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ON AN OUTBREAK OF TYPHUS FEVER  
WITH A DISCUSSION OF THE AETIOLOGY  
OF THE DISEASE

being

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by

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ON AN OUTBREAK OF TYPHUS FEVER  
with a Discussion of the Aetiology  
of the Disease.

INTRODUCTION.

"Every epidemic ought to have its own historian."

Sir Robert Christison.

"As a result of the gradual disappearance of typhus fever the younger generation of medical men have little or no acquaintance with the characteristic features of this disease, and few of the students who have graduated in Edinburgh during the last twenty years can have had the opportunity of becoming practically conversant with its clinical aspects."

Prof. Harvey Littlejohn.

"54 cases of typhus fever were notified in Scotland in 1908."

Registrar General's Report for Scotland.

The above obiter dicta are my excuse for this paper. Prof. Littlejohn's words exactly described my position when two years ago, being in charge of the Leeds Corporation Hospitals, typhus fever broke

out in the town and I shortly found myself with a series of cases on my hands. I soon had opportunity to repair my ignorance of its clinical features, and subsequent studies of the literature have induced me to make it the subject of an essay. The paper is not, however, an olla podrida of other writers, though there is little new to be recorded. I have described a rash occurring in a small boy such as I have seen described nowhere else, and in the part on Aetiology I have discussed and advocated the theory of flea carriage of infection, which had occurred to myself independently before I became aware that Hay had already enunciated it.

My thesis has been treated in several parts:

- I. Epidemiology of the Leeds outbreak.
- II. Its clinical features.
- III. The treatment adopted.
- IV. A discussion on the Aetiology of the disease.

#### I. EPIDEMIOLOGY OF THE LEEDS OUTBREAK.

"The belief that Typhus is spread by contagion has been attested by every writer during the last three centuries."

Stopford Taylor.  
M.o.H. Liverpool, 1884.

In these days when every considerable town such as Leeds is served by a skilled and efficient public health department epidemics are confined within narrow limits and all the important facts concerning them are soon known. This is so in the present case. Even though the cases are few, the history has many points in it common to much larger epidemics and they illustrate the essential features of the epidemiology of typhus.

The first cases were Mary and Agnes North, living at 17 Spring Street, West. They belonged to the lowest class, were very dirty in house and in personal habits, and were described to me by one of the inspectors as "only working at nights". In June, 1908, they were taken very ill. The prominent symptoms were headache and prostration, with pains in the back and limbs. They were not seen by a medical man and both recovered. They were nursed by Grace Hall who took ill in their house on June 28th and died on July 7th, without having had medical attendance. An inquest was duly held and the cause of death was given as influenza and cardiac failure. These events were discovered subsequently. In parenthesis it may be stated that the epidemic recorded by Littlejohn and Ker arose in similar fashion. The

early cases were not seen by a medical man and the illness was regarded as pneumonia or influenza.

The first cases which came to the notice of the Health Department were Mary Snee and her son. From June 15th-29th they had occupied the room in which the North girls had been ill and in which Grace Hall had died. They were admitted to the Union Infirmary on July 10th and to the Isolation Hospital on July 14th as typhus cases.

Of the other cases, James North slept in the same room in which Grace Hall had died on the night succeeding her death. He was admitted to hospital on July 24th with well-marked typhus. At the time of onset of his illness he was living in a common lodging-house containing some 360 beds. There were no secondary cases in this lodging house as far as could be known.

Herbert Mitchell, admitted on August 3rd, was an associate of North's. Both sold papers in City Square.

Mrs Smith, 21 Spring Street, West, and Mrs Nathers, 9 Spring Street, West, laid out Grace Hall's body. The latter took away with her the sheet which had formed Grace Hall's bed covering and used it, unwashed, for a similar purpose till August 1st when

she and her husband were admitted to hospital. Mrs Smith escaped the disease, but her sons, John and Robert Smith, were admitted to hospital on August 31st.

Perhaps the most interesting case of the series was that of Police Constable Teale. From July 25th-31st he was on special duty searching for stolen leather. His investigations took him to the infected houses. He then went on holiday to the country, visiting various places. During the later part of his holiday he felt far from well and took to bed on August 5th, and on August 13th he was admitted to hospital. No cases, so far as could be ascertained, arose secondarily to his case.

Prof. Littlejohn's remarks on the Edinburgh epidemic reported in the Edinburgh Medical Journal for 1899 might just as well have been written in description of these cases. He says: "If one point was brought out more clearly than another, it was that practically the disease is only communicated by direct contagion and that persons who are residing in the same tenement with an infected family, and who are living in conditions most favourable to receive infection, viz. dirt, squalor and intemperance, do not become infected unless they have been in

"close contact with the patient." He emphasised the fact that the "striking distance" of the disease was short and that actual contact with a previous case was essential; conversely that in no single instance did infection spread as long as there was no communication with a case. And he describes an instance in which "22 members of a family, living in "eight different tenements in widely apart places, "stuffed with inhabitants, suffered from the disease, "and yet in one instance only did another family in "the tenement become infected and it was proved they "were the only family in the tenement to enter the "affected house."

The Norths and Grace Hall, we have seen, infected North, the boys Smith, the couple Nathers, the woman Snee and her son. North infected his companion Mitchell, and very possibly Cannon, whose case has not yet been referred to, was infected by the girls North. Police Constable Teale, who entered the infected premises, contracted the disease, yet no case secondary to his could be found, nor did the boy North produce a fresh crop of infection in the large lodging house from which he was removed. These facts, though few in number, corroborate the principles laid down by Prof. Littlejohn. They are

the replica of experience in the history of modern outbreaks of typhus. Hay in Aberdeen, and Russell in Liverpool, quoted by Hay, give similar evidence. And in this matter modern experience is in accord with that of the observers of the past. Murchison emphasises these points again and again and brings as witnesses many of the great physicians of the early years of last century. Only in one point has an advance been made, and it is that secondary cases do not now arise in hospital due to our greater knowledge and more thorough practice of hygiene. Close contact, it is agreed then, is an essential condition for the spread of infection in typhus fever and this it will be shown is a feature of fly borne diseases. Ross has proved not once but many times that the removal of a camp a few hundred yards from the neighbourhood of stagnant water or from proximity to native lines protects it from the mosquitos which swarm there and the consequent risk of malaria. Close contact is also a feature in the spread of plague. Of interest in this connection is Murchison's view of plague. "Plague is perhaps the typhus of warm climates." He claims: "that there exists a strong analogy if not identity between typhus fever and true plague, the poisons being

"generated from similar causes and differing in intensity from the effects of climate and other collateral circumstances."

But Kala-Azar, a disease carried by bugs, is an even better example of a disease which is dependent for its spread on close contact between the sick and the healthy. Rogers in his Milroy Lectures, 1907, says: "Dr Dodds Price, suspecting the disease to be infectious, had placed 150 out of 200 freshly imported coolies in newly built lines of houses, while the remaining 50 had to be accommodated in infected lines from want of room. I got him to work out the results of this measure. It was then ascertained that although none of the 150 in the new lines had suffered from Kala-Azar during the two years they had been on this badly infected tea-garden, yet that no less than 8, or 16%, of those placed in the old lines were already dead of the disease, and that, too, in spite of the fact that the new lines were but 300 yds. from the old ones, a distance insufficient to prevent the spread of malaria through the agency of mosquitos." (Major Rogers was at this time attempting to show that Kala-Azar is not a form of malaria.)

This then is a house disease. But the same may

be said of typhus for, curiously enough, one year after the outbreak now under discussion another outbreak occurred in Leeds in precisely the same houses from which my cases came. This is no new fact, however, for Bateman observed "that successive occupants of the same dwellings often took typhus." Pringle, Lind and Jacquot give evidence to the same effect. This question will, however, be more fully discussed under Aetiology, Part IV.

## II. CLINICAL PHENOMENA OF THE LEEDS OUTBREAK.

### Age and Sex.

There were two women of 37 and 48 respectively. Of the males, two were boys of 8, three were youths of 17, 20 and 24, and three were men of 29, 33 and 42.

### Mortality.

Physique, according to Ker, is a matter of prognostic importance, especially in the case of alcoholic subjects. He considers that the greater the body weight and muscular development the more serious the outlook.

Of interest in connection with weight is an article by Dr Guelpa in the *Medico-Chirurgicale*, 15th December, 1909, in which he says: "Recherches expérimentales faites il y a vingt ans dans la clinique du Dr Dujardin-Beaumetz. Ce savant si actif, si original et si regretté, avait eu l'idée d'étudier les modifications du poids dans les affections aiguës et plus particulièrement dans la fièvre typhoïde.

"Les idées courantes de la médecine actuelle sur le besoin de soutenir les forces du malade auraient fait supposer que la maladie devait évoluer d'autant plus dangereusement que la diminution du poids serait plus prononcée. Or, il en est résulté précisément le contraire. Lorsque le poids ne diminuait pas, on pouvait être certain que la fièvre avait augmenté et que l'état du malade avait empiré."

The Leeds cases were spare and undersized with the exception of the policeman. None of them in any way suggested the brewer's drayman type. The youths were weedy slouchers, unemployed save at odd times as newsboys or casual labourers. One indeed was a pickpocket and was noted for the delicacy of his touch and the suppleness of his fingers.

Possibly this slightness of physique explains the fact that there was no death among the hospital cases. On the other hand all of them showed one or more of the grave signs which Murchison enlarges on - profuse rash, high temperature, severe nervous symptoms, early albuminuria and a pulse of over 120.

As regards the Incubation Period, much of the evidence is unsatisfactory. P. C. Teale's history is definite. At some date between July 15th and 31st he was exposed to infection. He became suddenly ill on August 5th and his crisis fell on August 15th.

As regards the other cases, the central facts are the death on July 7th of Grace Hall: that North slept in her room that night and was admitted to the Union Infirmary on July 22nd, 15 days later: that Mrs Nathers, who nursed Grace Hall, became ill on July 23rd and her husband on July 30th after sleeping since July 7th in Grace Hall's bedding: that Robert and John Smith, whose mother had also acted as nurse to Grace Hall, became ill not later than July 25th, their crises falling respectively on August 7th and 9th. In each case there was an incubation of about 14 days. The onset of the disease was in all cases sudden. Every case in which

a history was elicited complained of headache, pain in the limbs and back, and shivering, and these in the order named. Half the cases complained of vomiting, an unusually high percentage. Two complained of sore throat.

### The Clinical Picture

presented on admission varied, of course, with the stage of the fever; still the appearances presented appeared characteristic and unique. The patient lay on the back. The mind was confused. The face was bloated, in advanced cases cyanotic. The pupils were pinpoint and the conjunctivae injected and suffused. The tongue was thickly plastered in the early cases, small, almost shrivelled, cracked and with brownish crusts in the older ones. In the cases admitted at an early stage of the disease the rash consisted of rose pink raised spots disappearing on pressure and resembling typhoid spots. Soon they became brownish. In the other cases the so-called mulberry rash had appeared. It consisted of large irregular patches at first light in colour but soon presenting the appearance of an old bruise. These were always present and most distinct and persistent in the flanks, though they spread from there to the chest, abdomen and back.

True purpuric spots occurred in all the cases.

The pulse was frequent and the first sound of the heart very faint or inaudible. The respirations averaged 30. In several cases there was hypostatic pneumonia from the first. Constipation was the rule.

Certain of the chief clinical features of the fever were well illustrated in some of the cases and afforded excellent opportunity for observation.

#### The Rash.

##### G. Nathers, 33.

Sent to hospital with wife as a suspected case.

Admitted August 1st. History - headache, backache and pain in the limbs, shivering. Vomiting and constipation on the previous day.

August 2nd, 6 p.m. - Three or four raised pink papules on back.

August 4th, 11 a.m. - Numerous pink spots on flanks, back and chest.

August 5th, 11 a.m. - Typical measly rash out on flank.

August 6th, 6 p.m. - Rash well out, mainly pink in colour.

August 7th - Rash becoming darker.

August 10th, 11 a.m. - Petechiae abundant.

August 15th - Rash fading.

Robert Smith, 8.

Admitted July 31st from the Reception House where he had arrived that evening. Seen at 9 p.m. Temperature 102. Heavy eyed and somewhat dull. Rash out - faint subcuticular mottling on back, buttocks, flanks and chest. Numerous vibices.

August 2nd - Big wheals, urticarial in type, pale pink in colour and hotter to the touch than the surrounding skin, appeared on buttocks, neck, arms and legs some time between 8.30 and 10.30 a.m.

August 2nd, 12.15 p.m. - Urticarial rash gone.

August 2nd, 2.30 p.m. - Urticarial rash on buttocks and thighs. Pink mottling on arms, apparently beneath the skin and only disappearing partially on pressure. Erythema on brow, cheeks and chin.

August 3rd, 11 a.m. - Urticarial rash on buttocks and thighs appeared and disappeared twice during the night. Again present now, but only one spot on buttock. Wheals are grouped chiefly on abdomen, arms and hands, about knees and extensor aspects of thighs. Red spotty rash on face.

August 4th - Rashes gone. Mind clear. De-fervescence.

Nervous System.

Frontal headache and mental cloudiness were constant initial symptoms. In one of the women, the mildest case of the series, headache was most persistent and troublesome. Delirium was present in all the cases, violent at first and requiring restraint, later muttering and feeble. In one bed lay a policeman intent on an arrest and attempting to carry it out. Beside him lay a pickpocket earnestly trying to escape from the restraining bandages which kept him in bed. Even four days after his crisis this patient suffered from dizziness, delusions, insomnia and restlessness.

Herbert Mitchell shewed on admission, the 8th or 9th day of his illness, very marked subsultus tendinum and tremulousness of the lower jaw and facial muscles.

Involuntary action of the bowels and bladder occurred in several cases for several days.

There was no case of retention of urine so severe as to require the use of the catheter.

Of all the nervous phenomena the most intractable was insomnia. In the case of G. Nathers this gave rise to considerable anxiety. Early remedial measures were unavailing. Restlessness increased

and this rendered sleep more difficult to attain. Thereafter, it was realised that the loss of a night's sleep affected the prognosis of typhus very adversely and anticipatory medicinal interference was had recourse to.

#### Circulatory System.

The heart sounds in many cases were faint, the first sound often inaudible. In more than half the cases the pulse rate in the first week was over 120, Murchison's dangerous rate. In the second and third weeks the circulatory depression seen in a number of cases was a surprise to one who had never seen typhus. In one case, complicated by a mitral systolic murmur, the highest rate recorded in four and a half days was 48. One day the rates returned morning and evening respectively were 42 and 40, and the next day the pulse was not palpable. For a week thereafter the rate oscillated between 48 and 68. The value of the pulse records is small as they were rendered fallacious by the large amount of stimulant of various kinds exhibited.

#### Respiratory System.

The respiratory rate in all the cases oscillated between 30 and 40. In all but two cases rates

were noted, first at the bases and later all over the chest. In the case of G. Nathers there was a tuberculous vomica at the apex of the right lung. His crisis was delayed to the 17th day. The temperature oscillated thereafter for three weeks, sweats were profuse, cardiac collapse extreme, and insomnia most troublesome. This was the most dangerously ill case of the series.

#### The Fever.

The annexed charts show the temperature curve in some of the more interesting cases.

The day of the crisis varied considerably. In those cases in which the history has been found reliable the day of the crisis fell as follows:

Case 1	=	15th day.
Case 2	=	15th day.
Case 3	=	14th day.
Case 4	=	16th day.
Case 5	=	12th day.
Case 6	=	10th day (8 years old).
Case 7	=	16th day.

In Case 1 the crisis was preceded by vomiting and diarrhoea, in Case 3 by epistaxis, and in Case 7 by profuse sweats, while at the same time the brow, bridge of nose and malar eminences were markedly flushed. Wunderlich, quoted by Pye-Smith, states that the temperature on the first evening is

generally 104-104.9, and that on the fourth evening it is seldom less than 104.9, generally about 105.8 and even higher still. In none of my cases was there a temperature of that degree while they were under observation. It may be that the free ventilation of the wards, which was regarded as of prime importance, may have had some influence in keeping the temperature down and so serve as some explanation of the difference. In all cases the post-critical temperature was, like the pulse, unnaturally depressed. The most noticeable case in this respect was that of Mary Snee, whose post-critical chart is appended.

#### Gastro-Intestinal System.

The tongue presented a difference in appearance almost daily. In the early stages there was a thin, later a thick, white fur centrally, while the margins were pink. This fur became by gradations yellowish and then brown, finally dry and brown and coated with crusts which gradually separated in flakes and helped to compose the large amount of sordes present. The margins and tip remained clean, the latter becoming pointed. In the second week the tongue presented a shrivelled appearance. With the establishment of convalescence the tongue became

moist and cleaned rapidly.

In the case of Herbert Mitchell, admitted on the 9th day, and in whom nervous symptoms, muscular twitchings and prostration were prominent, the tongue had a red glazed appearance and the superficial epithelium had disappeared.

In seven cases enemata were required from the first on account of constipation. On admission one case had diarrhoea and in one there was abdominal pain. In this case the liver was tender and reached  $1\frac{1}{2}$  in. below the costal margin. The spleen also was palpable and tender. The same observations as regards the spleen were made in another case of the series.

Diarrhoea and vomiting as precursors of the crisis have been referred to. In certain of the cases vomiting was a troublesome feature of early convalescence.

The urine was scanty and high coloured throughout the fever. In three cases albumen was present from the time of admission.

The Typhus odour so often described and regarded by so many authorities as diagnostic was not noticed in any of the cases.

Complications.

Rales were present in the majority of cases and in some there were definite patches of hypostatic pneumonia. One case was complicated with an organic mitral murmur and another with phthisis. This case in particular shewed the pre-critical sweating which Ker considers of grave augury.

Mary Snee, the first of the series, had large bed sores on back and buttocks on admission. When taken in the first instance to the Union Infirmary she was found to be in an indescribably filthy condition. On the 13th day of her illness she developed a parotid bubo on the right side which had not entirely disappeared on her discharge from hospital a month later. It did not suppurate. In contra-distinction to the other cases, she was slightly affected with diarrhoea on admission. According to the history she had been so affected for 12 days.

III. TREATMENT OF THE LEEDS CASES.PROPHYLACTIC TREATMENT.

*measures* (9)

Disinfection of infected homes, bedding, etc., was carried out by the officers of the sanitary staff. It included stripping the paper off the walls, lime-washing them and cleaning and washing the floors and furniture. The staff used no special measures for personal protection, nor did any of them become infected. This was a piece of good fortune, however, as even yet ambulance officers, sanitary inspectors and hospital laundry women are frequent victims of the disease. Hay's method of prophylaxis should accordingly be used. His plan is to have as little exposed surface as possible, to make all garments close fitting, and to lubricate all bare parts (hands, neck and wrists) and portions of the clothing (the boots, sleeves and collar) with a eucalyptus solution. After adopting these measures the men were no longer plagued with fleas, nor was there another case among them.

Every advantage was taken of the Reception House for contacts. It lies in the grounds abutting on the hospital so that the inmates were constantly under the observation of the medical staff.

Certain inducements in the way of payment of rent and a fractional part of the weekly wage were offered so that no difficulty was experienced in persuading the people to enter the house.

Patients' clothes and linen were hung up in the open air outside the wards for several hours before removal in a disinfecting solution to the laundry.

The wards used are normally ten bedded, but only six beds were occupied to allow of ample air space. All ward windows were kept rigidly open, so much so that frequently papers could only be kept on the ward table by means of paper weights. The patients never complained of discomfort on this account. Of this line of treatment Curschmann thought highly. "I cannot say too much in favour of the "open-air treatment. The patients became quiet and "the symptoms of the initial stage - the violent "headache and the insomnia - were more favourably "influenced by the open-air treatment than by any "other means." At the same time he regarded it as "the most effectual means of guarding against the "spread of the disease." Long ago Sir Henry Littlejohn pointed out that it was essential to place typhus patients on the top floor of a hospital, as typhus infection ascended. Our wards were all in

single floor buildings.

The nurses employed volunteered for the work. Only the best physically and professionally were chosen. There were no cases among the attendants and only one showed signs of the typhus headache Murchison describes.

Nourishment. Milk diluted one third with barley water was the routine food, and as much was given as the patient could be persuaded to take. The feeds were two hourly. The patients were also encouraged to drink water or soda water as frequently as possible, the amount varying with the nursing skill. In the bad cases egg white and egg-flip were given and in one case saline enemata. Benger's Food and beeftea were also used.

The aim was to flush the body of toxins, to sustain the strength during the height of the fever and to stimulate recuperation with ample and frequent meals thereafter.

#### MEDICINAL TREATMENT.

Physical signs pointed to certain conditions requiring relief. These were mainly cardiac weakness and nervous phenomena. Circulatory depression was extreme in four or five of the cases. In all

the cases of the series brandy was used in amounts varying from two to six ounces in twenty-four hours; four ounces seemed the most useful dose, the higher amount being suspected of increasing the nervous excitement.

Strychnine formate, gr.1/100, was exhibited hypodermically, and to tide over periods of weakness, the description of which resembled syncopal attacks and which I had no opportunity of seeing as they usually occurred in the early hours of the morning, a mixture containing M.5 of each of the following was given: Spiritus ammoniae aromaticus, spiritus aetheris, spiritus chloroformi, and to which Tr. Digitalis M.5 was added.

Coffee enemata were tried in one case.

In the milder cases of sleeplessness and restlessness, sulphonal, gr.10, sufficed. In the more severe cases, chloral, gr.20, and pot. brom., gr.15, were exhibited, at times without effect. On account of the marked cardiac weakness presented by all the cases these were speedily given up, and resort was had to morphine. Any disadvantages it may possess were far outweighed by its certainty of action, if given in sufficient doses, and consequent avoidance of the debilitating effects of prolonged insomnia and restlessness.

During convalescence iron and quinine or acid and nux vomica mixtures were relied on.

#### IV. AETIOLOGY.

Typhus is one of the diseases whose aetiology has not yet been decided. The causes of this are not far to seek. It has ceased to be a common disease. Though it has not been stamped out with the dramatic completeness of plague, which has not been seen as an indigenous disease in this country since 1679, it is so rare that only the medical officers of isolation hospitals in a few towns have an opportunity of studying it. The last extensive outbreak in this country was in the years succeeding the Crimean War. At that time bacteriology was in its swaddling clothes, and the de novo theory of disease, of which Murchison was a stalwart champion, was generally accepted. It was most prevalent then when medical science was less accurate than now, when the most advanced thinkers alone ventured to suggest a living cause for diseases, a cause which they guessed

at but could not demonstrate. Of these the earliest, claims Monti, was Hildebrand of Vienna "who compares in a very precise way the unknown agent of the disease (typhus exanthematicus) to a vegetable or animal germ."

Again it and other fevers were held to be one and the same disease and were classed as continued fever. Before progress could be made in studying the individual disease, differentiation was necessary.

For centuries it was known by a hundred different names derived from its prevalence in prisons, hospitals, ships, in camps and armies, from its symptoms (typhus comatosus), signs (spotted fever), supposed aetiology (ochlotic fever), from its epidemic character (febris epidemica), its contagious nature (febris pestilens and contagious typhus), and from the countries where it was endemic (Irish ague, Hungarian fever). This also fostered confusion. In England it was associated with the plague and so over-looked till that disease disappeared from London in 1666. It was not till 1843 that Henderson differentiated it from relapsing fever: and it was only after 50 years of observation and discussion initiated by Erasmus Darwin that Jenner was able to

lay down the differential diagnosis between it and typhoid between 1849 and 1851. From the former year these diseases were differentiated at the London Fever Hospital, but it was twenty years later before the differentiation was made in the Registrar-General's Report, and by that time typhus in England was on the decline. It should be pointed out, however, that typhoid was not introduced into England, and more especially into Scotland, until well on in last century. Confusion arose in that French physicians were describing typhoid, their endemic fever, while typhus was the endemic fever of England. Both sets of observers imagined they were describing the same fever and used the same names. Clearer views prevailed in Germany, where Hildebrand introduced in 1810 the terms typhus exanthematicus and typhus abdominalis. In Scotland Christison writing of the 1817-20 epidemic said: "Of enteric typhus we saw "nothing, nor for many years afterwards," and remarks that if it had been missed during life it could not have been overlooked after death, because "to meet the bias of the day for finding a local "cause for all fevers every important organ in the "body was habitually looked to".

Goodsir seems to have been one of the earliest Scottish pathologists to find typhoid ulcerations.

He had a paper "On a diseased condition of the intestinal glands" in the London and Edinburgh Monthly Journal of Science, April, 1842. It was founded on dissections he had made on fever cases while assisting his father in Anstruther in 1835-39.

He described the lesions in Peyer's patches, but did not claim to have discovered a new disease, though he wondered whether it was "merely a form of "the ordinary continued fever". So few were the cases that Reid, pathologist to the Edinburgh Royal Infirmary, found that only in two out of forty-three post-mortems were the patches elevated and "seemingly ulcerated".

Little wonder then that with confusion of diseases and terms and the decline of the disease since the era of modern scientific medicine, that the cause of typhus has not been solved.

#### THEORIES ON THE NATURE AND CAUSE OF TYPHUS.

It was not till the Restoration that typhus attracted the attention of English physicians. Willis, the earliest of these authorities, described in 1661 three fevers, putrid, malignant and pestilential, according to the degree of severity. It was an epidemical fever "chiefly infestuous to the

brain and nervous stock". It was an unaccustomed fever, though he says he had seen it before in 1657-59 in Oxford. He is the first to describe it, though he says nothing as to aetiology. Pepys in his diary also refers to it as a new fever.

Aug. 16, 1661. "At the (Navy) office all the morning though little to do..... But it is such a sickly time both in the city and the country (of a sort of fever) that never was heard of almost unless it was in a plague time."

Aug. 31. "The season very sickly everywhere of strange and fatal fevers."

Sydenham is the great physician and epidemiologist of the time. He describes it as a new fever. In those days insanitary conditions were not regarded as of medical importance, so that looking for a cause he ascribed it to telluric and atmospheric conditions. Robert Boyle, his friend, elaborated the theory of miasmatic influence in disease, a theory still later developed by the Munich School into the so-called Grund-Wasser theory. This was the theory that Sydenham adopted, a theory of "epidemic constitutions". From Evelyn's diary we find that the winter of 1683-84 was one of intense frost - an ice carnival was held on the Thames during the whole of

January. The long dry frost of winter was followed by an excessively hot and dry summer, the drought being such as he did not remember. The winter of 1684-85 set in early and became a long cruel frost. The spring was again dry, and it was not till May 1685 that the two years long drought ended. The long drought and frosts affected the ground water and must have produced, thinks Sydenham, "some secret and recondite changes in the bowels of the earth pervading the whole atmosphere or some influence of the celestial bodies." Further Sydenham remarks on the similarity of these telluric conditions, frost, long winters and drought, to the years preceding the bad plague years of 1624-5 and 1665-6. This was an idea generally held. Pepys refers to the disease only occurring before at plague time, and a letter of the period says "a fever rages that proves very mortal and gives apprehensions of a plague." For years thereafter Sydenham's ideas dominated medical thought and practice. It was only in the latter portion of the first half of the 19th century that modern methods of research began to be used, that the stethoscope, the thermometer and the test tube were discovered as useful adjuncts to the eye, hand and experience to which the earlier

school of physicians had trusted. It was the time when medicine always an art became an exact science as well. The dominating personality in these days in Scotland was Sir Robert Christison. Born and trained under the old regime he lived a professional life of 60 years and combined in himself the medical thought of two generations. His evidence then on the fevers, and typhus in particular, is essentially unique. It became common, if his evidence is accepted, during his younger days: it had declined to insignificance at his death.

He held there were three types of continued fever seen in the Edinburgh epidemic of 1817, -

1. Cullen's synocha, now known as Relapsing fever:
2. Cullen's typhus mitior, to his mind a mild type of Typhus. Cullen's typhus gravior was rare, only one doubtful case occurring in his practice.
3. A form intermediate between the two - "exactly the features of Cullen's synochus". "There was no enteric typhus," he says, "nor for many years afterwards."

In 1827-9 the type of the fever was similar. But in 1833 a change in type was noticed. The fever became "strongly phlogistic" in character and the treatment of bleeding was given up. In 1842-4 the

type was synocha, and of all Edinburgh physicians he alone was able to identify it with the fever of 1817. Writing in 1840 he declared his belief that all varieties of fever were merely dependent upon the epidemic constitution of the period and were in no respect specifically distinct from one another. In the Edinburgh Medical Journal of 1858 he writes of synocha, typhus and synochus, "the intermediate form", as one disease. So late as a short time before his death he wrote to Carpenter that as regards typhus and typhoid he considered them "not as uniformly marked out from one another, by well defined boundaries, but as shading off graduationally into one another" (quoted by Gairdner from paper by Carpenter in the 19th Century for Feb. 1884). Such then was the belief of the last and greatest of the school of Sydenham.

The conclusions of Murchison, a man of the more modern school, and one who made the study of fevers his life work, will now be quoted.

- "1. Typhus is due to a specific poison.
- "2. It is communicated from the sick to the healthy through the atmosphere or by fomites, but is rendered inert by free ventilation.
- "3. The poison is also generated denovo in the exhalations of living human beings by overcrowding and bad ventilation.

"4. The great predisposing cause of typhus is defective nutrition."

Nothing authoritative or generally accepted has been added to this pronouncement of Murchison's.

Judging from analogy many writers have argued that there must be a specific micro-organism, but this has not yet been isolated. The bacteriologists have not been idle, however. Strepto-bacilli were found in Prague by Hlava in 1891, and Lewaschew in 1892 described small motile bodies of varying shapes. Kasan also found in the peripheral and splenic blood rounded bodies with filiform prolongations possessing movement which he called *Spirochaeta exanthemica*, a most suggestive observation in the light of present day knowledge of the pathogenicity of various amoebae. Dubief and Bruhl studied an epidemic in Parisian prisons in 1893. In several of the post-mortems they found a diplo-coccus in the lungs and bronchial secretion, and in infarcts of other organs. From the blood of some living patients a similar body was obtained, but it was impossible to cultivate it.

Balfour and Porter working in Edinburgh in 1899 isolated a diplo-coccus from thirty-six out of forty-three cases which closely resembled the one found in

the Parisian prisons. It was small and fragile, capsulated and non-motile, readily stained and retaining Gram's stain. They cultivated it from the blood and when obtained post-mortem they were able to grow it on blood serum, a point in which it differed from the Parisian diplo-coccus. But these observers had doubts as to whether their organism was not derived from the skin, and their work is inconclusive.

6  
THE HYPOTHESIS OF MATHEW HAY.

In 1907, after studying an epidemic in Aberdeen, Hay enunciated an hypothesis which may possibly solve the problem. The analogy of malaria suggested the possibility of flea-carriage of infection. He supported his hypothesis with, among others, the following considerations:-

- "1. Every case exhibited flea bites and those of the staff who complained most bitterly of flea bites were those attacked.
- "2. The disease in no instance spread in a family of clean habits, even though a case had lain in the house through most of his illness, and in two cases without any attempt at isolation.
- "3. Every nurse and ward maid in the city hospital who had been attacked was,

with two exceptions, employed in removing patients to the hospital or in cleansing or bathing them on admission. No nurse or ward maid who did not assist in removing or cleaning the patients, however intimate the contact was otherwise, was attacked.

- "4. Precautions, after the attack of the driver, were taken. Topboots, and overalls, close fitting at the neck and wrist, were worn, and necks, wrists, ankles and tops of boots were smeared with eucalyptus in olive oil. There were no further cases and no longer, practically speaking, did the staff suffer from flea bites."

Hay supported his theory from the experiences of others. Thus Dr Russell, in the Liverpool fever hospitals, has remarked on the curious freedom of wardmaids from attack, also that typhoid cases in the same ward did not take the disease. Further, during the period of three or four years in a Liverpool hospital where 800 cases were treated, no doctor or student took the disease.

"This theory," says Hay, "explains why typhus is confined to the poorer classes, though attacks on nurses show that other classes are not insusceptible." "Poverty," he continues, "is not the cause; many cases were well fed and clad, but dirtiness and vermin were always present."

THE ARGUMENT FROM ANALOGY.

In ignorance of Hay's work the same idea occurred to me, too late, however, for undertaking experimental work, had facilities for the same existed. In common with Hay it was the analogy of malaria which suggested this hypothesis of insect carriage of infection. An attempt to elaborate this line of argument and to support the hypothesis by reference to the recorded features of previous epidemics will now be undertaken.

Pasteur and Lister established the pathogenicity of bacteria; and Laveran of haematozoa. Carriage of infection by ectozoa has more recently attracted attention. Finlay of Havana was one of the early pioneers. He believed that mosquitos carried yellow fever, and that it was not merely a simple mechanical carriage of infection. The American Commission working on this hypothesis in 1909 announced the *Stegomyia fasciata* as the carrier, a discovery confirmed later by the Pasteur Institute Expedition, which has been acted on with the most brilliant results.

Manson, thinking of Finlay's theories and his own work on filariae, inspired Ross to study the question of mosquito carriage of malaria in India.

His epoch making discovery in 1897 may be said to be the foundation of all modern work on disease-carrying insects. Since then disease producing piroplasmae, passing part of their life cycle in ticks, have been found in the blood of animals and questionably in a "spotted" fever of man in America. Trypanosomes are found in fish, birds, batrachians and mammals, but most notably of all in the sleeping sickness of man. Its cause, *T. Gambiense*, is carried by *Glossina palpalis*.

In animals we have the so-called tsetse fly disease, Ngana, due to *T. brucei* and mechanically carried by *Glossina morsitans*. Rogers has shewn that very fatal of eastern diseases, Kala-Azar, to be carried by the bed bug, while in 1897 Simmond demonstrated the flea carriage of plague. Still later Ross and Milne found the *Spirochaeta obermeieri*, the cause of relapsing fever, to be also the cause of African tick fever, the tick being *Ornithodoros moubata* which transmits infection through its offspring. This work was subsequently confirmed by Dutton and Todd.

With the exception of yellow fever, which is supposed to have an ultra-microscopic cause, and plague, all the above conditions are haemo-parasitic.

Of their vehicles only some belong to the true diptera or blood suckers, but they are all blood sucking and parasitic. The fleas or pulicidae are closely related to the diptera. We have then the blood parasites transmitted by insect hosts: we have the plague bacillus carried by a flea. May we not consider, therefore, whether typhus is similarly carried?

IS THE ANATOMY AND LIFE HISTORY OF THE FLEA CONSISTENT WITH THE HYPOTHESIS OF FLEA CARRIAGE OF INFECTION.

There are many species of fleas. Indeed it is possible that there is a species of flea for each species of host. Thus man, rats, cats and dogs are the hosts respectively of *P. irritans*, *P. cheopis*, *P. felis* and *P. canis*. Further, the species of flea parasite varies geographically. In India, for example, *Mus decumanus*, the common rat, is the host of *P. cheopis*; in Western Europe *Ceratophyllus fasciatus* is the common parasite; in Russia, *Typhopsylla musculi* is the usual flea parasite found on *Mus decumanus*, according to Dr Verjbitski. Yet again the species of parasite on any given host may

vary with the temperature, for during the cold weather in the Punjab *Ceratophyllus fasciatus* partially displaces *P. cheopis* as the parasite of *Mus rattus*. Each species of flea has its true host, though under pressure of want it may become a casual parasite of another host. *P. felis* has a wide range of hosts. *P. irritans*, which quickly dies in the absence of human blood, has few alternative hosts.

*P. irritans* is parasitic on man. Its distribution is world wide. It is a blood sucker closely allied to the order diptera in its larval stage and in that it undergoes a complete metamorphosis. The pupa on the other hand has the characters of the hymenoptera. It is only when the adult stage is reached that it becomes parasitic. It lives in the house of its host and usually attaches itself to his person for feeding purposes. The eggs are laid on rugs and carpets and hatch in three or four days. The larval and pupal stages each last about a fortnight and the full grown imago may live, when under the most favourable conditions, six weeks. In the case of eggs laid late in the autumn, the larval stage may last throughout the winter.

The flea is equipped with ample mouth organs. They consist essentially of a pricking epipharynx

and of the hypopharynx with its salivary pumping apparatus. "In general it may be stated that the "epipharynx makes a way through the skin for the "mandibles, that the mandibles enlarge and lacerate "the hole thus bored and convey into it the salivary "secretion which is pumped by means of the salivary "pump down the salivary canal contained in them, and "the expiratory pharynx aspirates blood from the now "congested wound along the blood channel formed by "the approximation of the epipharynx and the two "mandibles." Ingested blood passes via the pharynx, oesophagus, stomach and intestines to the gland lined rectum, whence the excreta are expelled. In the case of plague, the bacilli are confined to the intestinal canal of the flea who suffers apparently no ill effects from their presence. To begin with, the bacilli increase in number, but they are gradually expelled in the faeces no less virulent than on ingestion. Fleas acquire the bacilli from animals in the septicaemic stage of the disease, viz. the last 24 to 36 hours. Death may occur from plague, however, without septicaemia supervening. "During the "plague season fleas might remain infective for 15 "days after imbibing infected blood." They probably convey infection by transferring bacilli from their

mouth organs to the puncture wound in the new host, though the exact method of transference is unknown. Again, they may be crushed by the host and their intestinal contents be rubbed into the wound. The slightest abrasion, in the recent state, is sufficient for the entrance of the organism. Lastly, the faeces are a source of infection when applied to a wound, and, in the case of plague, as the bacilli in such excreta can survive in linen for five months, the handling of clothes must be done with care. Experimentally it has been shewn that the bite of a single flea is not sufficient to produce plague.

The flea, then, is a carrier of disease. If the flea carries typhus, the probability is the infection is similarly of a bacterial nature. Its clinical history supports this idea. The course of plague and yellow fever differ from other fly carried diseases in being short and sharp, profound intoxications. Relapses and second attacks are almost unknown. How different is it in malaria and the other haematozoal diseases where the course is long and marked by relapses or steady progression to a fatal issue.

IS THE THEORY OF FLEA CARRIAGE CONSISTENT WITH AND  
EXPLANATORY OF THE EPIDEMIOLOGICAL FACTS OF TYPHUS?

The literature of typhus is rich in its epidemiological observations. Certain points are constantly affirmed, viz. its association with dirt, overcrowding and lack of ventilation; with war, famine, and misery from unemployment; and the great infectivity of clothes - every one of which is favourable to the theory of flea carriage. The presence of the flea does not necessarily imply dirt, but on the other hand, where there is dirt the flea flourishes unmolested. Dirt implies the presence of the flea. That dirt and therefore the flea is a sine qua non in the aetiology of typhus, a quotation from the old epidemiologists will abundantly prove. "Ireland," said Popham in 1835, "is its last stronghold. It smoulders on till some spark kindles it into a flame." The epidemics in Great Britain during last century all came from Ireland, with the exception of that following the Crimean War. In 1846 it was so imported into Liverpool, where there is a large Irish immigration and population, and, according to the M.O.H., it was almost entirely confined to the Irish quarter. "When typhus occurs in

Scotch or English towns it is always in consequence of importation of the disease from Ireland," says Graves. So true was this that a typhus outbreak was always expected at the end of the haymaking season during which many Irish labourers were employed.

The same principle holds for America. It was carried there continuously by emigrants but never secured an epidemic foothold outside the seaports. During the 1847 Irish epidemic no less than 3000 such emigrants sick with typhus were received into the hospital at Staten Island.

Russia, where there have been great epidemics, notably following the Crimean and Russo-Turkish wars, and Italy, are the remaining European typhus foci. In all three there is one common factor, low social conditions. It is when these conditions become aggravated by failure of crops, unemployment and war, producing poverty and want, that typhus bursts its endemic limits. The epidemics following the Seven Years War, the Thirty Years War, the Napoleonic Wars, and those during the Sieges of Saragossa, Torgau and Dantzic, illustrate these points. The epidemic of 1833 in Silesia was reported to have arisen de novo, "the cause being the customs of the inhabitants, the

"great want and penury in which they live, particularly the want of wholesome food."

"It is always and everywhere the wretched conditions of living which spring from poverty and are fostered by ignorance, laziness and helplessness, in which typhus takes root and finds nourishment. The prototype of these conditions is found in Ireland, which is the greatest sufferer from the disease, unconquerable in and inseparable from the Irish proletariat, faithfully following the Irishman wherever he transplants himself and his misery."

Poverty, however, is not the essential condition, for McLagan has pointed out that trade and manufactures were flourishing in Dundee at the time of the epidemic of 1865-6, so much so that people flocked to the town. "This proves," says McLagan, "that it was not want and misery but another factor, namely, the overcrowding in the workmen's dwellings that gave rise to the disease." Similarly Graves, while admitting that want of provisions and wholesome food are aggravating causes of typhus, says, "They are not the sole or the chief causes," and indicates the epidemic of 1826 as a remarkable example of an epidemic occurring in a year of plenty.

Graetzer says of the Breslau epidemic, 1868-9,

that "want and privation could in no wise be accused  
"of being factors in the aetiology."

Also, it was pointed out by Frank as regards  
the epidemic in the circle of Pless in 1856 that  
neighbouring places in a similar condition of want  
and misery escaped the disease.

"Famine and typhus, then, have no necessary con-  
"nection as cause and effect," concludes Virchow.  
They are only predisposing causes. "Crowded, filthy,  
"ill ventilated apartments and neglect of personal  
"cleanliness more immediately lead to the formation  
"of the fever," says Hirsch. These are factors on  
which everyone is agreed.

Conversely some interesting facts may be adduc-  
ed as to the effect of cleanliness and adequate ven-  
tilation.

Cheyne, Dublin epidemics 1801, says the disease  
was very rare in the higher ranks of society and  
"there were very few instances of the disease extend-  
"ing to a second person in any house in which proper  
"attention was paid to cleanliness and ventilation."  
Murchison's evidence on this point is invaluable.  
"In London, typhus is almost unknown among the better  
"classes save in a few isolated cases where there has  
"been direct intercourse with the sick."

"During the seventeen years I have been connected with the London Fever Hospital I have attended only six private cases of typhus, of whom one was a medical man, one a clergyman, and one a lady who visited the poor."

Haygarth in 1777 could not remember a second case in a clean airy house among the upper classes.

Christison attended 280 cases among students and calculated that those had 1200 attendants, but among this number there was not a single secondary case. In the 1897 outbreak in Edinburgh there were no cases among the hospital staff. Hay quotes the similar experience of Liverpool (Dr Russell) and fortunately we had no cases among the staff at Leeds.

Ventilation and cleanliness, we may presume, were perfect in these cases.

9 // But the better classes do not escape. Judge, lawyers and jury have failed suddenly and simultaneously after being pent up in a close stifling atmosphere with infected prisoners. This was the feature common to the Black Assizes of Cambridge, 1522: Oxford, 1577: Exeter, 1586: Taunton, 1730: Launceston, 1742: and the Old Bailey, 1750. This led to the belief that the danger lay in the bad atmosphere, especially as at Oxford and Exeter only those

who had been in court were affected. This was a view strongly held by Murchison, who quotes numerous instances where persons, chiefly doctors, failed with typhus even a few hours after being in a typhus atmosphere or inhaling the breath of a patient. But outbreaks can be quoted where this element was wanting. Pringle, in his account of the disease of the Dettingen campaign, gives a very apt illustration of this. After the battle dysentery and typhus broke out, and subsequently the convalescents were sent down the Rhine in bilanders. With these vessels went a bundle of tents to a tradesman of Ghent for repairs. Twenty men were put to the task, but of these every one contracted the disease and seventeen died. The tents were blamed for the infection.

Clothes have always been regarded as the most infective of fomites. So much so that Murchison says of the 1833 London epidemic that "so notorious was the risk to laundry women," and they "are so invariably and frequently attacked that few women will undertake the duty." Similarly Perry of Glasgow, in supporting the theory that the convalescent period is the most infectious, points to the occurrence of secondary cases among the patients in the convalescent wards. Murchison, criticising this

statement, very properly points out that such patients had then resumed their clothes in which the infection had lain dormant.

### Conclusions.

Thus, in short, we find that contact with a patient, his habitation or his clothes, is extremely dangerous, whilst contact with a patient in a clean private house or modern hospital is perfectly safe, that want, privation, overcrowding, war and famine, are factors in the aetiology of typhus only in so far as they tend to produce a state of filth and uncleanliness. If the cause of typhus were to be found in the emanations from the bed and clothes of the patient, the number of cases among his attendants would be larger. That the danger is removed with his filth, and that the risk is greatest to those engaged in removing it, points clearly to the source of infection.

Typhus is a house disease. Infection rekindles repeatedly in the same premises. The spread of infection is limited and its striking distance is short. Clothes are highly infectious. The flea theory is in full accord with these facts: indeed it explains them. In view of the fact that a flea has already

been proven the carrier of infection in a similar and once closely associated disease, it is submitted that prophylactic measures against it during typhus outbreaks are essential.

SUMMARY OF PART IV.

1. Why the cause of typhus has not been found:
    - (a) Decline of the fever in Listerian times.
    - (b) Confusion of diseases and terms.
  
  2. Theories on the cause and nature of typhus:
    - (a) Sydenham.
    - (b) Christison.
    - (c) Murchison.
  
  3. Hay's hypothesis - Flea carriage of infection.
  
  4. Argument by analogy in support of Hay's theory:
 

Haemoparasitic diseases, yellow fever,  
plague, etc.
  
  5. Is the anatomy and life history of the flea consistent with Hay's theory?
  
  6. Is the theory consistent with and explanatory of the epidemiological facts of typhus?
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DISEASE.

*Syphilis*

Notes of Case

*Schmidt*

Name

Age

20

Diet

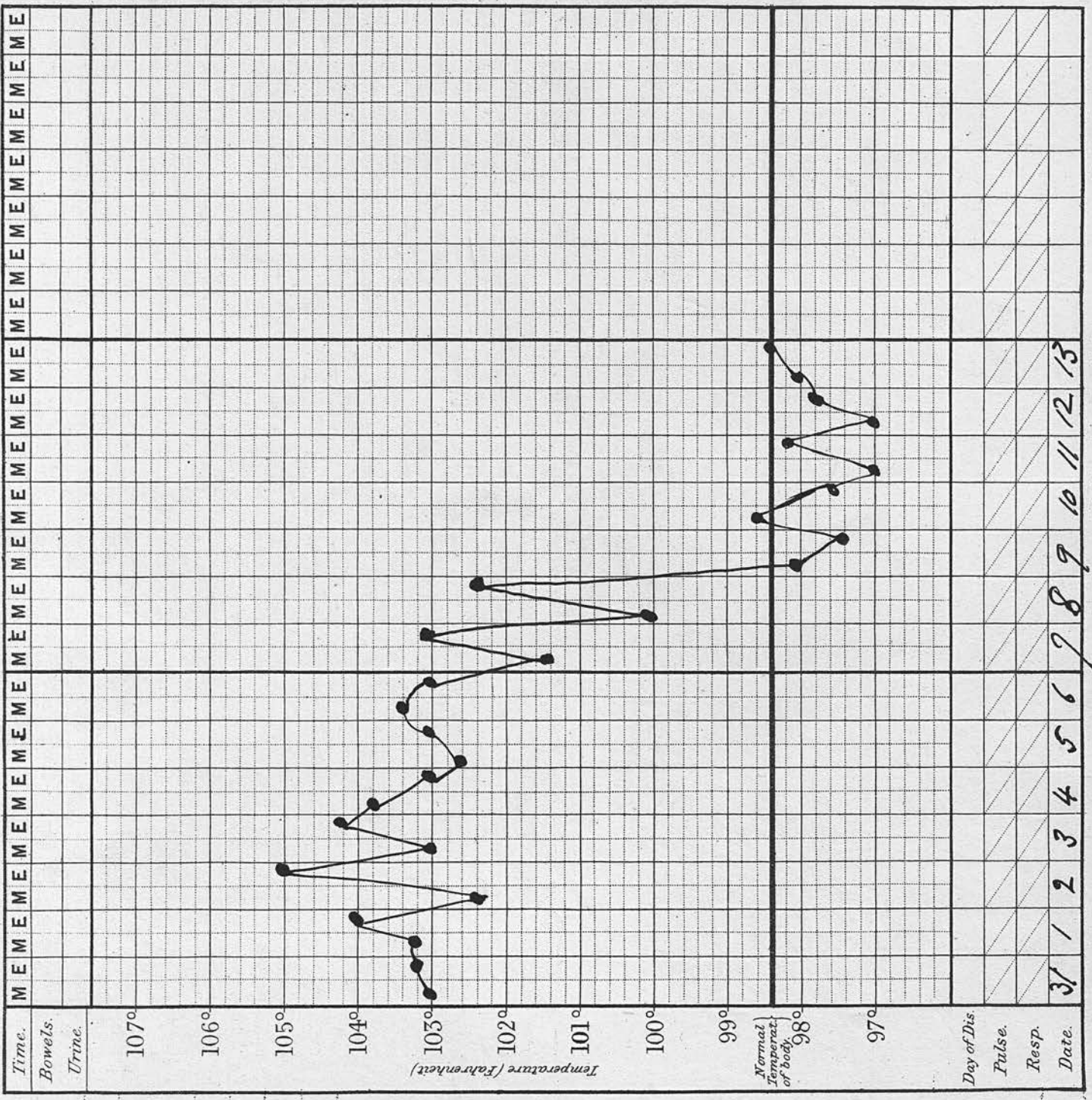
Case Book No.

Date of admission.

*July 31, 1908*

*Richard Schmidt*

*Sept. 12 '08*



DISEASE.  
Typhus.

Notes of Case.  
Herbert Mitchell

Name

Age 17.

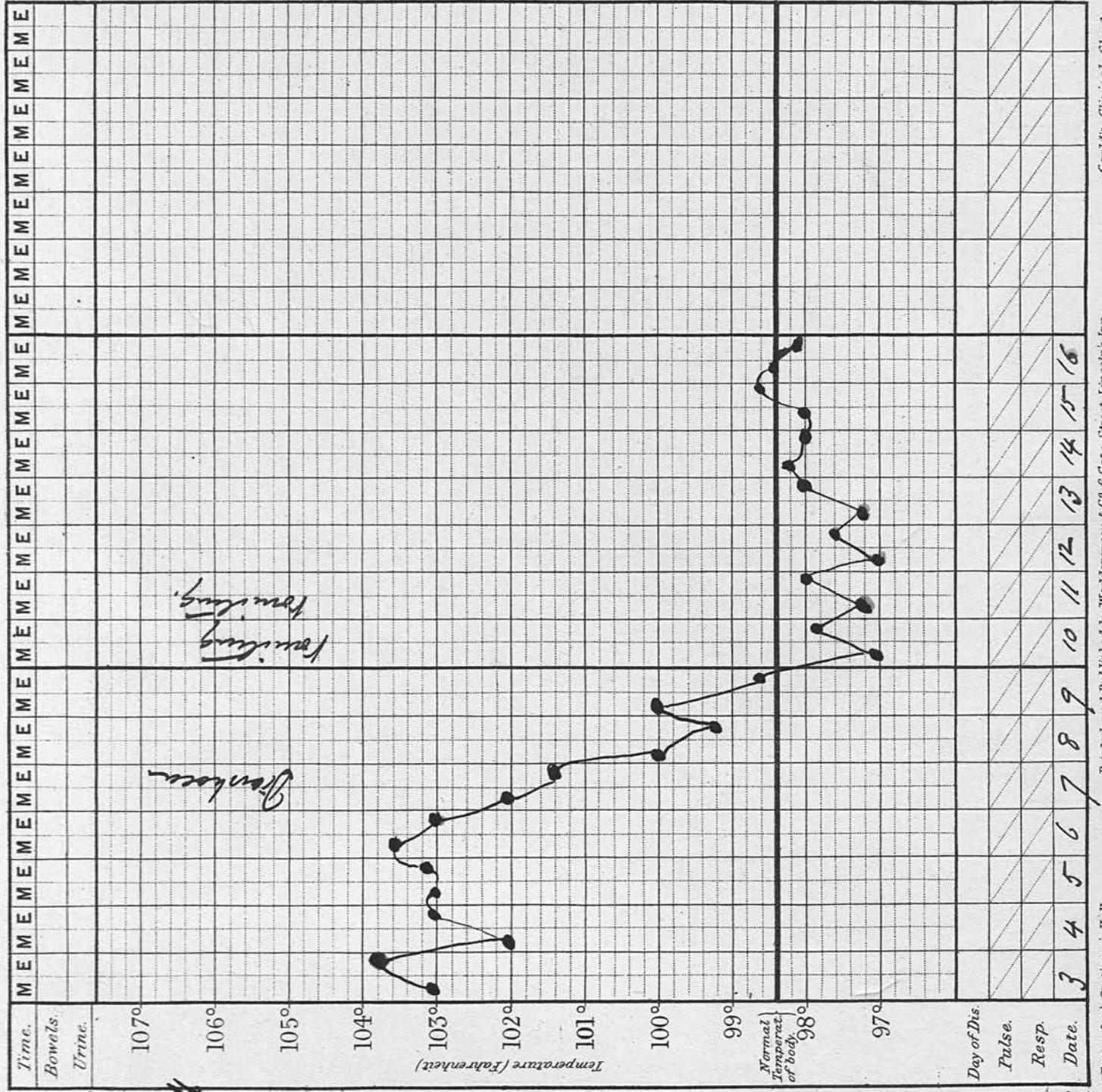
Diet

Case Book No.

Date of admission.

Aug 3. 08

Resident  
Elizabeth, Sept. 16. 08



DISEASE.

*Typhus.*

Notes of Case.

Name *George Teale.*  
*Police constable*

Age *29*

Diet

Case Book No.

Date of admission.

*Aug 13. 09*  
*Revised*  
*Discharge. Sept. 19. 09*

