products of the disintegration of the albuminous tissues. The occurrence of cases of a febrile typhoid shows that they occur independently; and, moreover, we often see very high temperatures, as in relapsing fever, without any marked disturbance of the nervous system.

But although it seems clear from these examples that many of the phenomena of fever which are ascribed by Brand and Liebermeister solely to the temperature may occur independently, yet it is quite certain that the high temperature exercises a most

injurious influence, inasmuch as it increases the frequency of the pulse and respiration, augments the metabolism of the albumen, and consequently the febrile consumption of the body, adds an additional factor to the albuminous infiltration and softening of the heart, muscles, and glandular organs, and injuriously affects the nervous system, both indirectly by favouring the accumulation in the blood of the products of the disintegration of the tissues, and directly, when it reaches a certain degree, by causing delirium, stupor, and coma.

Another point of great practical importance in the pathology of fever is the effect produced on the digestion. Usually, though not invariably, when the temperature rises above 102° Fahr., the appetite becomes very much impaired, and when the tongue becomes dry it is entirely lost; and it has generally been assumed, though on imperfect data, that a state of fever more or less completely stops the secretion of the gastric juice, and so arrests the digestive function. Modern observations, however, show that this is by no means always the case. Dr. Pavy, in this country, and Hoppe-Seyler, in Germany, have proved that an active digestive fluid can be prepared from the stomachs of animals in a state of fever. And Manassein has shown that though in dogs affected with septicæmic fever the natural digestive fluid, procured by introducing sponges into the stomach, had a much inferior power to that of healthy animals, nevertheless, if hydrochloric acid were added, then

the digestive power was equal to that in health. Hence it is probable that the impaired digestion in fever is rather due to a diminished secretion of acid than of pepsine; and this perhaps explains the beneficial effects often seen to attend the administration of hydrochloric acid. The disinclination, and even complete inability, to take food are doubtless largely due to the partial or complete arrest of the secretion of the saliva and the dryness of the mouth; and these symptoms, perhaps more than any others, are dependent on the temperature. No doubt in fever the stomach frequently rejects food, but the vomited matters are often a decisive proof that the gastric juice is still being secreted. How active the digestive power may be in typhoid is shown by a case where Dr. Heubner fed a patient on beef-steaks through a severe attack, without any injurious consequences. On the whole, I think we need not be deterred from giving fever cases sufficient food, with proper precautions, from fear that it is incapable of being assimilated.

But although in fever the gastric juice may be formed in sufficient quantity, there are strong reasons for believing that the secretion of the saliva and pancreatic fluid is greatly impaired: hence starchy foods are not digested so well as albuminous foods; and I am of opinion that any considerable quantity of starch, given in the form of arrowroot, corn-flour, and sago, is injurious, and tends to cause intestinal irritation.

I now propose to consider some of the phenomena peculiar to typhoid, among the most important of which are the type and duration of the fever.

The latter appears at first sight to be extremely irregular, and to differ remarkably from the much more regular course of the other zymotic diseases. The causes of this irregularity are in the main two. First, we have in an ordinary attack of typhoid two distinct fevers—a primary, due to the infection of the system by the typhoid poison; and a secondary, caused by the gangrene and ulceration of the intestine, and consequent septicæmia. The first of these two processes has, I believe, as definite a course as the other zymotic fevers; the second or septicæmic fever is much more irregular.

The second cause is the frequency of relapses.

The fact that we have in typhoid two forms of fever to deal with is of considerable importance, both in the pathology and treatment of the disease, and it is one which I think is sometimes neglected.

Many other fevers of course present an exact analogy in this respect to typhoid; thus in small-pox we have a primary fever caused by the direct action of the small-pox poison, lasting usually about three days, then a period of remission, which is followed by a septicæmic fever due to suppuration. In scarlatina we have, in uncomplicated cases, a primary fever, lasting five or six days, but where there is ulceration of the fauces or implication of the glands a septicæmic fever of indefinite

duration, which usually comes on before the primary fever has subsided, but sometimes after a period of remission. In typhus there is usually no secondary fever, and the primary fever subsides by crisis about the fourteenth day. Now, typhoid resembles typhus in the long duration of the primary fever, and scarlatina in the fact that the secondary or septicæmic fever supervenes before the subsidence of the primary, the two processes overlapping, so that there is no intervening remission. The change, however, shows itself by an alteration in the type of the fever, which, from being continuous in character with only slight diurnal fluctuations, becomes remittent with marked diurnal variations. This is at once seen on the temperature chart of a case of typhoid where the fever has been allowed to run its course unchecked. This change in the type of the fever usually takes place during the third week, sometimes even as early as the fourteenth day. Generally it is a gradual change, but not unfrequently it takes place very suddenly, and at the same time there is a perturbation of temperature—often an evening rise exceeding that of the previous days, and followed by a considerable morning fall. This resembles the critical perturbations of those fevers which terminate by crisis, and probably indicates a kind of crisis of the primary fever. Not unfrequently profuse perspirations now appear, and an eruption of sudamina; while in the majority of cases the roseola of typhoid ceases to appear after the middle of the third week.

The phenomena of the disease are now made up of three factors—first, the local lesions; secondly, the septicæmia; thirdly, the high temperature. Sometimes one of these predominates, sometimes another.

The great resemblance which other forms of internal gangrene may bear to the later stages of typhoid fever was strongly impressed upon me by a case of gangrene of the lung which was under my care in the Middlesex Hospital in 1876. The patient was delirous, very prostrate, with a dry tongue, diarrhœa, great enlargement of the spleen, a temperature varying from 105° to 101°, sordes on the teeth, and well marked *tache.* There were the physical signs of capillary bronchitis of both lungs posteriorly, with patches of bronchial breathing. These signs were attributed to hypostatic congestion and lobular pneumonia. In this case all the signs of typhoid fever except the roseola were well marked, and for some days the diagnosis from this disease could not be made. The case was ultimately regarded as one of acute tuberculosis. After death several circumscribed patches of gangrene were found in both lungs, evidently caused by embolisms blocking branches of the pulmonary artery, these embolisms being derived from thrombi of the prostatic veins. The ordinary signs of pulmonary gangrene—fœtor of the breath, and expectoration—were absent throughout the whole course of the disease.

This question has some bearing upon treatment. And first with regard to the use of alcoholic stimu-

lants during the first stage of the disease, when the patient is suffering from the effects of the specific typhoid poison, some difference of opinion prevails as to the advantage of stimulants. Some authorities are of opinion that alcoholic stimulants in all severe febrile diseases have a beneficial effect by serving as a kind of food, and diminishing the febrile consumption of the tissues, and so tending to counteract the degeneration and consequent failure of the heart and central nervous system. Others, again, disbelieve in these effects, and only administer alcohol as a true stimulant when the heart actually begins to fail. But in the later stages of the disease, when we have to deal with a septicæmia due to gangrene and ulceration, if we follow the established principles of surgery, we can have no doubt as to the utility of alcohol. I believe all surgeons, without exception, treat external gangrene, accompanied by symptoms of septicæmia and depression, by free stimulation; and the fact that the gangrene is internal, instead of external, cannot make any difference in the treatment.

So, too, with regard to the use of certain drugs, especially opium. Some physicians regard opium as contra-indicated, on the ground that it produces constipation, and so tends to retain the decomposing fæces in the intestinal canal. Others, on the contrary, regard it as beneficial by checking the peristaltic movements, and so favouring the healing of the ulcers, and diminishing the risk of perforation. But, apart from its local action on the intestinal canal, it

is evident that, according to the principles of surgery, its use in the second stage of typhoid fever is often indicated. External gangrene and phagedæna are unquestionably benefited by opium, which renders the system more tolerant of their injurious effects; and there can be no reason for treating internal gangrene on different principles.

The duration of the primary fever due to the direct action of the typhoid poison is probably between two and three weeks, occasionally about fourteen days—*i.e.*, the same length as typhus,—but most commonly a few days longer. This is shown by the perturbations of temperature not unfrequently occurring at this time, and by the change of type of the fever from a continuous to a remittent form. Moreover, not unfrequently we see cases of what are known as abortive typhoid, in which the early stages of the fever are severe, the temperature very high, the onset sudden, and the febrile condition strongly marked; then about the fourteenth day defervescence rapidly takes place. The explanation of these cases is, no doubt, that though the poison has powerfully acted on the general system, for some reason or another—possibly some idiosyncrasy on the part of the patient—the local intestinal lesions have been slight, the infiltration of the glands only moderate, and the deposit has become absorbed instead of sloughing. These cases are exactly analogous to a very common form of modified small-pox, in which we have the primary fever due to the effect of the small-pox

poison on the system of great severity, but in consequence of the slight local affection of the skin the secondary or suppurative fever is absent.

In typhoid, as in small-pox, the intensity of the primary fever is not always a certain indication of the amount of the local mischief and consequent severity of the secondary fever; and, as in these abortive cases, the former may be severe and the latter slight, though usually a severe primary fever is accompanied by extensive infiltration and subsequent sloughing of the intestinal glands. The converse condition is much more common, in which we have a slight primary fever and mild constitutional symptoms early in the disease, with very severe intestinal lesions and consequent gravity of the later stages. Hence the well-known difficulty of forming a correct prognosis in the early stages of the disease, and the importance of treating the milder cases with the same precautions as the severe ones.

There can, I think, be little doubt but that cases of typhoid which are from time to time described as having been cut short by special remedies—as by emetics or calomel—are really of this character, the observer ascribing to the remedy what is, in fact, one of the natural phenomena of the disease.

Another point of interest in the history of typhoid fever is the mode in which defervescence takes place. In most of the zymotic fevers defervescence takes place either by crisis or a very rapid lysis. Thus, typhus, relapsing fever, uncomplicated measles, the

primary fever of small-pox, usually terminate by crisis; scarlatina, when uncomplicated, usually by a rapid lysis. Crisis, therefore, or rapid lysis would seem to be the natural mode in which zymotic fevers terminate. Typhoid presents an apparent exception to this rule. But here too, I think, we have indications that the primary fever tends to terminate in the same way; and the usual prolonged lysis is a phenomenon of the secondary or septicæmic fever. This is indicated by the perturbation of temperature so often present about the middle of the third week, and the rapid defervescence of the abortive cases.

The second cause of the variable duration of typhoid fever is the frequent occurrences of relapses. Now, relapses in typhoid take place much oftener than is usually supposed. They may occur at any time to as late as three or four weeks after complete defervescence, the longest apyretic interval being, I believe, thirty-one days. Most commonly it varies from one or two to fourteen or fifteen days. The frequency with which these relapses occur after an apyretic interval has been variously estimated. At the London Fever Hospital, during a period of seven years, they occurred in about 3 per cent. of the cases; in the hospital at Basle, 8·6 per cent. Other authorities give numbers varying from 1·4 to 11 per cent. This great difference depends, perhaps, in part on a different estimation of what constitutes a relapse; but there can, I think, be no doubt but that the frequency of relapses is much influenced by treatment.

If the fever is allowed to run its course unchecked, relapses are much less frequent than when the temperature is systematically kept down by cold baths and antipyretic medicines. Thus, at Basle, under the expectant treatment, about 8·6 per cent. of the cases relapsed. Since the introduction of the treatment by cold bathing this proportion has been nearly doubled. About 10 per cent. of the cases treated by me in the same manner have relapsed.

It is of course necessary to distinguish between a true and a false relapse. In typhoid fever, long after defervescence has taken place, the temperature remains labile, and the vaso-motor system very unstable, as shown by the continuance of the *tache*. I have long looked on the persistence of this latter phenomenon as a valuable guide and as indicating that the intestinal ulcers have not yet healed, and that therefore the patient is still liable to the sequelæ of the disease. As long as the patient continues in this condition of labile temperature and instability of the vaso-motor system, very slight causes, as errors of diet—nay, the mere resumption of solid food,—mental emotions, slight exertion, will send up the temperature; so any extension of the local mischief will cause a fresh febrile paroxysm. These cases must be distinguished from true relapses, where there is a fresh infection of the blood with the typhoid poison, fresh deposit in the glands, and commonly a fresh eruption of the roseola.

But besides these relapses which take place after a

distinct apyretic interval, and about which there can be no question, it is very common for the relapse to take place much earlier, during the secondary or septicæmic fever. These early relapses are often called recrudescences, to distinguish them from the later ones; but there can, I think, be no doubt but that they are of exactly the same nature. They are indicated by an exacerbation of the fever, and usually by the appearance of a fresh crop of rose spots, which commonly cease to come out after the middle of the third week. It is well known that the later relapses may be repeated twice, or even thrice; and the same thing also occurs with the earlier ones or recrudescences. Hence we may have an attack of typhoid of an indefinite duration without any distinct remission.

The most prolonged case which has come under my own notice was one in which the temperature was maintained at febrile heights without any distinct intermission for nearly eighty days. The patient was a gentleman of middle age, who had a very severe attack with prolonged delirium and great prostration, and in the third week pretty free intestinal hæmorrhage; he then had pneumonia of the right base, which did not clear up for several weeks; then there was a distinct relapse, with a recurrence of the hæmorrhage. This was succeeded by thrombosis of the veins of the lower extremities, on which some serious cardiac lesion supervened, the nature of which was not quite clear, but which I attributed to throm-

bosis of the right cavities. This caused alarming dyspnœa and a tendency to syncope, and was followed by general dropsy which lasted many months, but the temperature became normal about the eightieth day, and did not again rise. This patient ultimately recovered, but he could be considered as convalescent for nearly twelve months; and when I saw him two years later there were still some indications of obstruction to the return of blood from the head and upper extremities.

We may then, I think, regard those cases in which the fever lasts for many weeks without intermission as due to early relapse or recrudescence.

We have now to consider the pathology and causation of these relapses. There can, I think, be little doubt but that in relapse there is a fresh infection of the blood by the typhoid poison; and the opinion has been entertained that this takes place from without, either from emanations from other cases in hospital practice, or from the patient's own dejections where cleanliness is neglected or the sanitary conditions are defective. Relapses, however, not unfrequently occur among isolated cases, and where every sanitary precaution has been strictly observed. Hence there can be little doubt that the re-infection is usually an internal one, though an external infection is of course quite conceivable.

The opinion is widely prevalent that relapses may be caused by errors of diet, and doubtless they often follow such errors; but I believe this to be an acci-

dental coincidence. In the first place, relapses or recrudescences are very common before the fever has subsided and the appetite returned; secondly, they occur later in cases where it is quite certain that the proper regimen has been strictly observed; lastly, errors of diet during convalescence from typhoid are, I believe, the rule, and not the exception,—certainly this is the case among the uneducated classes, who are seldom willing to put any restraint upon their appetites. Even in hospital practice, where care is taken to exclude improper articles of food, everyone must be aware how often this regulation is evaded, and all kinds of indigestible viands are smuggled in. When the poor are treated at their own homes, as a rule, directly the appetite returns they begin to gratify it with whatever most takes their fancy; yet it is very doubtful whether relapses are more frequent under these circumstances.

Some time ago I had under my care in Middlesex Hospital a boy about fourteen years of age, who passed through a very severe attack of typhoid, and whose appetite returned before the temperature became normal. He continued of course to be restricted to liquid food. He complained to his mother, who remonstrated with me. I endeavoured to explain to her the danger of beginning solid food too soon. She replied that she would rather see her boy die of fever than of starvation. She accordingly took him home, stuffed him with solid food, and he recovered rapidly—much sooner, I have no doubt

than if he had remained in the hospital on a restricted diet.

In fact, these errors of diet, *i.e.*, the early resumption of solid food, are in most cases beneficial. After an attack of typhoid a large amount of tissue has to be built up. The return of the appetite shows that the digestive and assimilating powers are again active; and the more food the patient eats, the sooner will this be effected. Hence by continuing to restrict the diet we retard convalescence. We are of course unable to act upon this view, as we know that in a certain number of cases the intestinal ulcers are in such a state that the passage over them of any irritating particles might easily produce an extension of the ulceration, and so cause perforation; and as we have no certain means of distinguishing between those cases where the ulcers are granulating and not likely to be injuriously affected by solid food, and those where they are in an irritable or an atonic state, we are compelled to restrict the diet of all cases for a time, the evil of somewhat retarding convalescence being of trifling importance when compared with the risk of causing perforation.

I believe, then, that errors of diet, though they may cause a spurious relapse by increasing the local mischief, are incapable of re-infecting the blood with the typhoid poison, and so producing a true relapse.

Assuming the cause of the relapse to be the re-infection of the blood by a fresh absorption of the typhoid poison, there is every reason to believe that

this takes place from the mesenteric glands and the glands of the ileum, in which there can be little doubt but that the poison is deposited. This re-absorption probably takes place in all cases, but generally the system is protected by having just gone through an attack. In some cases, however, we must suppose that the zymotic process has not been complete, and then a relapse takes place; and it is remarkable that, when the fever is prevented from running its usual course by persistently reducing the temperature, the frequency of relapses is nearly doubled.

In scarlatina we have an exact parallel to the relapses of typhoid, though they occur much less frequently. During the past year four cases of relapse in scarlatina occurred in the London Fever Hospital at varying times from five or six days to a fortnight from the commencement of the primary attack, sometimes after an apyretic interval, sometimes before defervescence had taken place; and, as occasionally takes place in typhoid, the second rash has been more intense than the primary. As in typhoid, it is of course necessary to distinguish these true relapses with a recurrence of the eruption from the very frequent spurious relapses due to absorption of septic poisons from the throat or glands. In scarlatina, as in typhoid, there is very commonly infiltration of the lymphatic glands, and in all probability the relapse here also is due to re-absorption of the poison contained in them.

Syphilis also presents a close analogy. Here also we have reason to believe that the poison remains deposited in the lymphatic glands, and relapses are due to re-absorption from these; the great difference from the acute infectious fevers consisting in the fact that in syphilis the glands remain chronically enlarged, and hence relapses may occur after long intervals, whereas in typhoid and scarlatina the process runs a rapid course, and after the lapse of a few weeks, the glands return to their normal condition, and the poison is entirely eliminated from the system. In those fevers, as typhus, where there is no affection of the glands, relapses are hardly ever met with.

LECTURE III.

Treatment.

In estimating the value of any particular mode of treatment, we base our judgment on two classes of facts. First, by the observation of individual cases we see whether it appears to relieve the symptoms and exercise a favourable influence on the course of the disease. Secondly, we apply the test of statistics, and by collecting a great number of instances, we ascertain the effect on the general rate of mortality. Now, both these methods are open to many fallacies. In the first place, if the observer be not familiar with the natural course of the disease, and the various deviations which are liable to occur, he is apt to ascribe to his remedies what is really the unaided effect of nature; secondly, he may confound temporary amelioration with permanent benefit, and may thus incur the danger of relieving urgent symptoms at the expense of diminishing the chances of ultimate recovery.

Both these mistakes have frequently been committed in the treatment of typhoid fever. Thus the early recovery of the abortive forms has repeatedly been ascribed to the effects of remedies, as calomel or

emetics. Again, the great relief which bleeding often gives to the urgent symptoms of this as of other acute febrile diseases has in former times caused it to be extensively practised, with the effect of greatly increasing the mortality.

The second method is still more open to fallacy, so that it is often said in opprobium that anything may be proved by statistics. But, nevertheless, they form the final test by which all modes of treatment must at last be decided. The fallacies of statistics depend on two main errors; first, an insufficient collection of facts; secondly, the comparing together of unlike instances. We must, therefore, in applying this test, take care to compare together only like instances, and if possible accumulate such an overwhelming number as ultimately to reduce all sources of error to the vanishing point.

Before, then, we are in a position to estimate the value of any particular mode of treating typhoid fever, we must be acquainted not only with the natural course of the disease, but also with the usual rate of mortality, which of late years has, I think, been but little affected by treatment apart from nursing and feeding—the treatment generally practised being what may be called the expectant, or, as some would prefer to term it, the rational method. This consists in not treating the disease at all. We begin by confessing that we are quite unable to arrest its progress or materially to modify its course, and all we aim at is to put the patient in the most favourable condition

to support its effects. We see that he is carefully nursed and properly fed, all injurious influences are as far as possible removed, and urgent symptoms are combated as they may arise. Thus, if there be severe diarrhœa, we probably give him astringents; if hæmorrhage, styptics; if he cannot sleep, we administer narcotics; if there be delirium, calmatives; and when his strength begins to fail, stimulants; if there is congestion of the lungs, we perhaps venture on a mustard poultice or a turpentine stupe; or if there is severe headache, an ice-cap. But we leave the fever itself to run its course unchecked.

We have now to consider what are the results obtained by this mode of treatment. The most trustworthy statistics are those afforded by hospitals, but in them the rate of mortality is no doubt higher than in private practice. This is partly owing to the previous condition of the patients, partly to the fact that the more severe cases seek admission into hospitals, and partly because they commonly do not come under treatment till late in the disease, and nothing so much increases the mortality of typhoid fever as delay in the commencement of treatment.

If we take the results afforded by the London Fever Hospital, we may divide them into two periods:—1. A period of twenty-one years, during which the Hospital received all the pauper patients of the metropolis. 2. A period of nine years, during which the pauper cases have been excluded, and the patients have consisted of artisans, domestic servants, police-

men, *employés* in shops and houses of business, together with persons in a higher position who have occupied private rooms. During the first period 8,000 cases were admitted and 1,519 died, giving a mortality of 18·9 per cent. During the second period 590 cases have been admitted and 80 died, giving a mortality of 15·9 per cent. These figures include a considerable number of cases which were admitted moribund and died within twenty-four or forty-eight hours.

The Pauper Hospital at Homerton: cases 1,509, deaths 255; rate of mortality, 16·8 per cent.

The Stockwell Pauper Hospital: cases 1,223, deaths 301; rate 22·6 per cent.

The principal general hospitals of London, taking the last ten or twelve years, give the following results:—

St. George's: cases 387, deaths 76; rate of mortality 19·6.

Guy's: cases 295, deaths 57; rate 19·3.

University College: cases 163, deaths 29; rate 17·7.

St. Bartholomew's: cases 635, deaths 104; rate 16·3.

St. Thomas's: cases 445, deaths 70; rate 15·7.

Middlesex: cases 461, deaths 72; rate 15·6.

King's College: cases 318, deaths 39; rate 12·2.

Total for the hospitals of London: cases 14,125, deaths 2,522; rate 17·8 per cent. These figures no doubt include a considerable number of cases treated specifically.

If we take foreign statistics, we find that, in France,

Jaccoud, with a collection of 60,000 cases, gives a mortality of about 20 per cent.

The General Hospital of Vienna, with 17,000 cases, has a mortality of 22·5 per cent.

The Hospital at Basle, with 1,718 cases, a mortality of 27·3; and generally in the principal continental hospitals a rate of mortality varying from 16 to 25 per cent.

The English Army gives the following results for a period of six years ending 1877:—On home service, cases 545, deaths 131; rate of mortality 24 per cent. On foreign service, cases 1,383, deaths 564; rate 40·7 per cent. The foreign returns are not, however, complete.

In the Royal Navy during a period of six years ending 1878, cases 414, deaths 110; rate 26·5 per cent.

Such, then, are the results of the expectant treatment of typhoid fever, with which, I think, we have little reason to be satisfied.

It does not fall within the scope of these lectures to give a description of the various methods which have been from time to time introduced for the specific treatment of typhoid fever; they have most of them again, sooner or later, fallen into disuse, till at last a complete scepticism on the effects of treatment in this disease has been proclaimed by many of the highest authorities. Thus Scoda says it makes no difference whether we do or do not administer remedies; and the same opinion has been expressed by many others.

Of late years, however, a plan of treatment has been revived, which aims at controlling the fever and preventing its injurious effects on the organism by keeping the temperature throughout the whole course of the disease under a moderate febrile heat.

Typhoid fever can hardly be looked upon as a malignant disease: upwards of 80 per cent. of the cases, if properly nursed and fed, will recover; and those who die are rarely struck down at once, overwhelmed by the virulence of the poison, but usually succumb after a protracted struggle. Hence it seems only reasonable to suppose that if we can succeed in striking out one of the factors adverse to the patient, even if it be not the most important, this will generally suffice. So evenly is the balance poised in most cases between life and death, that the removal of the smallest weight from the opposite scale will give the preponderance to the side of recovery.

Now, the factor in typhoid fever which is most under our control is the febrile heat: it lies in our power to prevent the temperature throughout the whole course of the disease from rising above a very moderate height; and hence we may obviate those injurious effects which are directly or indirectly caused by the febrile state as such. The great acceleration of the pulse and breathing, the febrile oppression and malaise, the dry brown tongue, the thirst, the headache, the delirium, the stupor—may all be controlled. Nay, not only can we prevent the accession of these symptoms, but sometimes we can remove them after

they have supervened. And, as a result, the granular degeneration and softening of the various organs and tissues, and the febrile consumption itself, may be in part checked.

Therefore, from Hippocrates downwards, to reduce the temperature has at all times been regarded as one of the most important indications of the treatment of fever. It would seem, then, that all we have to consider is how far we are able to effect this, and whether the means which we employ are in themselves devoid of danger; and I think a sufficient amount of evidence has been now accumulated to answer both these questions.

With regard to the first, it can be experimentally shown that by the repeated application of cold to the surface of the body—chiefly by means of cold water on the one hand, and on the other by the administration of certain drugs which have the property of reducing temperature, the most important being quinine, salicylate of soda, and digitalis—the febrile heat can almost always be controlled.

Such being the case, the second and more important question arises, Is it always, or even generally, a safe and expedient proceeding? We must accordingly apply to it the two tests I have mentioned above—first, whether it seems to relieve the symptoms and exercise a favourable influence on the course of the disease; and, secondly, whether it diminishes the general rate of mortality.

The history of the introduction of what is now

called the antipyretic treatment of fever is a very interesting and instructive one. The first person who treated febrile diseases by systematically reducing the temperature by cold affusions was the celebrated Dr. James Currie, and he seems to have been induced to try this method by the account published by Dr. Wright of an outbreak of typhus on board ship. Dr. Wright himself caught the disease, and, finding that the cold air of the deck relieved the febrile oppression, caused himself to be treated by cold affusions, and with such good results that he applied the same method to a passenger at his own request.

Dr. Currie, as is well known, systematically treated by cold affusions, and sometimes cold baths, a great variety of febrile diseases, especially typhus, typhoid, and scarlatina, making careful thermometric observations, and he laid down rules by which we may still guide ourselves. It is interesting to see how soon he discovered that although, when applied late in the disease, this method would occasionally rescue the patient from a condition apparently desperate, nevertheless, to insure success it was absolutely necessary to have recourse to it at the very onset, before the fever had, as it were, got the upper hand.

Although he does not expressly remark the lability of the temperature in fever, he was, nevertheless, quite aware how easily it may be raised or depressed by external agencies; and he describes a striking instance of a case of typhus where he caused a

patient, who in his delirium had thrown off all his bedclothes, to be stripped of his shirt, and exposed, in a state of nudity, to a current of cold air from an open window. The effect was to reduce his temperature from 104° to 101°, to calm the delirium, and to throw him into a quiet sleep.

Dr. Currie soon had numerous followers, both in this country and on the Continent. Thus, Dr. Gregory applied the treatment with great success in small-pox; Desgenettes, in Egypt, during an epidemic of plague, having witnessed the unexpected recovery of a patient who, in his delirium, threw himself into the Nile, was led to try cold bathing, and with great success, in this disease.

Giannini, in 1805, became an ardent advocate of Currie's method. In 1813, after the retreat from Russia, a fearful epidemic of fever broke out among the troops and spread among the civil populations. At Berlin it raged almost with the virulence of the plague, and, in the hope of doing something to check ravages, Dr. Horn, the director of the Charité Hospital, determined to follow the plan of treatment recommended by Currie. The results were most satisfactory, and fully confirmed Dr. Currie's experience. Since this time treatment, though very imperfectly applied, has never quite died out in the Charité at Berlin.

In 1821, the celebrated Priessnitz, of Graefenberg, founded what is commonly known as the cold-water cure. He was a man who had all the qualifications

necessary for a successful empiric. He was entirely ignorant of science and medicine, of great common sense, self-confident, and a firm believer in his own system. In consequence he soon became the fashion, and all the celebrities of Germany crowded to Graefenberg to be cured of all manner of diseases. Now, there can be no doubt but that Priessnitz met with the most remarkable success in the treatment of acute febrile diseases, especially pneumonia. His plan was to immerse the patients in a shallow bath, and keep them in it sometimes for several hours—the effect being, of course, to subdue the febrile heat, and so relieve the most urgent symptoms. When we remember that the orthodox mode of treating pneumonia at this time was free bleeding, tartar emetic, and calomel, we must confess that the empiric was far in advance of his time.

Chomel practised the treatment in typhoid fever, Graves and Trousseau in scarlatina.

In 1850, Dr. Armitage, a member of this College, after visiting Graefenberg, with the consent of Professor Schoenlein, and in conjunction with Dr. Traube, treated a large number of cases of acute disease, especially typhoid fever, in the Charité Hospital at Berlin, again with the most remarkable success. And on his return to England he published the results in a small work called "Hydropathy in Acute Disease." It is impossible to read this book without at once seeing the great advantages of this method. Nevertheless, it failed to obtain the recognition of the

profession, and the treatment of Currie may be said to have died out in the land of its birth. The reasons for this are not far to seek. In the first place, it conflicted with popular and professional prejudices; and in the second, it is somewhat difficult to carry out.

Now, when facts conflict with prevailing theories they are pretty sure to be ignored; and the prejudice that patients with fever are injuriously affected by cold is perhaps more deeply ingrained in human nature than almost any other. This theory or prejudice is by no means confined to the vulgar; it is largely shared—often, no doubt, unconsciously—by the profession. I expect most practitioners would be horrified at the idea of exposing a patient, prostrate and delirious from typhus, to a cold draught from an open window, in a state of nudity; and yet by this means Dr. Currie at once relieved the threatening symptoms, and the patient made a good recovery. Usually when we are called to a case of fever we find the patient warm in bed, covered by several blankets, a good fire in the room, all draughts carefully excluded, and everybody anxious lest he should catch cold. I have seen a case of typhoid where the temperature was 105°, and there were five blankets on the bed. Now, in health it makes little or no difference to the internal temperature of the body whether there are five blankets or none, but in the labile temperature of typhoid fever it not unfrequently means a difference of 2°.

The revival of the antipyretic treatment must be ascribed to Dr. Brand, of Stettin, whose method, variously modified, has now been adopted by most of the leading physicians in Germany, Austria, and Switzerland; and the number of cases thus treated is now so large that the results may be fairly estimated. Dr. Brand, in the last edition of his work, has collected upwards of 8,000.

From Germany the treatment was introduced into France in a remarkable manner. Among the French prisoners in the late war who were confined at Stettin was Dr. Glénard, a clinical assistant at Lyons. An epidemic of typhoid broke out among the prisoners (23,000 in number), and ninety-three French soldiers were attacked. They were all treated by Dr. Brand according to his method, Dr. Glénard (certainly no partial witness) assisting, and they all recovered. Considering the very high rate of mortality of typhoid fever in times of war amongst troops depressed by hardships and dispirited by defeat and imprisonment, such a result naturally filled Dr. Glénard with enthusiasm, and on his return to Lyons he introduced this mode of treatment into the Lyons Hospital, again with the effect of greatly reducing the rate of mortality; and it has since been extensively tried by many of the leading French physicians.

In England, since Dr. Wilson Fox published instances of hyperpyrexia treated by the cold bath, its utility in this affection has been generally acknowledged; but the systematic treatment of fever from

its commencement in this manner seems at present only to have been tried tentatively in this country. Where it has been so tried the results have been most satisfactory. I may refer to the series of cases reported by Dr. Ord, in the *St. Thomas's Hospital Reports.* Though the treatment, according to Dr. Brand's view, was only partially carried out, the mortality was decidedly diminished.

Dr. Edes, of Boston, introduced the treatment into America, and he also speaks most highly of it.

Such, then, is a short and imperfect sketch of the history of this method of treatment. I now propose briefly to describe the manner of applying it, and the results obtained by it.

The principle underlying the treatment is to keep the temperature throughout the whole course of the disease, from its very commencement to its termination, under a moderate fever heat, and the means employed to effect this are, in the main, abstraction of heat by the application of cold water, supplemented by the use of the drugs I have mentioned, though Dr. Brand is of opinion that the more we confine ourselves to the former and the less we use the latter, the more favourable are the results obtained.

The modes of applying cold water are the cold bath, cold affusion, cold packing, cold compresses, and the graduated bath, which consists in placing the patient in a warm bath and gradually cooling it down. Each of these methods is adapted to special cases, but for general use the cold bath is by far the

best and the least troublesome to apply, and the rule for its use is this: whenever the patient's temperature exceeds 102·2° he should be put into a bath. It must be remembered that Dr. Brand always takes the temperature in the rectum. If we measure in the axilla, a somewhat lower height must be taken. He insists very strongly on the great advantages of rectum over axillary measurements, and there can be no doubt but that he is right; but I am so convinced of the impossibility of taking rectum temperature six or eight times a day in this country, even in hospital practice, that, except under special circumstances, I never attempt to do so. Next with regard to the temperature of the bath. This must be regulated according to the patient's condition and the effect we wish to produce. As a rule it should not exceed 70° Fahr., and may often be advantageously much lower; 65° to 70° may be taken as the average limits. The duration of the bath must also be regulated by the patient's condition and the effect it produces. We ought to cause a reduction of at least two or three degrees, and if this is not obtained the next bath must be colder or longer. It must be remembered that the temperature usually continues to fall for some time after removal from the bath; it is therefore not necessary to take the temperature in the bath itself, but it should be done about half an hour after. The bath should be prolonged for some little time after distinct shivering has begun, watching, of course, the effect on the circulation. Usually the

bath will be well borne for from ten to twenty minutes. The patient should then be put to bed, wiped dry and covered with a blanket. In many cases it is advisable to give some brandy or other stimulant before or during the bath.

In severe cases the cooling effect of the bath often only lasts about three hours; therefore the temperature should be taken every three hours by day and night, and whenever the temperature rises to 102° the bath should be repeated. The number of baths required will vary with the severity of the case. The rapidity with which the temperature rises after the bath is one of the best indications which we possess of the impression which the fever has produced on the nervous system. It would be a great mistake to suppose that the only or even the principal effect of the bath is to abstract a certain amount of heat from the body. It exercises a most powerful influence on the central nervous system, especially on the vasomotor centres, and stimulates them to a resumption of their heat-regulating functions. This is shown by a well-known experiment. Heidenhain found that stimulation of the sciatic nerve of a dog under curare caused a fall of nearly 2° Fahr. in the temperature of the internal organs, but that this fall did not take place if the animal was fevered; if, however, when in this state it was first cooled by being placed in a cold bath, the usual fall took place.

When, as is not very infrequent, we meet with cases of typhoid in which there is stupor and great

nervous depression, subsultus tendinum, and meteorismus, with only a moderate elevation of the temperature, the stimulating effect of the bath on the nerve-centres is of more importance than its refrigerating effect, and in such cases a cold bath of short duration, or cold affusion, is indicated. In other cases a more prolonged bath of higher temperature is required for the more complete abstraction of heat. As soon as the immediate effect has passed off, the patient should be lightly covered with a sheet and thin counterpane, and perhaps a blanket over the feet, but all general woollen coverings should be strictly forbidden. During the past cold winter I never allowed even a single blanket to a patient with typhoid in the Fever Hospital as long as the temperature was raised. In other respects the ordinary regimen should be observed. Abundance of cold drinks should be given; if there is headache, an ice-cap may be applied: if meteorismus or abdominal pain, a cold compress to the belly. Dr. Brand strongly recommends the use of cold compresses between the baths in all cases.

The other methods of abstracting heat are inferior in their effects, and are indicated in those cases where, from any reason, the cold baths are inadmissible. The graduated baths are useful in persons advanced in life, in cases of extreme prostration, where there is organic disease of the heart or lungs, where there is a tendency to syncope, or where—which is not often the case—the patient has an invincible repugnance to the cold baths. As an exclusive mode of treatment

they would be found impracticable, especially in private houses. Six or eight hot baths daily would be found to overtax the resources of most middle and lower class establishments: whereas the cold bath may be kept ready in the room, and the water need only be changed once or twice daily, a little warm water being added each time before it is used, or in hot weather some ice. Cold sponging and cold affusion are especially adapted to the milder cases, and the former together with iced compresses when, from hæmorrhage or peritonitis, we are afraid to move the patients. Cold packing is very inferior to bathing as a means of reducing temperature, and very troublesome to apply, but is sometimes more readily submitted to.

Liebermeister, Jürgensen, and most of the physicians who practise the antipyretic treatment, supplement the cold baths by the administration of those drugs which have the effect of lowering the temperature, especially quinine, salicylate of soda, and digitalis. Brand, however, deprecates their use, and only employs them where bathing is inadmissible. The great advantage they afford is that of diminishing the frequency of the baths, but they do not produce so favourable an effect on the nervous system, and sometimes themselves cause unpleasant symptoms. Their great use, then, is as adjuncts to the bath, but they cannot be employed generally as substitutes.

The most important of these remedies is quinine. It probably reduces temperature by diminishing oxi-

dation, its effect being to impair the oxygen-carrying powers of the red blood-globules. Quinine is very rapidly eliminated by the kidneys, and in order to obtain the antipyretic effect it is necessary that the blood should be highly charged with it. For this purpose, then, it is useless to give small doses at considerable intervals. One large dose should be given at once, or in divided portions within half an hour. To produce a decided fall of temperature in an adult, from thirty to forty grains are usually required. I generally give ten grains in suspension every ten minutes till the required amount has been taken. This will often depress the temperature three or four degrees, and this depression is often prolonged for many hours. It is best given at night-time, when the evening exacerbation is subsiding, or after the temperature has been lowered by a bath, as it exercises a greater effect on a falling than on a rising temperature. Given in doses of thirty or forty grains in typhoid, quinine often causes some of the disagreeable symptoms of cinchonism, as deafness and singing in the ears. These, however, are of little consequence; a far more serious effect is vomiting, which sometimes follows it use. This may be sometimes prevented by combining it with opium and administering ice.

Salicylate of soda, given in large doses, has a more powerful effect in depressing temperature even than quinine, and acts more rapidly. It has very largely replaced quinine in the treatment of typhoid fever in

Germany, but after a pretty extensive trial of it, it appears to me to possess all the disadvantages of quinine, and in addition it often has a very depressing effect, so that I now never give it where there are symptoms of prostration or failure of the circulation.

The only other of these antipyretic drugs of which I have any experience is digitalis, and it appears to be universally well spoken of. Dr. Murchison, who had very little faith in the advantages of quinine, says that digitalis is far too much neglected. The antipyretic property of digitalis is probably due to its action on the vaso-motor system. The primary effect is to stimulate the vagus and so to slow the heart; in larger doses it accelerates the heart by paralysing the vagus; and in still larger doses it brings the organ to a standstill by directly paralysing its muscular fibres. With these effects it causes contraction of the small arteries, and so increases the blood-pressure in them and diminishes that in the veins. This effect appears not to be due to its action on the vaso-motor centre, but to its direct stimulation of the peripheral nerves. Now this contraction affects the internal arteries, especially those of the mesenteric system, much more than it does the superficial vessels; hence the blood is diverted from the heat-producing to the heat-dissipating organs, and consequently the temperature falls.

Considerable care is required in giving digitalis as an antipyretic in fever. We must administer full

doses, or no effect is produced on the temperature; but we must be very careful not to go beyond the first degree of its action on the heart. When so given, it diminishes the frequency and improves the tone of the pulse, and tends to counteract that general arterial relaxation which is so marked in severe fever, and at the same it lowers the temperature. It is, however, a dangerous remedy where the heart is softened by acute granular degeneration, and therefore its use requires great caution in the later periods of fever, when the protracted high temperature has already caused more or less of this change to have taken place in the heart.

Such, then, are the principal means at our disposal for keeping the temperature down during the course of typhoid fever. We have now to consider what are the effects on the symptoms and course of the disease.

We may divide the symptoms of typhoid fever into two classes—first, those due to the general febrile state; secondly, those depending on the local lesions. With regard to the former, we may distinguish those cases where the treatment has been applied from the first, and the fever prevented, as it were, from getting the upper hand, and those where it has not been resorted to till all the symptoms of the typhoid state have been allowed to become fully developed.

In order to show the effect which we not infrequently obtain even in the latter class, I will briefly

narrate a case which has recently been under my care.

Arthur C., aged twenty-two, was admitted into the Middlesex Hospital on the afternoon of November 17, the twenty-first day of his illness. On October 26 he had been attacked by rigors, followed by the usual symptoms of fever, and on the 29th he took to his bed. On the following day diarrhœa appeared. On November 2 he became delirious, and had so continued more or less till his admission.

The case then seems to have been a very severe one from the first. The onset was sudden; the patient took to his bed on the fourth day, and became delirious on the eighth.

On admission he was very prostrate and in a state of stupor, from which, however, he could be roused by loud speaking. Pulse 144; temperature 104·6°. My clinical assistant ordered him to be sponged with cold water, and at 6 p.m., the temperature being still 104°, gave him twenty grains of salicylate of soda. The temperature fell to 102·2°, but at midnight was again 104°. He now administered thirty grains of quinine; this caused vomiting, which continued during the night. (I may say I never gave these remedies under such circumstances, where there is much stupor; far better results are obtained by bathing, with less risk of causing unpleasant symptoms.)

When I saw him at ten the next morning he was in a state of profound stupor, from which he could not be roused; his face was livid and dusky, pupils

dilated, muscles tremulous; pulse so small and irregular that it could not be accurately counted, but was about 160; respiration 36; temperature 103°. There were fine crepitant râles audible all over the chest, extending to the apices.

I ordered him a pint of champagne and eight ounces of brandy daily, and a mixture of hydrocyanic acid and soda to check the vomiting (which it did not do), and directed him at once to be put into a bath at 65°. He remained in it for fifteen minutes, and when taken out was shivering violently; this I regarded as a favourable sign, as it indicated that the nervous system retained its powers of reaction.

At mid-day he was still shivering, and though still in a state of stupor his general appearance had improved. At 2.15, the temperature having risen to 104°, the bath was repeated at 70° for ten minutes, and again at 4 p.m.; but this last had so little effect that another was given an hour later. At 7 p.m. a fifth bath was given, and at 11 p.m. a sixth. This frequent repetition was necessitated by the rapid rise of temperature and the recurrence of the alarming coma.

During the day he vomited everything he took. Feeding by the mouth had to be desisted from, and an enema of beef-tea, brandy, and opium was administered every three hours.

At 3 a.m. on the 19th a seventh bath was given, and at 6 a.m. an eighth. When I saw him in the

afternoon his condition was manifestly improved: the stupor had passed off; he put out his tongue when told, and tried to speak; the vomiting had ceased. Pulse was now 132, of better quality; temperature 101·6°. From this time a slow but steady improvement manifested itself, but the temperature continued to rise a few hours after each bath, and they had to be repeated at gradually increasing intervals. Thus during the first twenty-four hours eight baths were given, during the second twenty-four hours, also eight; during the third, six; during the fourth, four; and they had to be occasionally given up to November 30.

His recovery was much retarded by the state of his lungs, which were very slow in clearing up. Not only was there universal capillary bronchitis, but also, no doubt, lobular pneumonia, for when the stupor passed off, he began to cough and to bring up characteristic rusty sputa; but from his great prostration it was impossible to examine the bases of the lungs with any accuracy.

I may say that in this case the urine was highly albuminous; but, as the temperature fell, the albumen gradually decreased, and had quite disappeared before the baths were finally left off.

In the autumn of last year I had the satisfaction of successfully treating in this manner a member of our own profession. Here the symptoms were also of extreme gravity—delirium, stupor, universal muscular twitchings, not only of the extremities, but also

of the face, inability to protrude the tongue, accumulation of mucus in the bronchial tubes, an irregular pulse of 140, and albuminuria.

This gentleman's father, himself a physician of large experience in the treatment of fever, when the cold baths were proposed, said he had a horror of them; but the effect of the first bath was completely to convert him, and he afterwards encouraged me to persevere, when from the patient's extreme prostration my own resolution had begun to fail. In this case the temperature was never very high, only once reaching 104° in the axilla.

Such, then, are the effects which we sometimes obtain from the baths when they are resorted to in the later stages of the disease; but such results must necessarily be exceptional. We ought, therefore, rather to aim at preventing patients from falling into this condition, than trust to the very doubtful chance of rescuing them from it. Now, all observers who have tried this mode of treatment agree that when it is applied from the first, and the temperature steadily kept down by any of the modes I have mentioned, these so-called typhoid symptoms—the febrile oppression, the delirium, the stupor, the subsultus, the fuligo, the dry brown tongue—do not occur at all, or at any rate in a much slighter degree.

We have now to consider the effect of the persistent reduction of temperature on the local lesions, and especially the intestinal sloughing and ulceration to which the fatal event is so often due, usually from

hæmorrhage or perforation, sometimes more indirectly from inducing a state of marasmus.

With regard to perforation, we must distinguish between the two main forms of this complication. In the first place, we have the early perforations which take place during the detachment of the slough, and which usually occur during the third week, sometimes as early even as the twelfth or fourteenth day. These are due to the typhoid infiltration extending into and through the muscular coat, so that when mortification takes place, the peritoneal covering, whose nutrition depends on the integrity of the subjacent tissues, itself dies and becomes detached. In examining the intestines of patients who have died at this period of the disease, I have frequently observed small patches of the peritoneum, corresponding to the infiltrations, to be slightly opaque and yellow; and sometimes, in washing the intestine with a stream of water, these opaque patches of peritoneum will separate, exposing the subjacent infiltrated tissue. The perforations which thus occur from separation of the primary slough are sometimes of very large size, so that one may sometimes even put the tip of the finger through them.

The later perforations, which are liable to occur at any time after the separation of the sloughs up to the establishment of complete convalescence, are due to the typhoid ulcers not granulating, but becoming atonic and gradually extending. These latter perforations are generally very minute.

Now, whether the ulcers granulate and heal, or pass into the atonic state, depends chiefly on the general condition of the patient, and the less his system has been shattered and his tissues degenerated by the febrile process, the less likely is this complication to occur. Hence, any mode of treatment which lessens the injurious effects of the fever cannot fail also to diminish the frequency of these later perforations.

With regard to the early perforations, which are due to the depth to which the typhoid infiltration has gone, the effect of reducing the temperature is less obvious, and if the process is to be influenced at all it is evident that the treatment must be applied from the very commencement of the disease, and, when this is done, Dr. Brand claims that these complications never take place, and he has now treated in private practice 211 cases in succession without a single death or the occurrence of these accidents; but after the first few days, whatever the good effects the bathing may have on the fever, the control of the typhoid process on the bowel is lost, and the infiltration and sloughing will run their course unchecked.

Arterial hæmorrhage may, like perforation, be divided into two classes—the early and the late,—and their pathology and causation exactly correspond.

On the whole, the effects of the antipyretic treatment appear to me to be most favourable where the

general febrile symptoms predominate over the local intestinal symptoms, and least favourable where the latter predominate over the former ; but, according to the experience of Dr. Brand, provided the treatment be begun at the very outset of the attack, an equally favourable effect is produced on both.

Granting, then, the general efficacy of the antipyretic treatment, we have next to consider whether it is adapted to all cases and ought to be indiscriminately applied. To this I think we must answer that under no circumstances ought we to allow the temperature to maintain itself continuously above 102°. But the means employed to reduce it must vary with the condition of each case.

The most efficacious and most generally applicable of these methods—the cold baths—may be inadmissible. Sometimes the patient cannot bear them ; they may cause a dangerous depression or even syncope. As a rule, persons advanced in life and of impaired constitution are less able to support the baths, but these are the cases in which it is especially important to prevent the injurious effects of the temperature. If necessary, then, we must have recourse to the graduated bath, to cold packing, cold sponging, cold compresses, together with quinine and digitalis. The cold bath may likewise cause other unpleasant symptoms, as cramps and neuralgic pains. I have recently had under my care, in the Middlesex Hospital, one of the nurses, in whom the early symptoms were extremely severe. The temperature on the third day

exceeded 105° in the axilla, and it was with great difficulty controlled. She had twelve cold baths, but the last two caused very severe neuralgic pains, principally in the sciatic nerves, so that they had to be discontinued, and we were compelled to resort to the graduated bath, sponging, packing, quinine in thirty-grain doses; and by these means, after the obstinacy of the temperature had been broken, as it were, by the repeated cold baths, we succeeded in keeping it under.

Another question of great importance is whether pneumonia, bronchitis, and congestion of the lungs are induced or rendered more severe by bathing. I think to this question we may give a negative answer. In typhoid fever the former are probably due to the action of the poison, and the hypostatic congestion to the failure of the heart, and they are usually manifestly benefited by reducing the temperature. With regard to pneumonia, it is now the common practice in Germany to treat the idiopathic form of the disease in the same manner, and with excellent results.

Cold baths are generally regarded as contra-indicated in those cases where the patient is the subject of venous congestion from chronic bronchitis, emphysema, or cardiac disease. Dr. Glénard gives an interesting case of a man, aged fifty-three, one of the male nurses in the hospital at Lyons, who had been employed in bathing the typhoid cases, and was himself attacked. This man suffered from chronic bron-

chitis and emphysema, but was so convinced of the advantages of the bathing treatment that he insisted on its being applied to himself, and he took sixteen baths, but the later ones produced such alarming suffocative attacks that they had to be discontinued. He ultimately made a good recovery.

The occurrence of profuse intestinal hæmorrhage and of peritonitis is an absolute contra-indication to bathing, which indeed has been accused of rendering the former complication more frequent by inducing congestion of the intestinal mucous membrane and increasing the blood-pressure in its arteries. Whether it really produces these effects is, however, very doubtful. Dr. Glénard performed the experiment of exposing and opening the intestines of rabbits, and then immersing the animals in cold baths, taking care that the water did not come into contact with the intestine, and the effect was to blanch the mucous membrane. I need hardly say that we are constantly in the habit of treating internal hæmorrhages on the principle that the external application of cold causes a reflex contraction of the vessels of the subjacent viscera. In hæmorrhage from the lungs we apply an ice-bag to the chest; in hæmorrhage from the stomach, to the epigastrium; in hæmorrhage from the bladder or uterus, to the hypogastric region; and no doubt with good results. In all probability, where the general surface is cooled, as by a cold bath, the blood tends to accumulate in the muscular layers of the body rather than in the internal organs. Statistics

also show that bathing does not increase the frequency of hæmorrhage. The reason why hæmorrhage renders bathing inadmissible is the necessity of keeping the patient perfectly at rest, lest by any movements we should displace coagula or increase the rapidity of the circulation. In these cases, then, we must arrest the peristaltic movements of the intestine by opium, apply iced compresses to the abdomen, administer styptics as ergotine and digitalis, which latter also has the advantage of lowering the temperature. If, in spite of the hæmorrhage, the temperature again rises, we can give quinine and sponge the patient with cold water. In peritonitis rest is still more imperative, and we must have recourse to the same remedies with the exception of the styptics. Very commonly, however, both hæmorrhage and peritonitis themselves induce great depression of the temperature.

Another abdominal symptom remains to be considered in relation to the bathing, viz., tympanites. When great, not only is it in itself a cause of much distress and even danger, but it indicates either very severe nervous depression or serious local lesions. The actual cause of the condition is, no doubt, paralysis or loss of tone of the muscular coat of the intestine. Sometimes it comes on early, before the middle of the second week, and is then a most ominous symptom, and is an indication of a profound shock to the nervous system. This early tympanites is, I believe, most beneficially influenced by reducing the temperature by bathing and cold compresses,

together with free stimulation. The tympanites of the later stages of the fever is often due, in addition to the local lesions, either to actual peritonitis, or, at any rate, extensive and deep intestinal ulceration, and, as well as the local application of cold to the surface of the belly, demands the free use of opium. I cannot say I have ever seen much advantage result from the remedies usually recommended for this affection, such as charcoal to condense the gas, or stimulating enemas to expel it. The latter sometimes give temporary relief, but unless the tone of the intestinal wall be restored the gas soon reaccumulates. Attempts to draw off the gas by introducing a tube into the rectum only occasionally succeed. In order to do so, the tube must be passed quite into the sigmoid flexure—no easy matter to accomplish,—and moreover the gas is often contained in the small intestine.

Seeing how little influence the antipyretic treatment appears to exercise on the typhoid lesion of the intestine, when, as is so often the case, it is deferred till the end of a week or more, it becomes a matter of great importance to recognise as early as possible those cases in which the primary infiltration has gone very deep, and where, in consequence, there is danger of arterial hæmorrhage or perforation taking place when the sloughs separate, for probably the best chance of preventing these accidents is to arrest as far as possible the peristaltic movements at this period of the disease by means of opium. Unfor-

tunately we have no signs on which we can rely with any certainty. It has been said that twitching of the muscles indicates severe intestinal lesions, but this does not accord with my own observation. In a large proportion of the cases of perforation which I have seen, this sign has been absent; and from my limited experience of typhus I should say that muscular twitchings were much more common in this disease where there is no ulceration. On the whole, I think that great tympanites is the most valuable indication which we possess of this condition.

Another point of interest in connexion with cold bathing is the effect on the kidneys. Dr. George Johnson has shown that prolonged cold bathing will often induce a temporary albuminuria in healthy persons; it might therefore be anticipated that, in a disease like typhoid, the severer forms of which are so often attended by albuminuria, repeated cold baths would tend very much to increase the frequency and danger of this complication. Such, however, does not appear to be the case, and none of those who have extensively employed this mode of treatment have found any increased frequency of the rare complication in typhoid—acute nephritis. One such a case occurred this year in the London Fever Hospital, but this patient had never been bathed. A case of acute nephritis and uræmia in typhoid fever was admitted last winter into the Middlesex Hospital, under the care of Dr. Greenhow, but neither had this patient been bathed; and two similar cases

have been reported by Professor Immermann as occurring in the hospital at Basle, but in both these the affection was already present on the patient's admission. Hence, albuminuria need not, I think, be regarded as a contra-indication.

Another point of great interest is the effect of the treatment on relapses, and I think it cannot be doubted but that relapses are rendered more frequent. The probable causes of this I have already considered. In order to prevent these relapses, Professor Immermann has tried the effect of salicylate of soda, and he gives four to six doses of fifteen grains daily for ten or twelve days from the time of complete defervescence. Of fifty-one patients treated in this manner, only two relapsed, giving a ratio of less than 4 per cent., while of 234 patients treated at the same time and under similar conditions, but without the salicylate, thirty-three relapsed, giving a proportion of 20 per cent.

His paper has come into my hands too recently to enable me to try this mode of treatment on more than a few cases, but so far none of them have relapsed.

Against the general adoption of this mode of treatment it has been objected that in the great majority of cases it is unnecessary; that upwards of 80 per cent. of the cases, if properly fed and nursed, will recover; and that therefore so troublesome and even distressing a method ought to be reserved for the comparatively few cases which are likely to end fatally. This argument is not without force, and no doubt a

large number of the milder cases do not need repeated bathing, but it is notoriously impossible to predict at the outset, with any certainty, the course of an attack of typhoid fever, and any delay in commencing the treatment deprives it of a large part of its efficacy. A mild case of typhoid, or any feverish attack of which the diagnosis is at first doubtful, should from the outset be treated on the same principles, though in a less rigorous manner.

The patient should be confined to bed in a cool room with thorough ventilation and be only lightly covered, and from time to time may be sponged with cold water or have a cold affusion. Most persons in the upper and middle classes are accustomed to have a cold sponging or a cold affusion daily, and if they are feverish this will be still more grateful to their feelings; at the same time, abundance of cold drinks should be given to quench thirst and keep the mouth moist. If the case should prove to be one of febricula or simple catarrhal fever, no harm, but rather good, is done; if it turn out to be typhoid, we shall have obtained control over the fever from the first, and as soon as the temperature, in spite of these milder means of refrigeration, begins to maintain itself above 102°, and signs of febrile oppression manifest themselves, we can have recourse to the more energetic means of reducing it.

We have now to bring this mode of treatment to the test of statistics, and to inquire what is the effect on the general rate of mortality. First we must en-

deavour to compare together like instances, and this we are enabled to do pretty satisfactorily by contrasting the rate of mortality in the same hospitals before and after its introduction.

Thus, in the hospital at Basle, of 1,718 patients treated on the expectant plan, 469 died, giving a mortality of 27·3 per cent. Of 982 patients treated partially on the antipyretic system, 159 died, giving a mortality of 16·2 per cent. Of 1,483 cases treated thoroughly in this way, 130 died, giving a rate of 8·8 per cent.

In the hospital at Kiel, of 330 patients treated on the expectant plan, 51 died, giving a mortality of 15·4 per cent. Of 160 cases treated by Jürgensen by cold baths, 5 died, giving a mortality of 3·1 per cent.

In the military lazaretto at Stettin, of 1,591 cases treated before the introduction of bathing, 405 died, giving a mortality of 25·6 per cent.; since, of 121 cases, 5 have died, giving a rate of little more than 4 per cent.

Similar results have been obtained at Zurich.

Perhaps the most striking results of this mode of treatment were obtained in the field hospitals during the Franco-German war, which show a difference in the mortality between the ordinary and the antipyretic treatment of between 20 and 40 per cent. for the former, and 3 or 4 to 12 per cent. for the latter. It is interesting to compare with these numbers the rate of mortality in the English Army on home service in times of peace.

If we now take the general results as given by Dr. Brand, we find that of 8,141 cases treated antipyretically, 600 died, giving a mortality of 7·4 per cent., while the average rate in hospitals under the ordinary treatment is certainly not less than 16 per cent.

One result of this is, that while formerly the rate of mortality in the English hospitals was decidedly lower than that in the German hospitals—a result which we were disposed to ascribe to our better sanitary arrangements, better nursing, better feeding, and more free stimulation—it is now more than double.

I think, therefore, that it is impossible to doubt but that this mode of treatment considerably diminishes the rate of mortality.

I will now give briefly my own experience of this mode of treatment, which, however, in itself has been too limited to justify me in drawing any positive statistical conclusions, though it has been sufficient to convince me of its great advantages. At first I confess I was prejudiced against it; not that I ever doubted that it was advantageous to reduce the temperature in fever, but I regarded this repeated cold bathing as in itself impracticable, and, moreover, an unnecessarily severe proceeding. I therefore proceeded very tentatively, at first only giving a bath occasionally when the temperature was very high and the symptoms severe. Seeing the good effects which often followed, I gradually administered the baths more frequently, and supplemented them by quinine

and salicylate of soda and the other means of reducing the temperature; but it is only quite recently that I have endeavoured systematically to keep the temperature below 102·2°, and I have rather followed Liebermeister than Brand in the free use of quinine and the other antipyretic remedies, and have moreover largely supplemented the baths by sponging with iced water, cold packing, and ice compresses.'

Including all cases—viz., those which were only bathed occasionally, as well as those in which the method was more thoroughly carried out—I have treated 130 patients, of whom eighteen have died, giving a mortality of 13·8 per cent. This is not much below the average mortality, during the last decade, of the Fever Hospital in London; but it must be remembered that these were all severe cases, while the general rate of mortality is reckoned on the mild and severe cases together; therefore, the results are really very favourable.

Of these 130 cases, 44 were treated systematically by applying remedies to reduce the temperature whenever it maintained itself above 102° to 103° in the axilla. In the earlier cases, the higher maximum was taken; in the later cases, the lower. The means employed were cold baths, cold sponging, cold packing, cold compresses, quinine (usually in thirty-grain doses), salicylate of soda in sixty-grain doses, and sometimes digitalis. With the exception of one fatal case, I have excluded from this list all cases where less than ten baths or equivalent modes or methods

were administered. The largest number of baths given in any case was sixty-four.

All these cases were of great severity, and in some the condition was almost desperate when the treatment was commenced. Four deaths took place, giving a mortality of 9 per cent. Of these four fatal cases, in one, a patient in the Middlesex Hospital, the treatment was not begun till the eighteenth day; the patient was then in a state of great prostration, with delirium, stupor, and hypostatic pneumonia. She had thirty-eight baths.

The second case, treated in the London Fever Hospital, was a woman aged twenty-nine. The treatment was commenced on the tenth day. She had fifteen baths, was cold sponged fifteen times, and had nineteen thirty-grain doses of quinine. She recovered from the primary attack, relapsed, and died on the forty-second day. During the relapse she was only bathed once.

The third case, also treated in the London Fever Hospital, was a youth aged eighteen. The treatment was commenced on the eleventh day. Number of baths, eleven. Quinine given fifteen times. He was apparently progressing favourably. His temperature had fallen, and no bath had been given for two days, when he was suddenly seized with a violent attack of tetanic spasms with opisthotonus, after which his temperature again rose and his urine became bloody. He died of asthenia on the twenty-fifth day. After the attack of convulsions he was not again bathed.

The fourth case I have already mentioned in a former lecture. The patient died, on the fourteenth day of the disease, of acute granular degeneration of the heart. She got out of bed in the middle of the night and fell dead on the floor. She had only been bathed eight times, as her temperature was never very high.

Of the fourteen deaths which occurred among patients in whom the treatment had only partially been carried out, in eleven cases it was not commenced till after the eleventh day of the disease, and in seven of these not till after the fourteenth. In five cases only one or two baths were given, and the treatment therefore could not be considered to have been tried.

One case was admitted with acute meningitis and maniacal delirium, and died within thirty hours, and the surface of his brain was found covered with lymph. Three cases died of peritonitis, in two of which there was perforation; of these one died in a relapse, during which he was not bathed. One case died of hæmorrhage. One was the subject of valvular disease of the heart, of pericarditis, and pleurisy; in this latter case the treatment was not commenced till the twenty-ninth day of the disease, and he was only bathed on two occasions when the temperature became very high.

The remaining cases died of asthenia and hypostatic congestion of the lungs. No patient has died in whom the treatment was commenced before the

eighth day, and only two in whom it was commenced before the tenth. Of the whole number of 120 cases, thirteen, or 10 per cent., relapsed; one relapsed twice. Of the forty cases systematically treated, five relapsed; four of the thirteen relapses proved fatal.

I may add that last year, in which the treatment by bathing, though still only partially, was more completely carried out than in any previous year, the rate of mortality in the London Fever Hospital has been lower than has occurred since the exclusion of the pauper patients.

In conclusion, I trust I have brought forward sufficient evidence—not indeed to convince my hearers of the superiority of this mode of treatment—but enough to show that it is deserving of a fair trial, by the results of which it must stand or fall. Its objects all will acknowledge to be rational, and the means, when judiciously applied, are free from danger. Moreover, we may claim in its favour the weight of authority; and though we may not say in medicine, "Errare malo cum Platone quam cum aliis vera sentire," nevertheless, when we follow Currie, Gregory, Giannini, Horn, Chomel, Graves, Trousseau, Traube, Liebermeister, Brand, Jürgensen, Binz, von Ziemssen, Wunderlich, Chavanne, Cayla, we may feel some assurance that we are not wandering far from the right track. And I think it can hardly be doubted but that we have here a potent weapon, originally indeed forged in this country by a skilful

artificer, but allowed to get rusty from disuse; but which, again sharpened and polished by the great physician of Stettin, has been restored to our hands—a weapon, by whose aid we may well hope that we shall succeed in saving a considerable proportion of those 8,000 victims who every year in England alone succumb to the attacks of this treacherous disease.

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