NOTES

on

AN OUTBREAK OF BUBONIC PLAGUE

IN GLASGOW IN AUGUST 1900.

with

A STUDY OF ENTERIC FEVER IN ITS INITIAL STAGE

and

OTHER CASES THAT SIMULATED PLAGUE

by

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The Outbreak.

On the afternoon of Friday, August 24th 1900, I was casually called in to 57 Thistle Street, Glasgow, E. to see a young man who was said to be very ill. I found my patient in bed in a very restless condition as he was tossing himself about from one part of the bed to the other. His name was Patrick Mallon aet. 20, Slipper-maker. I was told that he had been in good health till Wednesday (August 22nd) when he had a rigor with nausea and vomiting. He had also diarrhoea with abdominal pain. The following day the diarrhoea continued and he became very restless. During that night he was delirious and could not be kept in bed. He was allowed up and staggered aimlessly about the room from place to place muttering incoherently like a man in a drunken stupor. The delirium and restlessness had continued during the next day till my visit. There was nothing of medical importance in his past history.

Physical Examination. He was a fairly well - nourished but slightly built young man of the working class type. He was evidently in a semi-comatose condition and very prostrate. He was dull and stupid and I could elicit no answer to any question I put to him regarding his illness, nor did he make any response to a repeated request to put out his tongue. His temperature was 103 F. His pulse was 130, small and feeble. As my first impression was that it was a case of acute lobar pneumonia the

result of a drinking bout - at this stage of the Examination I did not know of the other two patients - I made a careful examination of the chest but it yielded nothing definite except congestion at both bases which was the natural sequence of cardiac failure as the first sound of the heart was weak and indistinct. The diarrhoea next led me to think of Enteric fever so I made careful enquiries about his stools. My informant said that he had had diarrhoea at the inset of his illness and that it became worse after the doctor had prescribed castor-oil. She also volunteered the information, without any suggestion of mine, that the stools were like "pea-brae" and had an "awful smell". This was my first intimation that another medical man had seen the patient. I was also informed that the mother of the patient was ill in the "kitchen" bed and his sister Christina Mallon aet. 10. had sickened on the previous Sunday and died on the following Tuesday. Dr. Charles E. Robertson who had seen this girl for the first time ten minutes before her death had certified the cause of death as "acute pneumonia". Dr. Robertson had also seen the mother and the patient I was examining, on the Wednesday, Thursday, and the forenoon of Friday the day of my visit. On continuing the examination of the patient I could find no rose-spots on his abdomen nor a rash of any kind. There was no gurgling on pressure in the right iliac fossa. As for caecal tenderness it was no use trying to elicit it as the patient was semi-comatose. No enlargement of the spleen could be made out on palpation or percussion. His tongue was tremulous and had all the appearance of a tongue in a typhoid state. There was no vomiting

after the first day. He had passed little urine during his illness.

Leaving the patient, I went into the kitchen to examine his mother, Mrs. Mallon, aet. 40, housewife, widow. She was in bed and quite conscious. She said that she had been in good health till Monday (August 20th) when she had a rigor, severe headache, nausea and vomiting. The headache and vomiting had continued more or less till my visit (Friday) and her one appeal to me was to relieve the persistent vomiting. She had slept little since her illness but had not been delirious. She had been able to go about till Wednesday and since then had been confined to bed. She had had no diarrhoea and her bowels had been well moved with castor-oil. She was the mother of seven children and had always had good health.

Physical Examination. She was a well nourished well developed woman. Her temperature was 102 F. Her tongue was flabby and coated with a yellowish white fur. She had persistent vomiting as already noted. There was no diarrhoea. Her pulse was 100 and of good strength. The heart sounds and lungs were normal. There were no rose-spots on abdomen or rash of any kind. No enlargement of the spleen could be made out on palpation or percussion. On palpating in the caecal region there was no gurgling but she said that I caused her pain. It is important to note for the sequel that the tenderness and pain may have extended along the right groin but I had no reason to extend my examination beyond the caecal region and did not do so. Had I done so I would have found a very palpable, painful, swelling in the

right femoral region extending to the inner third of the right groin, and the pain I elicited on pressure in the caecal region, was no doubt due to indirect pressure on this swelling. I was only told of this bubo two days afterwards by Dr. Robertson who had his attention drawn to it by the patient and had ordered it to be poulticed. She passed urine freely.

Having completed my examination of the Mother I noticed a little boy lying beside her in bed. On inquiring about him I was told that he was William John Malloy aet. 3, and he was now well but he had been ill for the past three days with headache, pains in his abdomen, and his skin had been "roasting". There was no vomiting, nor diarrhoea nor delirium.

Physical Examination. His temperature and all his organs were normal. I could find nothing the matter with him but as his eldest brother was positive his skin had been "roasting" for three days and he appeared to be ill, I concluded that he had the same disease as the other two patients.

Diagnosis. I rapidly summed up on the spot the symptoms and physical signs of the three patients and also took into account the death of the fourth patient after an illness of two days. In the first place I excluded ptomdine poisoning from unsound meat, or fish, or fruit, or tinned goods, inasmuch as there was a distinct absence of a sudden and a common onset. The onset of the disease was gradual and in the following order: Christina

sickened on Sunday and was dead on Tuesday, her mother had a "shivering fit" on Monday, and her two brothers became ill on Wednesday. When we find four apparently healthy individuals of the same household struck down within a day or so of each other with an illness characterised by a temperature of 102 F. or 103 F. I think that it is a safe deduction that the illness must be some acute infectious disease. Hence I had no hesitation in diagnosing the Malloy cases as cases of an acute infectious disease. Of the particular infectious disease it was, I had my doubts. I excluded Typhus Fever for I had had an opportunity of studying a case of Typhus in a neighbouring street two years ago, and while Patrick Malloy resembled it in the sudden onset, early delirium, extreme prostration, dull and stupid appearance, he had not its dusky, measly rash, and the symptoms of the other two patients negatived Typhus. I also excluded Smallpox, although at the time there were a few cases in the city, and I had had a case of Smallpox in the adjoining street two months previous. Here again there was a well marked popular rash to guide me in my diagnosis. The only acute infectious disease I thought it might be was Enteric Fever in its initial stage and that the classical symptoms of that disease might appear later. To my mind it resembled Enteric in (a) temperature, (2) abdominal symptoms, especially the pea-soup stools with offensive odour according to my informant, and (3) the caecal tenderness in the case of Mrs. Malloy. Against Enteric there were (1) the sudden onset,

(2) early and marked delirium and great prostration of Patrick, and (3) the rapid recovery of Wm. John.

Hence my diagnosis of the Mallon cases, on my first and only examination, in a dark room and kitchen with filthy surroundings, was that they were cases of an acute infectious disease and I determined to notify them at once as cases of Enteric Fever with a note of interrogation to imply that the diagnosis was indefinite and that they were cases demanding complete isolation and close observation for developments.

Before I left that infected house I warned the inmates that the patients were suffering from a dangerous and highly infectious disease. I forbade the entrance of all neighbours and friends. I gave them instructions about the utensils the patients were using, and isolated the latter as best I could until they would be removed to Belvidere Hospital. On leaving the house I sought Dr. Robertson who had been in attendance three days, and found him two hours later in his consulting room. He could throw no further light on the diagnosis, so he readily assented to my proposal that it was better for the patients and better for the community at large to notify them at once. Half an hour later, or two-and-a-half hours after I had examined them, I posted the three following notifications to Dr. A. K. Chalmers, the Medical Officer of Health for the City.

Mrs. Mallon, aet. 40.
57 Thistle Street, E. E.,
Enteric Fever?

Patrick Mallon, aet. 20
Enteric Fever?
Wm. John Mallon, aet. 3
Enteric Fever?

I was afterwards informed by Charles Mallon that his mother and two brothers were removed the following day (Saturday) to Belvidere Hospital for infectious diseases. On the morning of the day following (Sunday) Dr. Knight of the Sanitary Staff called and informed me that on careful examination of the Mallon cases by the Medical Staff of Belvidere, besides the femoral bubo in Mrs. Mallon already noted, a bubo had been found in the right axilla of Patrick Mallon and that Dr. John Brownlee, the Medical Superintendent of Belvidere hospital had made a provisional diagnosis of Bubonic Plague. My co-operation was solicited in detecting similar cases.

Another Case of Plague.

On the morning of September 10th, 1900, I was casually called in to see a young woman in 23 Florence Street, E.E. She was lying in bed and quite conscious. She said that her name was Rosina Murphy, aet. 25, unmarried, hair-worker. Excepting an attack of Typhoid Fever seven years previous she had always had good health till August 23rd or 18 days prior to my visit. She then had a "shivering fit", severe headache, nausea and vomiting with abdominal pain and diarrhoea. These symptoms only lasted two days

and they were also accompanied, according to the statement of her mother, with delirium which passed away on the fifth day of her illness. On the third day of her illness she felt pain in both groins and on examination she found a "lump" in each groin which was very tender and painful to the touch. She was too poor to have regular medical attendance but she was seen on three separate occasions by Dr. Maclean Smyth. She said that she had no association with any of the Plague Cases in the city, but admitted that two of the young women who sat beside her in the hair factory had been to the "wake" of a plague patient in Rose Street, S.S.

Physical Examination. She was a well developed young woman but very weak which was no doubt the result of her 18 days illness. Her temperature, heart, lungs and abdomen were normal. There was no rash or mottling of the skin. In each groin was a very palpable bubo about the size of a pigeon's egg, very painful to the touch. No other glandular enlargement could be made out and excepting the two buboes and great debility there was nothing else apparently the matter with her at the time of my visit.

Diagnosis. I excluded gonorrhoeal buboes, soft and hard chancres, by exposing her and after a careful examination finding no evidence of any of these diseases. A careful inquiry for a specific history also gave a negative result. From the history of the case and my experience of the Mallov cases, I had no hesitation in diagnosing bubonic plague. Before

notifying Dr. Chalmers, I called on Dr. Maclean Smyth. He said that when he saw her three days after the onset of the illness her temperature was 104 F. and when seen ten days later it was normal. He thought that the buboes were gonorrhoeal. I shall discuss later the differential diagnosis of gonorrhoeal and plague buboes. The patient was at once removed to Belvidere, where the diagnosis of bubonic plague was confirmed.

The Origin of the Outbreak.

When the provisional diagnosis, in the Malloy cases, of bubonic plague was confirmed by finding typical forms of the bacillus pestis in blood films taken from the buboes, by cultures on glycerine agar giving the typical appearance macroscopically and microscopically of the bacillus pestis, by inoculation experiments on animals, by post mortem examination of the body of Patrick Malloy, and, later, by the opinions of plague experts from different parts of the world, the first question that arose was: from whence did the plague come? Plague is a specific disease. It has, therefore, a specific cause, the bacillus pestis which was discovered by Kitasato and Yersin during the epidemic in Hong Kong in 1894. When I come to describe the district mapped out as the infected area, it will be very evident that it offered a suitable nidus for the growth and multiplication of the plague bacillus. But no matter how suitable the nidus,

or how insanitary the conditions, we know from biology as well as bacteriology that no micro-organism can arise de novo. Hence the plague bacillus must have found its way into the city, on living or dead matter, from some plague infected parts as the Malloy's were inhabitants of Glasgow, and there was no known case of bubonic plague in Great Britain at that time. Excepting two cases in 1899 and four cases in 1900, which were detected at the London docks, plague had not been known to exist in the British Isles since the Great Plague of 1665 which is so graphically described by Defoe and so quaintly mentioned in Pepy's Diary. Radcliffe in a Local Government Report in 1875 has traced the history of the Plague in the Levant from the Great Plague of 1665. While the disease died out in the seventeenth century in Western Europe it continued in parts of Central, South, and East Europe, and became very active in the eighteenth century. The area of prevalence decreased as the century advanced but again in 1812 it became widely diffused in the Levant and in 1834 it was again epidemic in European and Asiatic Turkey and Egypt. It prevailed more or less in these countries till 1894 when it became pandemic and appeared in May of that year in Hong Kong from whence it spread to other parts of China, a few cases being imported into Japan. Plague is endemic in India on the Thibetan frontier, and it appeared epidemically in 1896, beginning at Bombay it rapidly spread to the

Bengal, Madras and other presidencies. During 1899 and up till the Glasgow Outbreak in August 1900, plague had appeared at Mauritius on the Indian Ocean, at Alexandria in Egypt, at Oporto in Portugal, at San Francisco in North America, and at Sydney, Adelaide, Melbourne, and Brisbane in Australia. Hence the pandemic which began at Hong Kong in 1894 and spread to the other countries mentioned, was the first appearance of plague south of the equator and the western hemisphere in modern times. To the freer communication, in recent years, between countries the spread of the disease can be traced, plague extending along the trade routes. Just as was the case in Oporto, in Alexandria, in Mauritius, in Madagascar, in Bombay, and in almost every place to which plague has spread in recent years, no direct importation of infection from a pre-existing focus of the disease could be traced in the Glasgow Outbreak.

Thistle Street where the Malloy's lived was on the south-side of the river Clyde, and nearly three quarters of a mile from Kinaston, which was the nearest dock to it. None of the Malloys were in any way connected with the docks or with the shipping trade. On close inquiry it was found that Mrs Malloy ten days previous to her illness was present at a "wake" held over the body of a Mrs. Bogan, who died at 71 Rose Street, South-side, from what was certified as "acute gastro-enteritis". The day previous to this death a child had died in the same house of what was certified as "Zymotic enteritis", and 15 days

later the father of the house was admitted into Belvidere suffering from what was notified as "Enteric fever", but which on examination in Belvidere was found to be bubonic plague. There is no doubt that the other two deaths were due to plague. How the Eogans contracted the disease will remain a mystery, for the closest investigation did not reveal any known source of infection. The Malloys were infected by the Eogans through Mrs Malloy attending the Eogan "wake". Of the 36 cases, 30 were traced as having had association either direct or indirect with the Eogans or the Malloys; 4 appeared in Dale Street, South-side, 200 yards west of the infected area, and they had no known association with the other cases; it appeared in Robert Street, Govan, and after being in the Glasgow Western Infirmary was sent to Belvidere, and he had no known association with the other cases, and I died in Govan Road, Govan, from what was undoubtedly plague, and he likewise had no known association with the other cases. Hence there were four distinct foci of the disease.

Mode of transmission of plague. It was not probable that plague came into the city overland. It probably was brought into the city by a ship from India, China, North or South America, or Australia, where plague was prevalent at the time of the Glasgow Outbreak. The disease could have been transmitted to the healthy from persons suffering from plague; by animals, especially rats, infected with plague: by insects and body parasites;

and by articles of clothing and other goods which have been contaminated by the plague bacillus. Plague cannot be said to be very infectious as doctors, nurses, and even native attendants in plague hospitals, as well as workmen employed in disinfecting plague houses, rarely fall a victim to the disease. In the Glasgow Outbreak only one ward-maid, and also a ward cleaner in the plague ward had a mild attack of the disease, but it may be stated that all the nurses and those in immediate contact with plague patients were inoculated with Yersin's antiplague serum. Charles Malloy and Mrs Murphy, who were not inoculated, were brought into the most intimate contact with their plague relations. Malloy not only nursed his plague mother and plague brothers but also slept with the latter for almost a week prior to their removal to Hospital, yet he escaped the disease, while Mrs Murphy nursed, ate, and slept with her plague daughter for 18 days, and developed no signs of the disease. As a rule the danger of infection by the air is not great for it is only by living for some time in an atmosphere impregnated with plague bacilli from the excreta and sputa of plague patients that one would fall a victim to the disease. That the plague bacillus may gain entrance to the system by the alimentary tract is borne out by the fact that they are often found in the faeces and the intestinal follicles and glands of the mesentery are swollen before the appearance of the femoral,

axillary or cervical buboes. Animals also fed on organs taken from those who have died of plague show changes post mortem chiefly confined to the alimentary tract. It may be here said that the faeces and urine of several of the Glasgow plague patients were examined for plague bacilli but with a negative result. That the infection may find its way into the system by food and water there is little doubt although the evidence at present is not conclusive. Infection by the unbroken skin is not believed to be probable but only through wounds in the skin made by the bites or stings of insects. The conclusion of the Indian Plague Research Committee on this point was, "That no definite skin lesion specifically indicative of infection by this channel was demonstrable, but in certain cases the bacillus was present in the skin lesion and as in each instance the bubo was situated in the glands corresponding to the lesion, this place was believed to be the point of entrance of the bacillus." But as Cantlie points out that were the plague bacilli in the blood their distribution is so general that they might be met with in any skin scab, scar, or pabule, and as plague bacilli have been found in skin lesions where they could not be demonstrated in the blood, the conclusion is that they may enter through a skin or abrasion. In none of the Glasgow cases was any solution of continuity found on the surface of the leg or foot which might have admitted the infection. Animals are common carriers of plague, especially the rat.

The rat has played an important part in most plague epidemics in spreading the disease and Simond has demonstrated that its chief danger is from the vermin that infest it. In the Glasgow Outbreak the rats which were plentiful in the district at the commencement of the Outbreak decamped as soon as the disinfection of the houses and drains was begun. Of those caught and examined by the City bacteriologist no evidence of plague was found, nor was there any evidence of infection found in rats obtained from vessels at the harbour which had arrived from India, China and elsewhere. Insects, especially fleas, bugs and other body parasites, as well as flies and ants, may transmit plague. The reason why the glands in the groin are so often affected is said to be due to the tendency of plague infected vermin to settle into the lower extremities and by their bites the contagion to find its entrance through the lymphatics and glands of the lower limbs. That clothing and bedding can carry the plague contagion there is no doubt. Three of the Glasgow cases were traced to infected bedding. How clothing infects is yet to be demonstrated for the mere wearing of clothing of those dead of plague is not in itself sufficient as was proved long ago by a French physician in Egypt. The infectivity of clothing has been attributed to the vermin that infest them, but Wilm has shown that sterilised pieces of clothing soaked in a culture of the plague bacillus and afterwards protected from extraneous infection gave cultures of the bacillus after four weeks, thus showing that

clothing can infect independently of insects. It is, however, possible that the body parasites become infected from the infected clothing and then in turn by biting the skin infect the wearer or anyone with whom they come in contact.

Clemow has shown that a person may be exposed to the infection of plague and may carry away some of the infectious materies morbi clinging to his clothes or to some article taken by him from the infected area or district and days, weeks or even months may elapse before the infectious material gains access to his tissues. He cites, among other examples, two cases of plague that occurred on the Thames in 1896. In the one the patient had left Bombay 39 days before the onset of the disease and in the other the patient had left Calcutta 45 days before he developed the disease. That plague can be transmitted from a plague patient to a healthy person through a third person who escapes the disease was clearly demonstrated in my case of Rosina Murphy. She was positive that she had no direct association with the other cases, but at her work she sat beside two girls who had been to the Regan "wake", and by them she was undoubtedly ~~in~~fected although they themselves remained free from the disease. The vehicle of infection may have been in this case fleas which did not bite their hosts, or if they did bite them, their hosts were immune to the disease. A similar case was that of the wife of one of the men employed in removing clothing from the infected houses for disinfection. The clothing and bedding were wrapped in sheets

wetted with formalin and conveyed to the steam oven. The men employed in this work had ~~immunising~~ doses of Yersin's serum and none of them were affected but the wife of one of them developed plague and, as she had no association with other plague patients, and lived fully a mile from the infected area, the probable explanation is that her husband carried the infection to her in his hair, as he wore overalls at his work. Hence the only new fact that the Glasgow Outbreak added to what was already known of the communicability of plague was the transmission of the disease from a plague patient to a healthy person through a third person who developed no evident symptoms or signs of the disease.

The Infected Area.

For the purpose of house to house visitation for cases of illness and for special cleansing and prompt removal of refuse, a considerable area surrounding Thistle Street and Rose Street was delimited as an infected area. This district was bounded by Cumberland Street on the South, South Wellington Street on the east, Adelphi Street and Carlton Place on the North, and Bridge Street and Eglinton Street on the West. The following map taken from the Glasgow Post Office Directory for 1900 gives a bird's eye view of the area.

Bird's eye view of the infected area from Cumberland Street looking North towards the Clyde.

I know the city well for I was born within a stone's throw of Glasgow Cross and. excebtina two years in England, have lived all my days in the city.

Thistle Street where the Outbreak took place was one of the most densely populated streets in the city. and No.57 where the Mallows lived was notorious as being one of the most unsanitary parts of the city. It consisted of a tenement of three flats, bounded on the right by a tenement of two flats with an outside stair entrance, and on the left by a carpenter's shop. In going from the street to the tenement a rectangular passage had to be traversed about 25 by 15 yards. In the right hand corner of this passage about 4 yards from the side of the infected house was an open ashpit but of still more importance from a sanitary standpoint was the fact that this passage was the common resting place of hand-barrows, used by the dwellers in the tenement for hawking fish and cheap fruit, and not uncommonly the passage was on Saturday night the receptacle of putrid herrings and putrid fruit, which would be unsaleable on Monday, as well as refuse of all kinds, including faeces. The following is a pen and ink sketch of 57 Thistle Street taken from the street.

Whistle Street in August, 1900.
1. Rectangular passage.
2. Stair entrance to Mallow's house.
3. Kitchen window of Mallow's house.

There were 20 separate houses in the tenement with 2 entrances from the rectangular passage. Each house had affixed to it a ticket stating its dimensions and the number of inmates it was supposed to shelter. These tickets were instituted by Sir W. J. Gairdner, when Medical Officer of Health of the city, and by limiting the number of inmates in each house were one of the means adopted to stamp out Typhus which was at one time the scourge of congested parts of the city. The ticket on Mallon's house stated the dimensions as 1612 cubic feet and it was to shelter 4 adults. The house consisted of a room 18 by 12 feet and a kitchen 15 by 12 feet. The Mallon house was the only house in the tenement not overcrowded for the Mallon family was composed of 4 adults and 2 children. In the district the average number of adults above what is stated on the door I have found to be 2, and in a single apartment with 2 adults marked on the door, I have found as many as 9 adults, while Dr C. E. Russell has stated that in hundreds of houses one may find from 8 up to 13 inmates eating, sleeping, washing and dressing within the four walls of one room.

The district delimited as the infected area covered about half a square mile or 320 acres. There was no official census of its population as part of it was in the Hutchesontown and part in the Gorbals registration districts. To bring out its density of population, its high incidence of infectious diseases, and its high rate of mortality, I took myself the census of the area in my daily rounds. I counted in the area 934

closes leading to 9168 separate houses. From personal knowledge I averaged the number of inmates in each house as 6, which gave a total of 55008 inhabitants. To this total I added 800, the usual number in two large model-lodging houses in the district. Hence the approximate population of the infected area was 55808. The population of Greater Glasgow according to the census just published (April 1901) was 760,329 and its acreage 11861 which gives an average density of 64 persons per acre. The average density of the infected area was 174 persons per acre or 110 persons per acre in excess of the average density of the city. To find out the number of cases of notifiable infectious diseases in the infected area, I took the average number of cases I had notified during four years ending May 1900, during which there were no epidemics. I multiplied this number by 17, which was the number of medical men in the district, as I found on comparison that mine was a fair average. The result was 1 case of notifiable infectious disease per 113 inhabitants in the district. For the same period the average number of notifiable infectious disease for the whole city was 1 case per 230 inhabitants. Hence the approximate number of notifiable infectious diseases such as Typhoid, Typhus, Smallpox, Diptheria, Scarlet Fever, Erysipelas, and Puerperal Fever in the infected area was more than double the city's average of the same diseases. On the same principle I calculated the average weekly mortality of the infected area and found that it was 32 per 1000 of the population per annum whereas the average weekly

mortality of the city for same period was 21 per 1000 of the population per annum. Hence these figures conclusively prove that the incidence of infectious disease and mortality of a district is in proportion to the density of its population, and when the resultant evils of overcrowding such as impure air, absence of sunshine, imperfect drainage, and filth are considered it becomes only too evident that the bacillus pestis found, in the infected area, a suitable soil for its growth and reproduction. Another striking proof that plague, like Typhus, is a filth disease, was the fact that while there were four distinct foci of the disease, and while the disease was carried by the associates of Mallon and Boaan to every part of the city, yet it was only in the centre of the infected area that it flourished, and only flourished there until it was discovered when it was easily arrested by the vigorous sanitary measures adopted as well as by the dwellers in the district, in fear of the peril to their lives, keeping themselves and their houses clean. Hence the practical lesson of the Glasgow Outbreak was that only the inhabitants of Great Britain, or of any other country, who live under insanitary and bad hygenic conditions, need dread a visitation of plague.

Symptomatology of Plague.

I shall first describe the symptoms of plague as recorded by various clinical observers and then compare them with my own and the other Glasgow cases as well as with a series of cases that simulated plague including a study of Enteric Fever in its initial stage.

Plague may be defined as a specific infectious disease which usually appears in epidemic form and has a tendency to recur once it has attacked a community. The main feature of the disease is a febrile state with, or without, inflammatory glandular enlargement and with, or without, pneumonic or toxaemic symptoms.

Plague has three main types, the Bubonic, the Pneumonic, and the Septicaemic. All forms of plague can be placed under these types and the terms fulminant, toxic, siderans, puerperal, intestinal, gastric, typhoid, convulsive, nervous, typhus, pestis ambulans, and pestis minor as applied to plague, refer either to the severity and rapidity of illness, or to the leading symptom of a particular case or group of cases.

Incubation. - The usual period between exposure to plague and onset of symptoms is from 3 - 5 days. The minimum incubative period so far

determined in authenticated cases is 24 hours, and the maximum 12 days. There are no definite indications of infection during the period of incubation.

Onset. The disease is usually ushered in with a rigor accompanied with high fever, severe headache, nausea, vomiting, extreme prostration and aching in the back and limbs. The patient may be found in a state of excitement or delirium or may be apathetic and unable to answer questions but to him. The features may be pain-drawn or expressionless as if the patient were intoxicated. The eyes are sunken, conjunctival vessels injected, the face pale with dusky congestion round the eyes extending to the forehead and cheeks. In children an attack may be ushered in with convulsions.

Temperature. The temperature of the body in most cases during the first day of the illness will be between 101 F. and 103 F. but may suddenly rise to 104 F. and 107 F. within a few hours of onset. In the more favourable cases of the bubonic type it may drop on the third or fourth day to near, or even below, the normal and again rises on the fifth or sixth day followed by a moderate temperature which gradually subsides during convalescence. The initial rise of temperature is no indication of the severity of the illness unless hyperpyrexia is maintained. When after the second rise of temperature in the bubonic form fever recurs it

usually indicates septicaemia or pyaemia and a fatal issue. In the septicaemic form the temperature rises to a considerable height at the onset, 104 F. or 105 F. and rises higher or is maintained at this height to the end. In the fulminant form, which is the most virulent form of plague, the temperature may scarcely if at all rise above the normal. The probable explanation is that the patient becomes so prostrate by shock on account of the effects of the disease that a re-action does not take place. In the pneumonic form the initial rise is usually high 103 or 104, and may remain so to the end or fall just before death.

Alimentary system. The dorsum of the tongue is coated with a cream white fur except at the tip and sides which are clean and red. At first moist, in a day or two it may become dry, brown, and parched like the tongue in the typhoid state. Nausea and vomiting are usually present at the onset and may persist for a day or two longer. The vomited matter is bilious and haematemesis is seldom observed. The rule is for the bowels to be constipated but diarrhoea may be present at onset or the constipation be followed by a diarrhoea of a bilious nature with blood occasionally in the stool. The liver and spleen are often tender on palpation and both are enlarged especially the latter. Independent of this enlargement of viscera the abdomen may be distended and tender.

Circulatory system. At onset there is increase of pulse rate, usually above 100. It is at the beginning full and tense and may become easily compressible and dicrotic and towards the end irregular. Dilatation of the right side of the heart, a shortened first sound, and a feeble second sound, are often present. A systolic murmur is sometimes heard and there may be pulsation of the carotids.

Respiratory System. The pneumonic form of plague will be described later when I come to differentiate it from a case of acute lobar pneumonia that simulated it. In other forms of plague, excepting an increase in the respiratory rate, which may be 30 to 40, or even 50 a minute, symptoms of the respiratory tract are rare.

Cutaneous System. In plague no characteristic skin rash is observed. In a few cases in every epidemic there may be a typhus-like rash. Before death petechiae may be seen over the abdomen or over enlarged glands. Boils and carbuncles are rare. In some epidemics there may be subcutaneous haemorrhages, usually spreading from an inflamed gland, and the effused blood may be absorbed or there may be a slough with severe haemorrhage.

Urinary System. At onset the excretion of urine is often diminished, the specific gravity is high, and the re-action is intensely acid. There may be suppression of urine with lumbar pains at onset but this is more apt to occur towards the end in fatal cases. Albumen in the urine was rarely found

in the first Hong Kong epidemic of 1894, but during its second recrudescence and in all the Indian epidemics a trace of albumen was rarely absent from the urine. Granular casts with blood corpuscles are sometimes detected, while urea, uric acid and chlorides are deficient.

Nervous System. Headache, vertigo, muscular weakness and prostration are early symptoms. Tremor, especially of the muscles of the upper extremities, early develop as well as the loss of co-ordination which is seen in the staggering gait when the patient attempts to walk. Delirium is one of the most prominent features of the disease and commonly developes in the second day of onset being worse at night. In some cases there is furious delirium with suicidal intentions, the patient struggling when restrained but when left alone his movements seem to be without a definite purpose. Terrifying dreams, picking at the bed-clothes, muscular twitchings, and clonic convulsions have been noted in different cases of plague. When the patient sinks into a comatose state, it usually indicates death. Among more remote nervous affections are deafness, hiccough, thickness of speech, due to loss of co-ordination of lingual muscles, impaired sense of touch and hyperaesthesia.

Lymphatic System. The characteristic feature of the disease is the bubo except the pneumonic and septicaemic forms of plague in which the enlarged glands are only found post mortem. As a rule three-fourths of plague patients develop buboes. The

percentage of buboes in epidemic varies between 60 and 90. The appearance of the bubo may be preceded by tenderness or pain over its seat, or the bubo may be present from the very onset of the illness. The time of its appearance is, as a rule, during the first 36 hours, but it may not appear till the third day or later. In 50 per cent of those suffering from buboes, the glands affected are in the groin (femoral), 30 per cent in the axilla, 15 per cent in the neck, and the submaxillary and parotid glands are occasionally, and the supratrochlear, iliac and popliteal glands more rarely affected. Usually it is a single group of glands that are inflamed and simultaneous enlargement of glands in different parts of the body is not common. The size of the gland varies from a hen's egg to a small bean, and its proper dimensions are obscured by perivascular effusion or sero-sanguinolent extravasation. As a rule one gland of a group is markedly affected, the others only to a small extent. In the process of adenitis the tissues and glands are matted together with fluid exudation which becomes sero-sanguinolent. the skin reddens over the gland, then sloughs and a large cavity is left which discharges pus. In favourable cases the glands do not suppurate but are resolved, and may be left as hard nodules for a long time after the symptoms of the disease have disappeared. When suppuration does take place it is seldom before the seventh or eighth day, and as the most fatal period of the disease

is the fifth day in severe cases, the majority of the patients die before suppuration takes place. The glands are very tender to the touch and most painful during the inflammatory process, the pain may be so acute as to keep the patient awake at night. When the patient's mind becomes affected, the evidence of pain in the gland can only be elicited on deep pressure and the inconvenience he feels by the attitude he assumes.

Diagnosis. The main points in the differential diagnosis of plague are (1) sudden onset of the disease, (2) temperature and its course, (3) congested conjunctivae, thickness of speech and intoxicated appearance, (4) early and extreme prostration, (5) early and marked delirium with sleeplessness, (6) buboes, (7) finding the bacillus pestis in gland, sputum, faeces, urine, or blood.

Symptomatology of the Glasgow Cases.

There were 36 cases. Of these 26 were admitted to Belvidere, 2 occurred there and 8 died in their own homes from what was no doubt plague from the symptoms of their illnesses. The first known case occurred on the evening of August 3rd. 1900, and the last known case was admitted to Belvidere on September 20th, 1900, from Professor Macewen's Ward in the Western Infirmary, where it has been sent from Govan for a suraical operation on a glandular swelling.

Type of disease. - All the cases were of the bubonic type for although 5 of the 8 who died at home were certified as having died of pneumonia, 3, gastro-enteritis, 1, and zymotic enteritis, 1. I afterwards learned on close inquiry that in each case there was distinct painful glandular enlargement. There was a case of pneumonia removed from the Western Infirmary to Belvidere as a suspected case of pneumonic plague, but this case is not officially recognised as plague.

Degree of severity of the attack. - The 26 cases admitted to hospital may be divided into

three groups. In the first group there were 8 cases which may be classed as very severe and of these 5 died. In the second group were 11 cases not so severe but intermediate between the first and third group of 7 cases which were so mild that they could only be recognised by the fact of their contact with plague cases.

Sex - 20 were females and 16 males.

Age - From birth 1; 2 months 1; 14 months 1; one to ten years 4; ten to twenty 7: twenty to thirty 11: thirty to forty 4; forty to sixty 7.

Incubation. - So far as it could be ascertained it was the usual plague period of 3 to 8 days.

Onset. - In almost all the cases the onset was sudden. It was usually ushered in with a rigor, severe headache, nausea and vomiting and a general feeling of malaise and prostration. The intensity of these initial symptoms was in proportion to the severity of the case and in the 7 mild cases there was only headache, slight malaise and rarely vomiting. In none of the cases in children was there convulsive seizure at onset. The facies of the patient was in most cases characteristic.

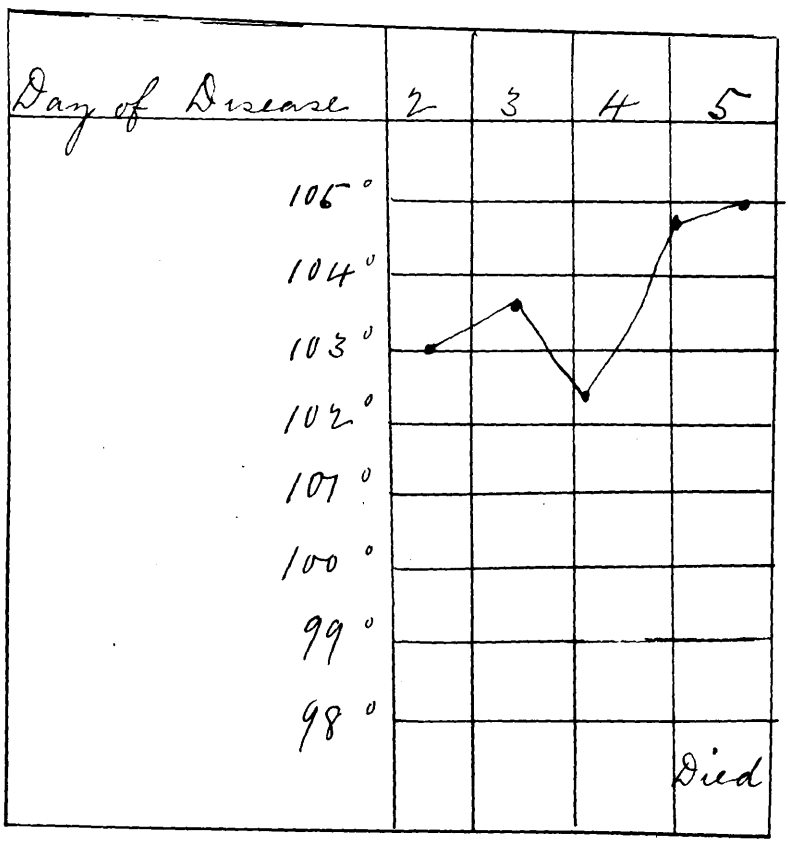
In the severe cases the expression of the face was heavy and dull with an anxious look and a degree of stupor depending upon the gravity of the illness. In the milder cases the patients looked ill, out of all proportion to the fever or the dearee of enlargement of the glands, and they had this facies even for some days after the disappearance of all constitutional symptoms. A greyish pallor of the face with injected conjunctivae and dilated pupil were noted in the severe and to a less extent in the mild cases.

Temperature. - In the severe and intermediate severe cases the initial temperature was high and ranged from 102 F. to 104 F. In the seven mild cases the initial temperature as well as the temperature throughout the few days they were ill was usually about 99 F. and seldom 100 F. The course of the temperature in the severe cases resembled that of the type of true bubonic plague and was not unlike the pyrexia in typhus. In all these cases a crisis was present from the twelfth to the twentieth day. In those cases in which the buboes suppurated and the inflammation spread to the surrounding tissue the temperature varied with the extent of the inflammatory process, while in

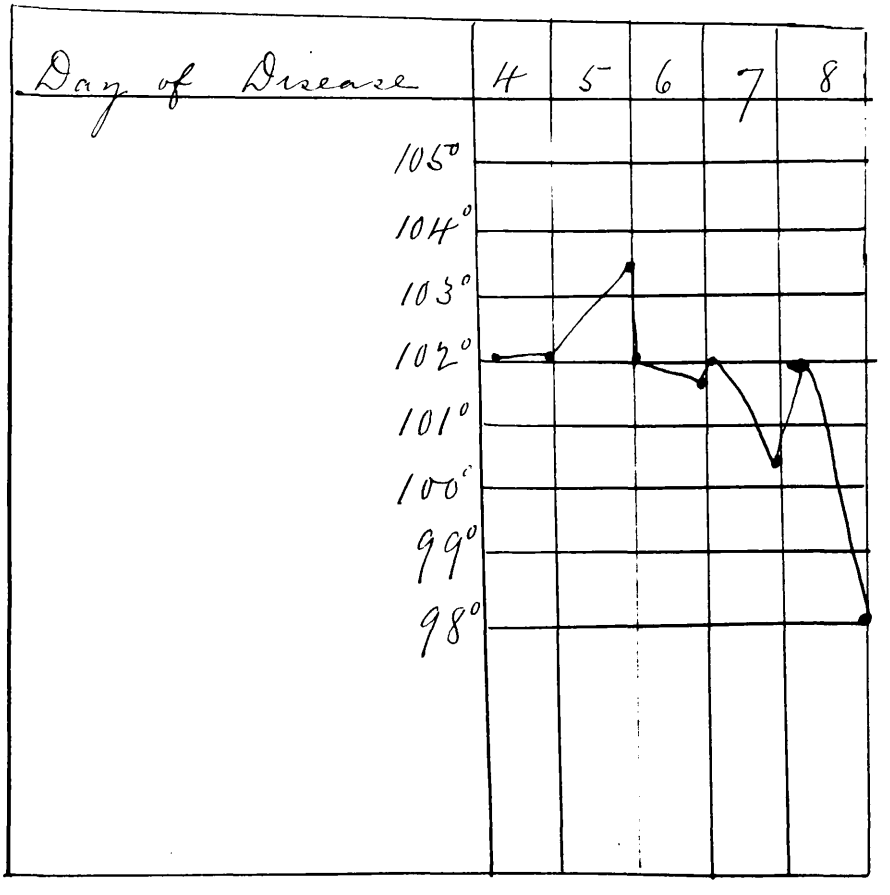
other cases it was modified with Yersin's serum.

The following charts of the Mallows may be taken as typical of the temperatures of the three groups of cases excepting the cases where the illness was much more prolonged and ended in recovery or death from asthenia. In one of these latter cases, on admission to the hospital on the sixth day of onset of illness, the temperature was 104.6 F. For four days it ranged between 103 and 105 F. when he received an intravenous injection of 15 cubic centimetres and a subcutaneous injection of 25 cubic centimetres of Yersin's antiplague serum. The following day there was no remission of temperature but his glands were less painful, and on the second day after the injection the temperature fell to 100 F. and he felt much better. The pyrexia remained low for three days when it again rose to 101 F. which was due to suppuration of his bubo and then gradually became normal. In another case the temperature after admission to the hospital ran a febrile course varying between 100.6 and 103 F. for ten days when there was a distinct crisis and it fell to 97.6 when the patient felt much better. Four days later it again rose due to suppuration of a large bubo in his

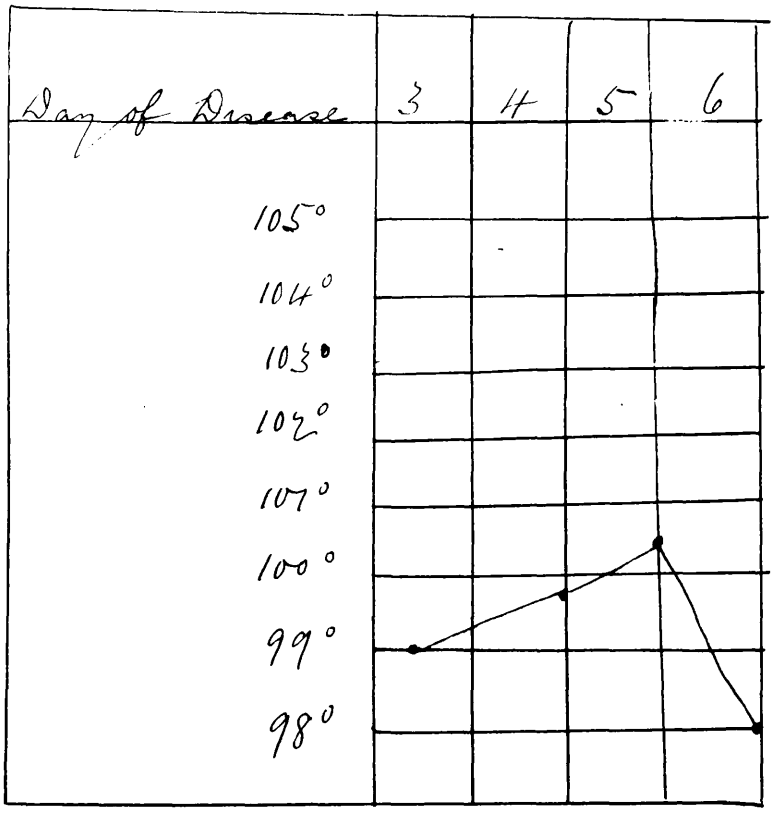
Patrick Malloy. severe Case.



Mrs. Malloy, moderately severe Case.



Wm. John Mallon, mild Case.



groin and it reached 101 F. and kept fluctuating between this and 102 F. till the patient's death six weeks after his admission to the hospital. In this case the inflammatory process was very extensive spreading from the suppurating bubo in the groin downwards to the mid thigh and backwards to the buttock and upwards until the whole abdominal wall pitted on pressure.

Alimentary system. - The tongue in the severe cases was moist and usually covered with a greyish fur on the dorsum and the tip and edges were clean. It was only in the two cases that ran a fevered course for 20 days that there was any tendency to dryness of the tongue. In the moderately severe cases the tongue was simply a furred tongue while in the mildest form it had no characteristic feature.

Nausea and vomiting were, excepting the headache and rigor, the most common initial symptoms. Nausea or a feeling of sickness was present in almost all the cases while vomiting was present in about 70 per cent of the severe and moderately severe cases but seldom present in the mildest cases. The vomiting usually persisted for two days, but in Mrs Malloy's case it remained one of her most distressing

sumptoms for six days. The vomited matter was bilious and haematemesis was not observed in any of the cases.

Diarrhoea was a prominent sumptom in three of the first cases that died outside the hospital and gave rise to the diagnosis of enteritis or enteric fever. In the 19 severe and moderately severe cases admitted to the hospital 7 had diarrhoea, which did not persist for any length of time. 8 had constipation and in 4 the bowels were not irregular, while in the mild cases there was neither diarrhoea nor constipation. No blood was noted in the stools of any of the patients.

In most of the cases there was a complaint of slight abdominal pain at onset which did not persist and was probably associated with the vomiting and irregularity of the bowels. Only a slight enlargement of the spleen was noted in some of the cases and enlargement of the liver was not noted.

Tenderness or pain in the right iliac fossa was not noted.

Circulatory system. - In the severe cases the pulse was rapid from 120 to 130. When noted on the 2nd. or 3rd. day of onset of illness it was feeble and usually dicrotic and in fatal cases towards the end irregular. In the moderately severe cases it was 100 to 110 more or less feeble in quality, while in

the mild cases except a little quickening it was normal.

In the severe cases the first cardiac sound was feeble and indistinct and in one case a faint systotic murmur was heard both at the apex and in the pulmonic region. No pulsation of the carotids was noted.

Respiratory System. - As already said none of the cases belonged to the pure pneumonic type of plague. Hence the respiratory symptoms and signs had no special characteristics. As in other plague epidemics it was noted in almost all the severe and moderately severe cases that the respirations were more rapid than could be accounted for by the fever and no pulmonary lesion was discoverable. For example in Mrs. Malloy's case on the 8th day the respirations were 30 and 26 with corresponding pulse rates of 78 and 64 and temperatures of 102 and 101.8. In all the severe cases there was basal congestion and in one case that recovered there was an attack of acute lobar pneumonia six weeks after admission to the hospital.

Urinary System. - Presented nothing characteristic. There was the usual scanty urine, high specific gravity and acid reaction of the febrile state. A trace of albumen was

noted in some cases. No suppression of urine was observed.

Cutaneous System. - No definite skin rash was noted. In all the severe cases there was a mottling of the skin of a faint purplish hue and most marked across the lower part of the abdomen, the arms and the buttocks. This eruption had a resemblance to the mottling of typhus. In the moderately severe and mild cases the skin was free from any eruption and it will be observed that in none of the 36 cases was there present any of the grosser haemorrhagic skin lesions which in the plague epidemics of olden times caused the disease to be called the "black death."

Nervous System. - Headache was the most constant symptom in all the cases. In some cases it was very severe and persistent especially in the case of Mrs Mallon in which it persisted till the fall of temperature on the 8th day of her illness. Muscular weakness and prostration were present in all the cases, its dearee being proportionate to the severity of the case. Tremors of the muscles and loss of co-ordination as evidenced by the staggering gait of the patient seen in Pat Mallon's case was typical of the severe cases. Delirium was

markedly present in severe cases, less so in the intermediate cases and absent in mild cases. As a rule it was present at or shortly after the onset of the disease, most marked at night, and persisted till the fall of pyrexia. In Mallon's case it was of a quiet type, the patient always wanting to get up and constantly muttering incoherently, but in another of the severe cases it was of a violent type. This patient took ill in the morning, was unconscious at noon, and at night and during the night was very delirious and violent. He was removed next day to hospital where he was evidently not conscious of his surroundings. When he was left alone he tossed himself restlessly about in bed, but when touched he resisted strongly and it was impossible for the nurses to wash him. It was noted that his resistance was most marked if palpation of his left axilla was attempted where there was a mass of enlarged glands, evidently acutely painful, and this movement of resistance made in opposition to the examination of these glands was that of a person conscious of his actions and not the movements without a purpose that are seen in the delirium of typhus or enteric. At midnight he received an injection of 20 cubic centimetres of Persin's serum, the patient struggling violently while the injection was being made. On the following day his temperature had fallen 4 dearess and he became

quite conscious and said he felt better. He said that he remembered nothing from the onset of the delirium on Saturday at Midday till the evening of the following Monday when he found himself in the hospital. In another of the severe cases delirium with delusions lasted for 11 days, and in this case it was noted that although the temperature kept high from 103 to 105 F. one of the common nervous symptoms of high fever, subsultus tendinum, was absent.

In the moderately severe cases delirium was commonly present but less marked than in the severe cases and never violent. In my case of Rosina Murphy it persisted till the fifth day of onset of disease, and in another case it was present till the crisis on the eleventh day from the onset of illness.

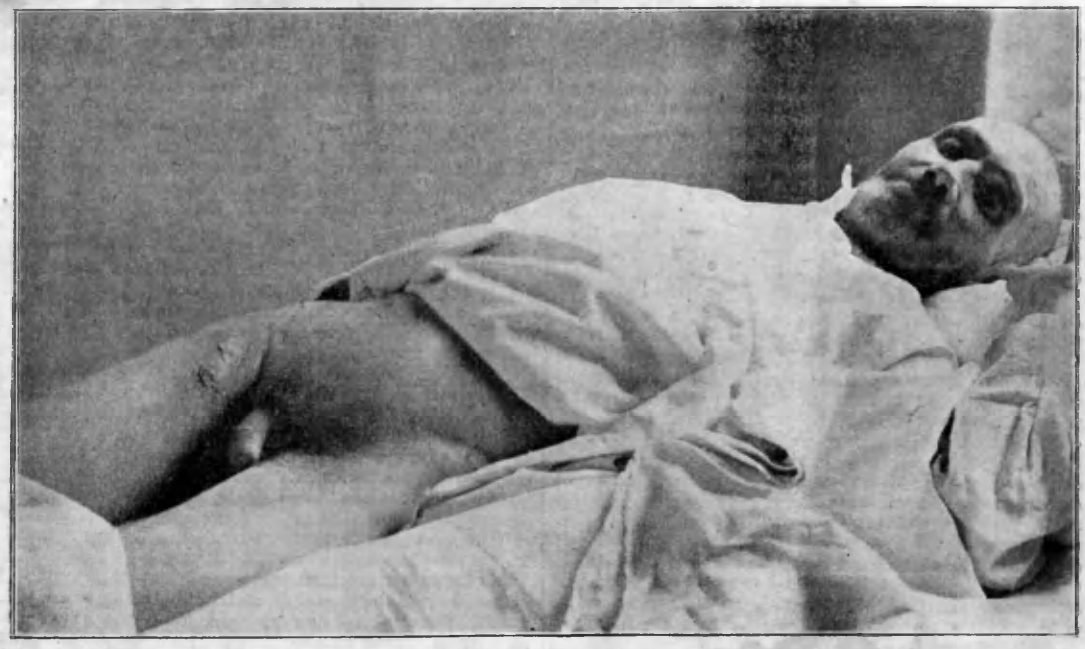
In the 7 mild cases there was no delirium. The less important nervous symptoms noted were sleeplessness in most but the mild cases, thickness of speech in severe cases, huperaesthesia in the beginning of the fever, and hiccough in one case.

Lymphatic system. - As already stated, buboes were present in the 36 cases. Of the 26 cases admitted to hospital the buboes showed all degrees of severity from the single slightly enlarged gland of the abortive type through the very large and very tender buboes of the generalised type, with or without reddening and oedema of the skin, to the large and intensely inflamed mass of glands seen in the case of solitary buboes where the redness of the skin and oedema of the neighbouring tissues might extend nine inches or even more from the centre of the disturbance. The date of appearance of the bubo so far as could be ascertained was generally during the first two days of the onset of definite symptoms of illness. It was preceded by tenderness over the seat of the gland affected soon followed by a swelling which in the severe and moderately severe cases was acutely painful. As in other epidemics the most common situation of the bubo was the groin. Of the 26 cases, 10 had buboes in the groin, 8 in axilla, 5 in neck, and 3 had generalised glandular enlargement. The degree of severity of the bubo had some relation to its situation for in all the severe and moderately severe cases the glands

affected were the inguinal or the axillary, while in the very mild cases it was the submaxillary, cervical, or submental. In the eight cases that died the enlargement was in the inguinal region in 5, in axilla in 2, and in neck in 1. The seat of bubboes in the seven mild cases were in the submaxillary 3, axilla 2, anterior cervical chain 1, posterior triangle of neck 1. A solitary bubo consisting of a mass of glands was, with three exceptions, the rule in the severe and moderately severe cases. Suppuration was the rule in the solitary bubo. In Mrs Mallon's case, which may be taken as a type of the moderately severe cases, she said that the day after the onset of headache, nausea and vomiting, she felt uneasiness in her right groin, and on examination found a small swelling which was very tender to the touch. When seen three days after, there was a large mass in the right groin composed of enlarged lymphatic glands surrounded by inflammatory tissue. The skin over the mass and for some distance around was reddened and oedematous and most tender to the touch. Six weeks after admission to the hospital the bubo burst and discharged for nine days when it healed. In one of the most severe cases that of Fagan,

there was in the right groin a large tense red oedematous swelling occupying the situation of the verticle group of glands. (See photo) The swelling was exquisitely tender and the tissues surrounding it were so infiltrated as to make the appreciation of individual glands impossible. After admission the redness and oedema surrounding the swelling in the groin slowly spread until it extended downwards almost to the mid-thigh and backwards over the buttocks. At the same time oedema without redness spread from the right iliac region until nearly the whole abdominal wall pitted on pressure. This was associated with a distinct increase in the abdominal distension. The inflammatory process reached its height four days after admission to the hospital when there was a slow but distinct subsidence of the redness and oedema around the bubo. Five days later suppuration began in the bubo as evidenced by secondary fever, and this was followed by sloughing of the skin and a gangrenous opening discharging pus with extensive suppurative infiltration of the surrounding tissues of the thigh and the pelvis. He died six weeks after admission to the hospital. Of the buboes in the patients who died, 3 were in the acute stage without suppuration, in 1 the bubo consisted of several glands

James Brogan, aet. 60.
Had a large Bubo in right groin, which suppurated and discharged with extensive infiltration of surrounding tissue.
Died 6 weeks after admission to Hospital.



Taken from Lancet, September, 15th, 1900.

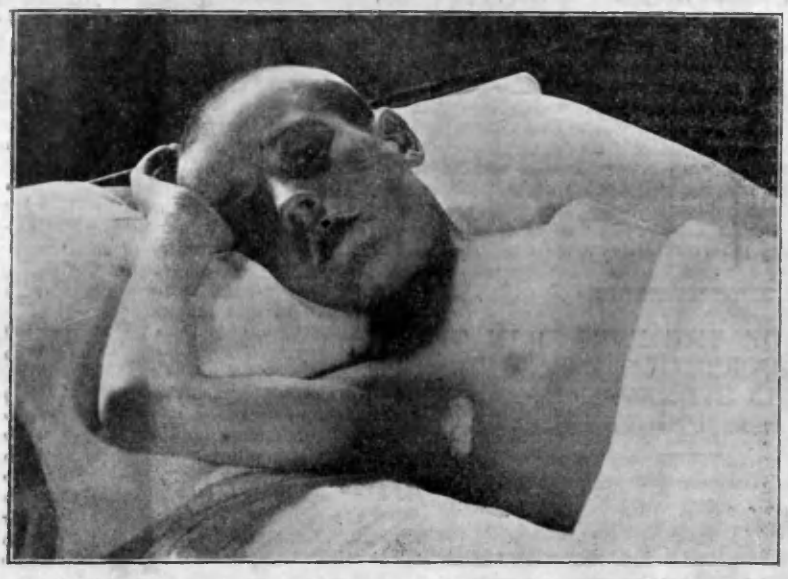
in the condition of distinct abscesses and in 2 the necrotic and suppurative process resulted in gangrenous openings discharging pus and connected with extensive suppurative infiltration of the tissues of the thigh and pelvis. The bodu of the infant born in the hospital of a plague mother and who died when it was 10 days old, presented post mortem a chain of characteristic buboes on each side of the neck involving the deep cervical glands. In the very mild cases the glands were only slightly enlarged with a little periglandular infiltration and only in one case redness of the skin. Tenderness was usually present on the second day but not so marked as in the other cases. It disappeared after two or three days though the swelling could be felt for nearly a fortnight later.

Duration of the disease and its after effect up to the end of May. 1901. or 9 months after the Outbreak. -

The most fatal period of plague is usually the fifth or sixth day and of those who died inside and outside of Belvidere, death occurred in most of the cases on or before the sixth day. The severe and moderately severe cases that recovered were kept in hospital from two to three months before they were discharged well.

During the week ending May 25th, 1901, I have examined at their own homes the three living severs, and the principal moderately severe cases with the following result: Charles Mc Menemy, aet. 30, Carter, now residing at 31 Dale Street, S. S., was admitted to Belvidere, September 9th, 1901, in a highly delirious and most prostrate condition with a temperature of 105.8 F. pulse 134 and respirations 40. His left axillary glands were enlarged and acutely painful. Delirium of a violent type when interfered with was a prominent symptom of his illness. He was unconscious from mid-day on Saturday till the evening of the following Monday, when he found himself in hospital and remembered nothing that had taken place since the Saturday. He was treated with Persin's serum. Three weeks after admission to the hospital the axillary bubo burst and discharged for 7 weeks before it healed. He was in the hospital 10 weeks. On examination, (23rd May, 1901) he said that he felt so well after coming out of the hospital that he resumed work as a carter the following week. His work was no light occupation as he had heavy goods to lift, but he felt quite equal for the strain and had enjoyed excellent health since. I found all his organs normal. A slight scar was seen in left axilla which is still tender but no indurated glandular enlargement could be detected.

Thomas Horn, aet. 16,
Bubo in Right Axilla.

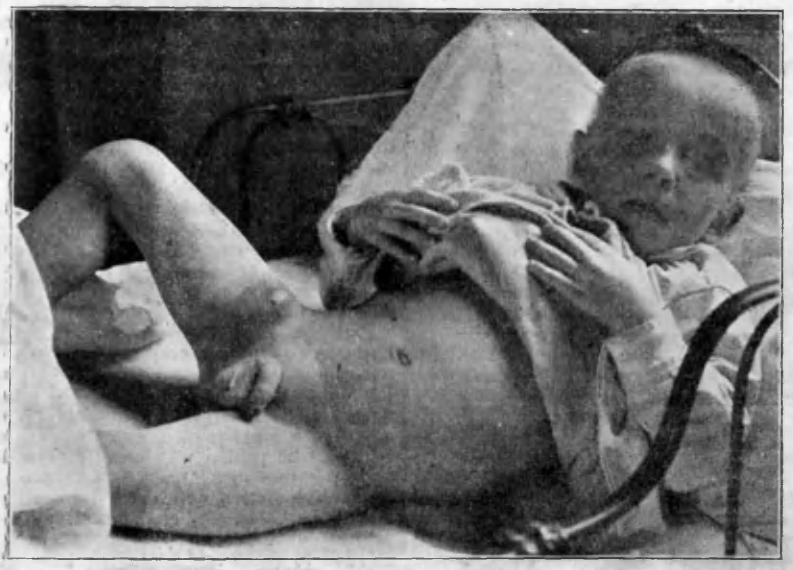


Taken from the Lancet, Sept. 15th, 1900.

Thomas Horn, aet. 16, now residing at 7 South Coburg Street was admitted to Belvidere on August 29th, 1901. He was conscious, but at times delirious and was also very prostrate. His temperature was 104.6 3, pulse 140, respirations 26, with general enlargement of the glands in the groins axillae and neck. the most prominent bubo being in the right axilla. (See photo) He was treated with Dersin's serum. None of his glands opened and discharged. He was 13 weeks in hospital. On examination, (23rd May, 1901) he said that he felt quite well on leaving hospital and was able to resume work the following week as a message boy. Since then he has had excellent health. In his right submaxillary region there could still be felt distinct indurated glandular enlargement which was not tender. No glandular enlargement could be detected in his axillae or groins.

Mrs Tierney. aet 40. housewife, now residing at 18 Errol Street, N. S. was admitted to Belvidere Hospital a fortnight prior to the Mallows, and as on admission she had a history of lately giving birth to a premature baby, a provisional diagnosis of pelvic cellulitis was made. When the question of plague was raised in the Mallow cases she was also found to be suffering from that disease. She had a large bubo in

Denis Tierney, aet. 7. Bubo in Right Groin.



Taken from Lancet, September, 15th., 1900.

her left groin which suppurated and burst 6 weeks after admission to the hospital and discharged for a month before healing. During the fortnight in the Puerperal Ward in Belvidere before the question of plague was raised she was very delirious and most prostrate. She was altogether 3 months in hospital, and during that time had an attack of left lobar pneumonia from which she made a good recovery. On examination (21st May, 1901) all her organs were found to be normal and she expressed herself as being in excellent health since leaving the hospital. In her left groin was a distinct scar about 2 inches, and around it was the resulting inflammatory fibro-cicatricial thickening. No glandular induration could be detected.

Denis Tierney, aet. 7, her son, was admitted to hospital a fortnight later than his mother, with a bubo in his right groin which suppurated and discharged three weeks after admission but soon healed. His was a mild case. He was in hospital 2 months. On examination (21st May, 1901) he is and has been in excellent health. A distinct scar of about one inch is seen in right groin and around it a mass as large as a chestnut of indurated enlarged glands can be felt. This was the only case examined in which the glandular enlargement was so palpable.

Mrs Molloy, aet. 41, housewife, now residing at 4 Spring Lane, E. E. Her case was

fully described in the outbreak. The bubo in her right groin suppurated and discharged 6 weeks after admission to hospital, healing 9 days later. She was three months in hospital. On examination (May 20th, 1901) all her organs are normal. She says that she had not felt so well since she had the attack of plague, although she appears to be in excellent health. A distinct scar about two inches long is seen in right groin which is not tender. There is the resulting fibro-cicatricial thickening, but no indurated glands can be detected.

Wm. John Malloy, aet. 3, her little boy, whose case is also described in the outbreak and was of a very mild type. He was two months in hospital and came out in excellent health. I was called to see him on March 11th, 1901, five months after leaving Belvidere and found him suffering from Scarlet Fever. He was removed to Kennedy Street Hospital where he remained for two months and returned home well. On examination (May 20th, 1901) he is in excellent health and no indurated glandular enlargement can be detected.

Rosina Murphy, aet. 24, hair-worker, now residing at 131 Rose Street, S. S. whose case has been fully described in the outbreak. The right bubo suppurated and discharged 14 days after admission to the hospital, and did not heal for two months. She was two months in hospital. On examination (21st May, 1901) she said that she did not feel well on leaving the hospital, and was not able to resume her work for a

fortnight later when she regained her normal strength and has been in excellent health since. A scar about three inches is seen in right groin which is not tender. There is a little libro-cicatricial thickening but no glandular induration can be detected.

Patrick Ford, aet. 60, shoemaker, now residing at 71 Rose Street, E. E., was admitted with a solitary bubo in left groin which was incised 9 days after admission to the hospital and took a month to heal. He was 3 months in hospital. On examination (May 19th 1901) he said that he felt so well on leaving the hospital that he resumed his work at once. All his organs are normal and in excellent state of preservation for his age. A scar about two inches, not tender, and with a little libro-cicatricial tissue is seen in left groin but there is no nodular glandular enlargement. He says that he still has, at times, a feeling of numbness in the anterior surface of left thigh down to the knee.

Mrs. Muir, aet. 41, housewife, now residing at 11 South Stirling Street, was two months in hospital with solitary bubo in right groin which did not suppurate. Hers was a mild case, and on 20th May, 1901, she was in excellent health with no evident nodular glandular enlargement.

Mary Muir, aet. 14, her daughter, was also two months in hospital with axillary bubo that did not suppurate. This was also a mild case. She resumed work immediately on

coming out of hospital, and on 20th May, 1901, is in excellent health with no evident nodular glandular enlargement.

Mortality. - Of the 26 cases admitted to hospital 8 died, the cause of death being recorded as the action of the toxin on the heart. This gives a mortality of 28 per cent which is approximately the same as in recent outbreaks. In Smyrna it was 40 per cent, in Sydney 30, in Alexandria 48 and in Oporto among cases not treated with serum 63. Including the 8 cases occurring at home before the plague was recognised, the death rate in the Glasgow outbreak was 44.4 per cent. In the Hong Kong epidemic of 1894 the death rate in the Chinese hospitals was 95 per cent, while in the recent Indian epidemics the mortality among natives was between 60 and 70 per cent and among Europeans between 30 and 40 per cent.

Cases that simulated Plague.

It is evident that the early diagnosis and complete isolation of cases of plague was of supreme moment, on account of the far-reaching results, and as my work was mainly in the infected area every case that came under my care that simulated plague was carefully observed during the three months that the city was under the ban of a plague-stricken city. And while the city has now (10th May) been free from plague for the past six months it is well to remember that plague has a tendency to recur, once it has attacked a community, and its possibility should still be considered in any obscure febrile condition with glandular enlargement. That the first cases of plague to which the Glasgow outbreak was ultimately traced simulated enteric fever was evident from the fact that the medical man in attendance certified Mrs. Eogan, as having died of "acute gastro-enteritis", her grandchild as dying of "zymotic enteritis", and sent her husband to Belvidere hospital certified as suffering from "enteric fever." I have already noted the points of resemblance to enteric in the Malloy cases and if we exclude the bubo, which no man would search for unless a suspicion of plague was entertained, or his attention drawn to the

region of a gland by the patient himself, the mild forms of plague in the Glasgow outbreak and an atypical form of enteric had many symptoms in common.

A Study of Enteric Fever in its initial stage.

The fact cannot be too strongly emphasised that the early diagnosis of any infectious disease is of dual importance. It is of the utmost importance to the patient himself inasmuch as it enables us to confine him to bed at once and thus early in the disease conserve his energy to contend against it in its later stages. It is, likewise, of equal importance to the community as it leads to the patient's complete isolation and thus prevents the spread of the contagion to others. Of all the infectious diseases, it will be readily granted, that there is none so perplexing to the mind of the physician on account of the difficulties that beset its diagnosis than enteric fever in its initial stage. From personal observation I venture to assert that there is no disease so often missed in general practice, especially in children, and missed with so much disaster to the patient and to the community, as early enteric. This is not only my own but the opinion of every general practitioner -

and they were numerous - that I consulted on the subject and many of them, like myself, can look back to puzzling febrile cases which in the light of later and riper experience they would diagnose as enteric. Again and again we read in the medical, and even in the lay, press of serious outbreaks of enteric in town and village that were ultimately traced to some ambulatory case that was thought to be "influenza", "bronchitis", or a mild case of "pneumonia." Clinically early enteric may simulate almost any febrile condition that has no evident or discoverable cause to account for the pyrexia. Moreover, the disease at its onset may be masked by some prominent symptom, or symptoms, and the question of enteric only raised when after a week or more the classical symptoms of that disease become manifest. On the other hand there may be no symptoms whatever of the disease except the diurnal remittent temperature and the diagnosis only arrived at after a length of time by a process of exclusion. A common source of error is in making a positive diagnosis, on first examination of a case of enteric, of some disease which the prominent symptom, or symptoms, then suggested and when later the typical symptoms of enteric appear not assigning to them their proper value on account of the biassed opion of the first diagnosis. During the past seven years I

have had under observation 86 cases of enteric fever in the initial stage of the disease. By the term initial stage is meant the first week of the onset of definite symptoms of illness and before the appearance of the classical abdominal symptoms and signs of the disease. The following are the symptoms observed, or history of symptoms noted, in my 86 cases during the stage of the disease mentioned:

Sex - 61 cases were males and 25 females.

Age - Five to fifteen, 11; fifteen to twenty-five, 34; twenty-five to thirty-five 29; thirty-five to forty-five, 10; forty-five to fifty-five, 2.

Social Status - 82 belonged to the working class and 4 to the middle class.

Onset - 74 cases were insidious and 12 were sudden. Of the sudden cases 7 were children.

Anorexia - 76 cases.

Lassitude and aching in the back and limbs when in motion - 72 cases.

Headache - 71 cases.

Sleeplessness - 60 cases. When asleep for a short time the sleep was usually disturbed by dreams or slight delirium.

Constipation - 35 cases.

Diarrhoea - 30 cases. The diarrhoea here meant is the initial diarrhoea and not the characteristic diarrhoea of enteric. In most of these 30 cases a laxative or cathartic had been administered before the onset of the diarrhoea.

Cough - 26 cases.
Vomiting - 12 cases.
Epistaxis - 10 cases.
Sore Throat - 5 cases.
Erythematous rash - 3 cases.
Deafness - 2 cases.
Tenderness on pressure in right iliac fossa - 2 cases.
Enlargement of spleen - 1 case on child on 6th day of onset of noticeable symptoms of illness.
Temperature - As a rule it ranged from 100 F. to 103 F. and was only taken once a day till a suspicion of enteric was raised.
Pulse and Respiration - The rate as a rule was proportionate to the height of the pyrexia. Pulse tracings were not taken so that dicrotism which is said to be more often associated with early enteric than any other acute disease was not noted.
Ehrlich's or the Diazo-reaction - The urine was not tested for this reaction.
Widal's reaction - A positive reaction was obtained in all my later doubtful cases.

On comparing these initial symptoms with those present in other febrile states, infectious or non-infectious, it is evident that there is no pathognomonic symptom of early enteric. But while no single symptom by itself is of any diagnostic value there can be no doubt that an association of certain symptoms will often enable us to arrive at an early and correct diagnosis of the disease.

One of the most characteristic of these associated symptoms is the mode of onset. With the exception of phthisis pulmonalis there is no common febrile condition in which the onset is so gradual and so insidious as enteric in an adult. Of 75 of my adult enterics, in only 5 was the onset sudden. Of these 3 were males and confirmed alcholics, while of the 2 females one had not recovered from the effects of the puerperium and the other had suffered much from chronic poverty. Hence a possible explanation in these cases of the suddenness of the onset is that the resisting power of the individual was so lowered by ill-care, or disease, that it could not resist for any length of time the attack of the bacillus typhosus. Then in no less than 7 of the 11 cases in children had a sudden onset. Here again it will be evident that on account of its immaturity the resisting power of a child's tissues will be less than that of adult tissue and that the onset of a disease in a child will be more sharp and more explosive than in an adult. A case is recorded by Osler and one by Holt in which the onset of enteric in a child was ushered in with convulsions, but this is extremely rare. Three of my 7 cases in children with a sudden onset were most instructive, as in each case a provisional diagnosis of scarlet fever was made on first examination and the patients removed at once to

Belvidere on account of the density of the locality in which they lived. Their ages were respectively 10, 11 and 13, and two of them occurred in the same house a day after each other. The onset in each case was sudden with a temperature of 102 F. or 103 F. vomiting, headache, rapid pulse, sore-throat and an erythematious rash on the chest and abdomen as well as on the extremities. The rash was not punctiform nor was the rash rose-coloured spots. There was no doubt of the sore-throat for I observed the fauces and tonsils much inflamed. I have also observed the same form of sore-throat in 2 adult enterics. On being informed that the provisional diagnosis of scarlet was incorrect and that they were cases of entertic, I wrote to the Medical Officer of Health and raised the question of enteric plus scarlet. I was afterwards informed by the physician - superintendent of Belvidere - that at the end of six week's observation they had ran a course of uncomplicated enteric. Jenner has noted a case of early enteric in which there was a bright scarlet rash and which was mistaken for scarlet fever. Similar cases have been recorded since his day while Murchison has recorded cases in which scarlet fever ran a concurrent course with enteric fever. My 3 cases had no resemblance to the exanthematic form of infantile enteric described by Weill (British Medical Journal March 31st, 1900). The characteristic of

this form is the copious rash of rose-coloured spots which come out early, sometimes on the fourth day, and may cover the whole cutaneous surface. There is no vomiting, no sore-throat and the abdominal symptoms and signs are very mild. It is well to note that the history of onset of symptoms in a child as detailed by itself, or its parents, is of less value than the same history in an adult, and in 69 of my adult enteric cases the history was clearly that of a slow and insidious disease. Indeed the characteristic point in their history was that they knew that they were ill, but on account of the absence of an outstanding symptom pointing to some particular organ of the body they could not define their illness. The symptoms of gastro-intestinal irritation were too mild to convince them that they had an "inflammation," - the term inflammation bulks largely in the lay mind - of the stomach or bowels, just as the little cough and respiratory symptoms were not severe enough in their opinion to constitute an "inflammation" of the chest. Hence the worried look and anxiety written on their face to know what was really the matter with them.

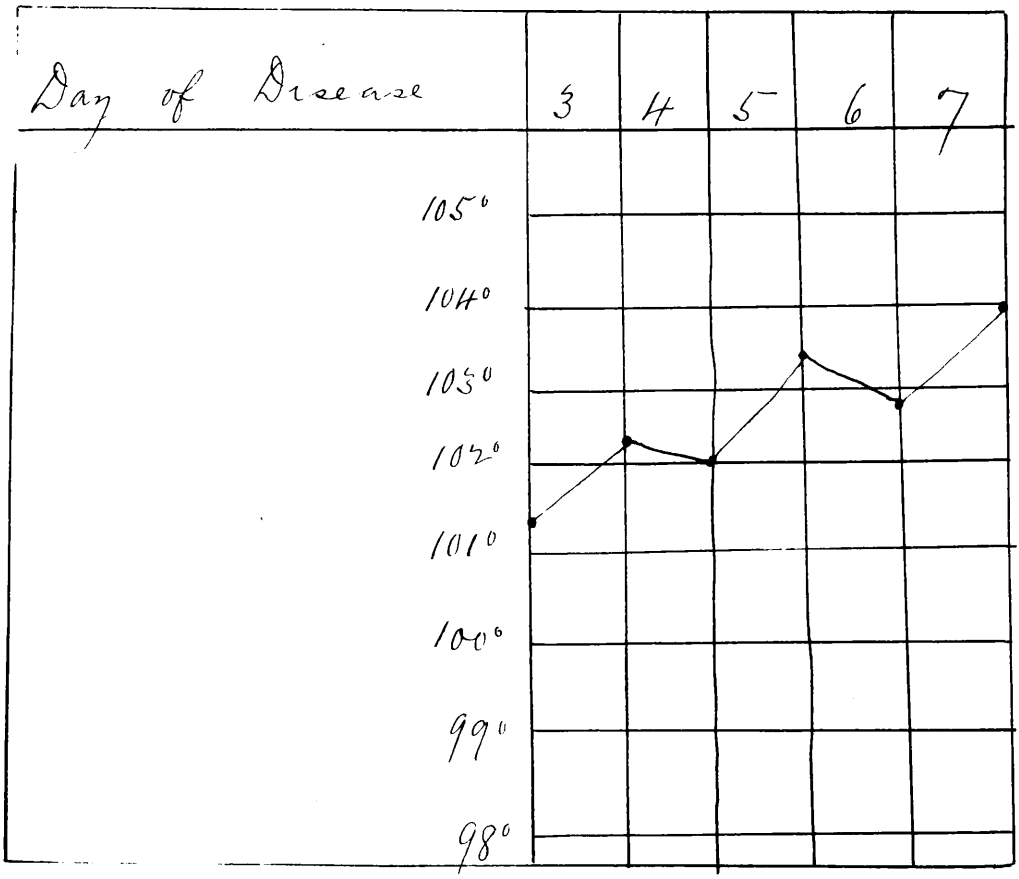
Another associated symptom of diagnostic value in early enteric is epistaxis. It is of less value in a child than in an adult, for epistaxis is a common affection of childhood from a variety of causes. The

blood in epistaxis is due to diabedesis or capillary oozing, and probably results from the cerebral congestion of the febrile state. Why it is more common in enteric than in any of the other exanthemata may be due to the fact that in that disease in the early stage there is no gross lesion with huperaemia, no copious huperaemic skin rash as in measles, scarlet, smallpox and typhus, to relieve or deplete the cerebral circulation. Be that as it may the occurrence of epistaxis in an adult febrile condition, with no evident cause, and with the history of an insidious onset, ought to suggest enteric. In two of my later cases the appearance of epistaxis between my first and second examination of the patient caused me to have the blood examined for the Widal reaction and to obtain a positive result.

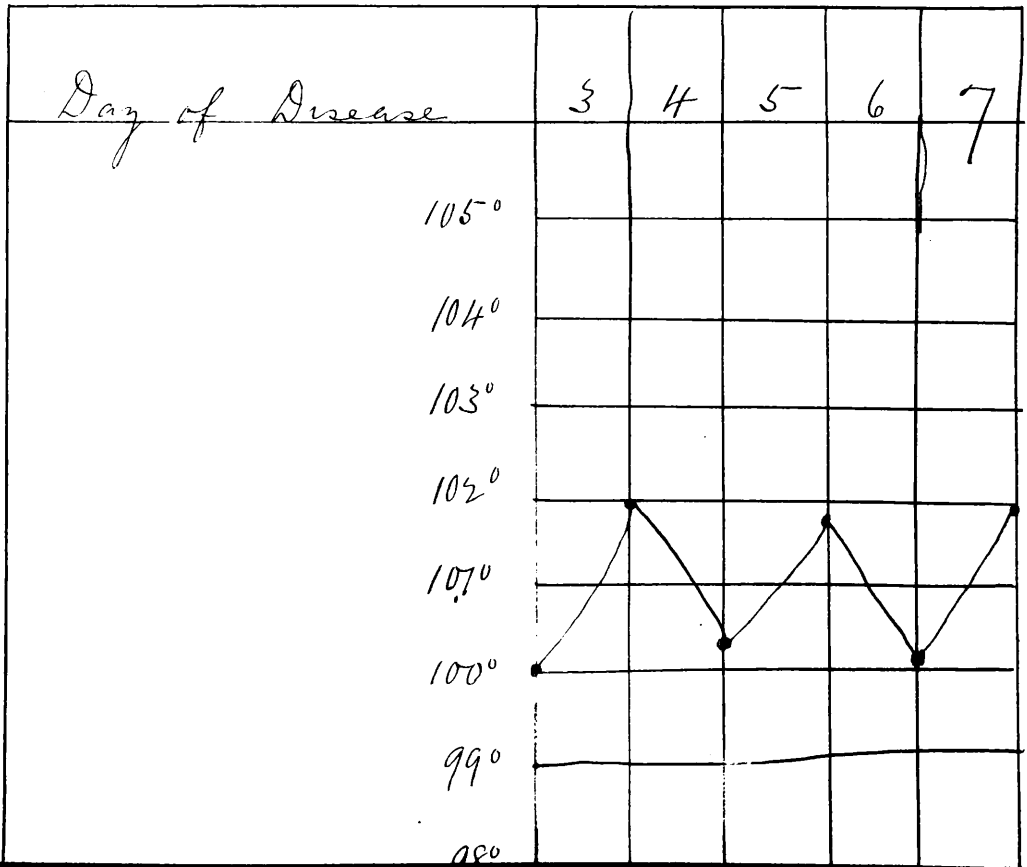
Another associated symptom of some value in early enteric is the little cough or the mildness of the bronchial symptoms, and the point I wish to specially emphasise is that a careful examination of the chest is often the key to the correct diagnosis inasmuch as it enables us to differentiate early phthisis and acute lobar pneumonia, the two diseases with which enteric is most apt to be confounded. The symptomatic resemblance of early phthisis pulmonalis to early enteric is at first sight marked. In both there is the insidious onset with increasing weakness, the impaired digestion and gastric disturbance,

the diurnal remittent temperature and the little cough and bronchitic symptoms. But on closer study it will be seen that apart from the tubercle bacillus in the sputa of the phthisical, and the Widal reaction in the enteric, there are other points in the differential diagnosis of the two diseases. The insidious onset in phthisis is, as a rule, much more prolonged and the weakness comparatively more gradual than in enteric. It is also accompanied with hectic symptoms, especially night-sweats, or there may be at the onset haemoptysis, or the husky voice indicative of laryngeal phthisis. The gastric disturbance in both diseases is similar except in that form of phthisis in which the mode of onset is pronounced dyspeptic symptoms, with an irritability, vomiting and acid eructation that are rarely found in enteric. Then there is a marked difference between the early daily remittent temperature. The characteristic remittent temperature during the first week of the onset of enteric is a steady rise in the pyrexia, the evening temperature rising a degree or so each evening until it reaches 103 F. or 104 F. while in phthisis there is a morning remission of a degree or two degrees but no marked elevation of the evening temperature above that of the previous evening. (See charts). But the main point in the differential diagnosis of phthisis and pneumonia from enteric is the careful examination of the chest.

I. Grant, aet. 23, Enteric.



M. Scott, aet. 29, Phthisis.



In 26 of my 86 cases of initial enteric there was a cough and symptoms of bronchial catarrh. In only one of these cases was there a localised pain in the chest. The sensation in the chest was a little tightness, or not the usual freedom in breathing. In no case could the breathing be accurately termed dyspnoeic. The cough was slight and by no means as troublesome as they have often experienced in what they termed a "bad cold". On percussion slight dulness was occasionally made out at both bases. On auscultation the general breath sounds were often normal, and when a deviation from the normal was present, it was exaggerated breathing, or a prolonged expiratory murmur. Basal breathing was seldom normal for it was either feeble or had prolonged expiration simulating bronchial breathing. Rales were seldom present unless at the bases where fine mucous rales were sometimes heard. There was no marked difference in the vocal resonance or fremitus. From a study of the physical chest signs of early enteric I am of opinion that they are due to active pulmonary congestion the natural sequence of the weakened cardiac action of the febrile state. In only one of my 86 cases was there in the initial stage a simulation of acute lobar pneumonia. The onset in this case was sudden with a temperature of 103 F., headache, vomiting, a moderate cough, and a localised pain in left chest but no dyspnoea. A careful examination of the chest

each day during the first week of onset of illness revealed nothing definite except basal congestion and the pain in the left side of the chest, which had not the severe character of the pleuritic pain of pneumonia, may have been due to gastritis as the patient had been drinking heavily a fortnight prior to his illness. The appearance of typical rose-spots on the abdomen on the eighth day of illness clinched the diagnosis of enteric. This case resembled the pneumo-typhoid form of enteric described by French and German writers in which the onset is characterised by the symptoms and signs of an acute lobar pneumonia. The exact nature of the pneumonic process in these rare cases is not yet decided as both the pneumococcus and bacillus typhosus have been found post mortem in the affected lungs. Some observers maintain that the pulmonary affection and the intestinal lesion are the result of the action of the bacillus typhosus while others hold the view that the onset of pneumonia with enteric is simply accidental. At anyrate I am inclined to think that the pneumo-typhoid form of enteric is rarely seen in this country. On the other hand early enteric is not uncommonly diagnosed in general practice as pneumonia, not because the typical symptoms and chest signs of that disease are present but because the bronchitic symptoms with the temperature of 102 F. and general constitutional disturbance are more suggestive of a sub-acute

pneumonia than any other febrile state when no suspicion of enteric is entertained just as a diagnosis of influenza is suggested when early prostration, severe headache, and cough and generalised pains predominate. Acute lobar pneumonia as a complication in the advanced stage of enteric does not come within the scope of my thesis, but it may here be said that a simple lobar pneumonia nearing the crisis when the patient sinks into a typhoid state, especially with diarrhoea, may be easily mistaken for a case of advanced enteric unless one has seen the case from the onset or differentiates with Widal's reaction. The following illustrative example of this also shows the absurdity of attempting to diagnose, on a first examination, what must have been at least a very atypical case of enteric.

A month ago (April 1901) I was called to M. C. aet. 21, outdoor labourer. He had a history of onset, and all the symptoms and signs of an acute lobar pneumonia. I saw him for three days and the disease ran its natural course, the patient becoming weaker each day. On calling on the fourth day I was surprised to find that my patient had disappeared and on inquiry learnt that, as he was getting worse instead of better, another medical man had been called in who had diagnosed enteric, with a suspicion of typhus, on account of a mottling on his forearms which is very common in outdoor workers and had the patient removed to Ruchill hospital for infectious diseases. I at once wrote to Dr.

Johnston, the physician-superintendent of Ruchill for his diagnosis of the case. He kindly informed me that on admission to the hospital enteric and typhus were excluded and as, on examination, some pleurisy was found at the left base and the patient had unmistakable evidence of having suffered from an acute disease, he had no doubt that it was a case of acute lobar pneumonia in which the pleuritic element had not resolved with the pneumonic process. That cases of simple acute lobar pneumonia are not uncommonly sent into fever hospitals certified as enteric is evident from the fact that according to the two latest published reports of the Glasgow fever hospitals for 1898 and 1899, among 589 certified enterics in the former year, and 1002 certified enterics in the latter year, there were 39 and 60 cases respectively of simple acute lobar pneumonia. A closer inquiry into the history of onset, as well as a more careful examination of the chest and a more common use of the Widal reaction should lead to this error of diagnosis being considerably reduced.

The physical chest signs of early phthisis when present can be easily differentiated from the chest signs of early enteric. The form of phthisis that is most apt to be confounded with early enteric is acute general miliary tuberculosis. In the two cases of this disease I have seen in general practice the one presented little difficulty in diagnosis for the patient

was a distinctly phthisical subject and had long suffered from an old tubercular knee-joint but the other case so closely simulated enteric that it had been diagnosed as that disease. While house-physician to the City of London hospital for consumption and diseases of the chest, I had special opportunities of studying chest work, and on careful examination in this case the signs of an old cavity could be detected at the right apex while the harsh exaggerated breathing with prolonged expiration and no rales to be heard all over the right chest, back and front, and the generalised feeble breathing accompanied with numerous fine crepitating rales all over the left side presented a clinical chest picture that could only be found in general pulmonary tuberculosis apart altogether from the strikingly urgent and rapid breathing of the patient. This case was before the introduction of the Widal reaction and the finding of the tubercle bacillus in the sputum and the subsequent course and termination of the illness left no doubt as to the diagnosis of general acute miliary tuberculosis.

Tenderness on pressure in the right iliac fossa is not so common an associated symptom of early enteric as is generally supposed. It is due to the inflammatory process in the solitary and agminated glands or Peyer's patches which are most abundant near the

ileo-caecal valve and will, therefore, be more often present in the later than in the earlier stages of the disease. It was only present in 2 of my 86 cases on the fifth and sixth day respectively from the onset of definite symptoms of illness. It is obviously of little diagnostic value in children. I have already noticed how tenderness in the right iliac fossa was one of the simulating symptoms of enteric in one of the first recognised Glasgow cases of plague and Cantlie says that it is not uncommon to find the primary mass of swollen glands in the iliac fossae which will be tender to deep pressure. The tenderness or pain in the right iliac fossa in enteric will rarely be confounded with the sudden sharp agonising pain of acute appendicitis.

To my mind the most delusive symptom in 35 of my 86 cases of enteric was the initial constipation for we so constantly associate diarrhoea with the symptomatology of enteric that we commonly fail to discriminate between the initial bowel symptom and the characteristic diarrhoea with pea-soup stools in the later stage of the disease. When consulted by a patient in a febrile state with no evident cause and with diarrhoea a suspicion of enteric is at once raised, but when consulted by a patient in a febrile state with no evident cause and with constipation, we rarely think of enteric unless we know that the patient has had direct or indirect association with enteric cases.

In all febrile condition there is irregularity of the bowels, and we find either constipation or diarrhoea. The febrile state tends, as a rule, to constipation, and when diarrhoea is present it is caused either by the action of a specific poison or by irritation from food which it is not able to digest on account of its lowered tone. The point to be specially noticed is that the initial bowel symptom in enteric may be either constipation or diarrhoea and that the specific diarrhoea of the disease is a late and not an early symptom. Maclagan in a suggestive paper has correlated the specific diarrhoea of enteric with the ulcerative process. He clearly points out that in the bowel lesion besides the action of the bacillus typhosus there is another agent at work, the irritating discharges of the sloughing glands. Clinically, he divides, enteric into three classes,- (1) Those in which the glandular lesion does not pass beyond the stage of inflammatory thickening but terminates in resolution without sloughing and in which convalescence begins at the middle or end of the second week of the disease,- (2) Those in which the lesion terminates in sloughing of the glands, but in which the process is limited to the glands and the mucous membrane immediately over them, and is accompanied by no evidence of active disturbance and no serious indication of bowel irritation and in which convalescence begins at the end of the third week,- and (3) Those in

which the sloughing process is more extensive and in which come into play other and new morbid agencies, the various forms of cocci associated with suppuration and sloughing. It is here that diarrhoea, haemorrhage, abdominal distension, peritonitis, with attendant typhoid general symptoms show themselves during the third and fourth weeks of the disease and in which life is seriously threatened by these complications. Hence diarrhoea is not an essential feature of enteric for many cases run their course from beginning to end without its occurrence and in not a few cases the bowels are constipated during the whole course of the malady. The latter are mild cases in which the bowel lesion is not sufficiently severe to give rise to any symptoms, and in which we know that it exists only from what we know of the natural history of the disease. Mere inflammatory swelling of the glands in the submucous coat without any participation of the mucous lining in the mischief is what takes place during the first week of the disease. It is not till the glands begin to slough and break down and the mucous membrane over them becomes involved in the morbid process that the characteristic diarrhoea is apt to occur. Such diarrhoea is symptomatic not of inflammation of the glands of the submucous coat but of the extension of the ulcerative process to the mucous membrane. In fatal cases in which diarrhoea has been a marked symptom during life, the turgid redness of the mucous membrane around and between the ulcers,

gives ample post mortem evidence of the extent to which that membrane had suffered during life.

It will be noted that in my 86 cases of enteric rose-spots, pea-soup diarrhoea, enlargement of the spleen (excepting one case) not febrile enlargement but enlargement evident on palpation or percussion, and the other prominent abdominal symptoms of the disease were not observed in the initial stage or during the first week of the onset of definite symptoms of illness. Hence while admitting the great difficulty of fixing the exact day of onset of definite symptoms of illness, I am of opinion that the classical symptoms and signs of enteric fever will oftener appear for the first time at the beginning, or in the earlier part, of the second week, than at the end of the first week of the disease, and that, in this country in a much larger proportion of cases than is generally supposed they will be absent from the beginning of the malady to its end.

Serum-diagnosis. - The value of Widal's reaction as an aid to an early and correct diagnosis in an atypical case of enteric cannot be over-estimated. Prior to its use in clinical diagnosis in cases with constipation and no definite symptoms beyond the daily remittent temperature and its resultants one was a fortnight, or even longer, in perplexity and doubt as to the exact nature of the

illness, and during that time the patient was partially, if at all, isolated, until on the basis of duration of fever and the exclusion of all other possible causes a diagnosis of enteric was made. With Widal's reaction we can now in at least 95 per cent of cases make a correct diagnosis on the sixth or seventh day of the illness and for his own good and the good of the community at large, have the patient sent to hospital or completely isolated. The serum diagnosis of enteric is due to the fact that clumping takes place when living and moving typhoid bacilli are placed in the diluted serum of a patient suffering from enteric. While the observations of others, and especially of Gruber and Durham, led up to this reaction, it was solely used in bacteriological diagnosis till June 1896, when Widal applied it to the clinical diagnosis of enteric. Since then the validity of the reaction has been tested on a large scale. The largest number of cases is that of Kneas and Stengel who found that in 2283 enteric cases the reaction was present in 95.5 per cent and in 1365 non-enteric cases there was no reaction in 98.4 per cent. Horton-Smith in the Goulstonian Lectures for 1900 states that in 97 per cent of all enteric cases it can be obtained at some period of the disease in a dilution of 1 to 20. It is evident that the value of the reaction depends to a considerable extent on the date of its appearance. The exact period is not yet definitely fixed but occasionally it has been obtained as early as the fifth day although the usual period is

about the seventh day of the fever. It may be delayed till the second week of the disease or longer and may be present at one period of the disease and absent at another. The reaction is more marked in severe than in mild cases of the disease. In regard to the fallacies of the test, the first to be noted is the complete absence of the reactiin throughout the disease in 4.5 per cent of all cases. according to Kneas and Stengel, and in 3 per cent of all cases according to Horton-Smith. Hence if the reaction gives a negative result, one is not to absolutely exclude enteric from the diagnosis for it may be one of the 4 per cent cases in which no positive reaction is obtained with a dilution of 1 to 20, and one hour limit. A second fallacy is in testing the blood at a too early stage of the disease and when a negative result is obtained to conclude that the case is not one of enteric. While the reaction has been obtained as early as the fifth day from the onset of definite symptoms of illness, the clumping in an early stage of the disease is evanescent and not of much diagnostic value as it has been found in the blood of patients with other diseases. The permanent clumping of enteric is commonly not obtained till the sixth or seventh day of the fever. In a paper on the differential diagnosis of plague and enteric (Lancet, October 27th, 1900) I emphasised the importance of the general practitioner, sending at once the blood to be tested for Widal's reaction in every obscure case of pyrexia, and when a negative result is obtained and the fever

continues without a discoverable cause a second, and later, specimen should be tested. The city bacteriologist now adds to his report of the reaction that "When the result does not accord with the clinical symptoms the physician is requested to send another sample."

A third fallacy is failing to inquire into the history of the patient's previous illnesses. While the serum may cease to react three months after an attack of enteric, the reaction may persist for a longer period. In the majority of cases it disappears at the end of a year, but Widal himself has reported a case in which it was found 2b years after an attack of enteric in a dilution of 1 to 30. Its persistence may be explained by the persistence in the body of the bacillus typhosus long after an attack of enteric. Two cases are on record in which the bacilli were found six years after the primary fever (Eusche and Sultan), one case seven years after in a pure culture in an inflamed gall-bladder (Hunner), and one case fourteen and a half years after in a pure culture in pus (Von Dungern.) Hence the importance of the patient's medical history, and in those cases where we find a history of anterior enteric it would be safer not to test the blood for the Widal reaction.

The fourth and last fallacy is that while the positive reaction is rarely at fault it has been obtained in a comparatively very small number of cases that were not enteric. It has

been obtained in one case of Typhus (Caiger Lancet, June 17th, 1899), one case of septicaemia, one case of malignant endocarditis and two cases of pneumonia (Goulstonian lectures, 1900.) These rare instances may be possibly due to an anterior attack of enteric, or to faulty technique especially in the earlier days of the reaction. As illustrative of this, the case of septicaemia in which it was obtained may be taken as an example. It occurred at St. Bartholomew's hospital where 546 cases were tested for the reaction. Of these 200 cases proved themselves later by their clinical course, and some verified post mortem, not to be enteric, yet only in one case was a positive reaction obtained. This was a woman who died of septicaemia, but no bowel lesion was found. On close inquiry it was ascertained that four months before her death she had been treated at home for enteric.

A method of earlier differentiation of enteric than Widal's is described by Piorkowski (Lancet Jan. 6th, 1900). He reports 40 cases in which he was able to diagnose enteric from the third day of the disease to three days after subsidence of fever and was successful in doing so on occasions where the Widal test failed. The method consisted in making cultures from the faeces of suspected cases. His results are confirmed by other observers especially by Schutze who says that the method is of "remarkable, even decisive importance in establishing a positive diagnosis in the early stage of enteric."

Notwithstanding the earlier result, I am afraid that this method for obvious reasons will not be readily adopted in general practice.

Five other cases that simulated Plague.

These five cases occurred in the infected area during the prevalence of plague.

Acute lobar pneumonia. - On September 12th, 1900, I was called to C. Mc K. aet. 29, old clothes-dealer, residing at 29 Crown Street, E. E. He had been delirious all the previous night, and the history of the case was that the previous afternoon he had a rigor followed with an acute pain in his left side, and a short frequent cough with scanty coloured expectoration. On examination he had all the symptoms and signs of an acute pneumonia in the lower lobe of his left lung. The three points of simulation to plague in this case were that (1) he lived in the centre of the infected area and his occupation also exposed him to infection. (2) the early delirium and (3) the symptoms and signs common to simple and plague pneumonia. On close inquiry I excluded association with the other plague patients as well as the probability of infection in the district or at his calling. The delirium was accounted for by the fact that he had been drinking heavily for a week up till his illness, and

the difference in his sumptoms and physical signs will be seen by describing the first case of pneumonic plague on record which was reported by Childe in the British Medical Journal, May, 15th, 1897. Childe observed in the epidemic then raging in Bombay that pneumonia and so-called remmittent fever increased with the spread of plague, and on examining the sputa in these pneumonic cases it was found teeming with plague bacilli. The typical case described by him was that of a native who had a rigor, nausea and vomiting, severe headache and a tired aching feeling in his limbs. His temperature was 103 F. and his tongue remained clean and moist. He passed a bad night, and on the 2nd. day his temperature was 104 F. and he felt a pain in left axilla but no glandular enlargement could be made out. His sumptoms increased and he passed a restless night, and on 3rd. day he began to cough and brought up some watery seromucous fluid slightly blood-tinged, while the pain in left axilla persisted and at this spot could be heard fine crepitations of early pneumonia which were also heard just below the left clavicle, but the other parts of the lungs and all the other organs of the body, including the lymphatic glands, were normal. On the 4th day his temperature

was 104 F. his symptoms increased, and he was extremely weak and died on the 5th day.

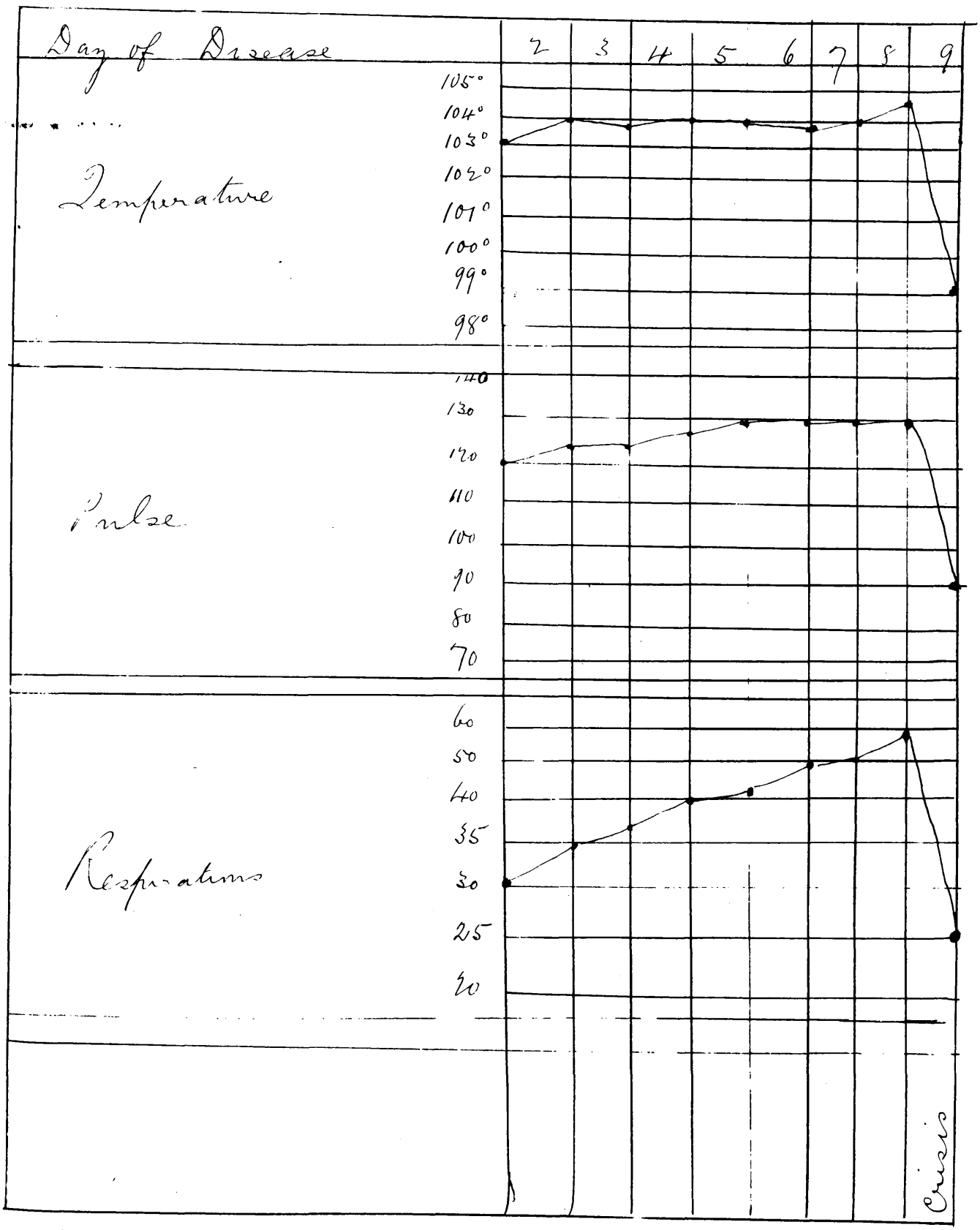
His sputum in life was full of plague bacilli. On post mortem examination it was found that his lungs showed general enlargement and oedema with sero-sanguinous frothy fluid in the bronchi but no pus, while the usual appearance of acute bronchitis was absent. Two small pneumonic patches, in second stage of the disease, were found below the apex in front of left lung and one patch the size of a walnut in same situation in right lung. There was a recent pleurisy over these pneumonic areas. The cervical, axillary and lumbar lymphatic glands were slightly enlarged also the left iliac. All the others, including the bronchial glands, looked absolutely normal.

Differential diagnosis. - The main points to be noted in the differentation of simple lobar pneumonia and plague pneumonia are (i) the onset in the plague form had the initial symptoms of plague, the severe headache, nausea and vomiting, and there were absent the localised chest pain, short and frequent cough, and urgent dyspnoea, although the latter two symptoms are sometimes present but not so constantly nor so strikingly as in simple pneumonia.

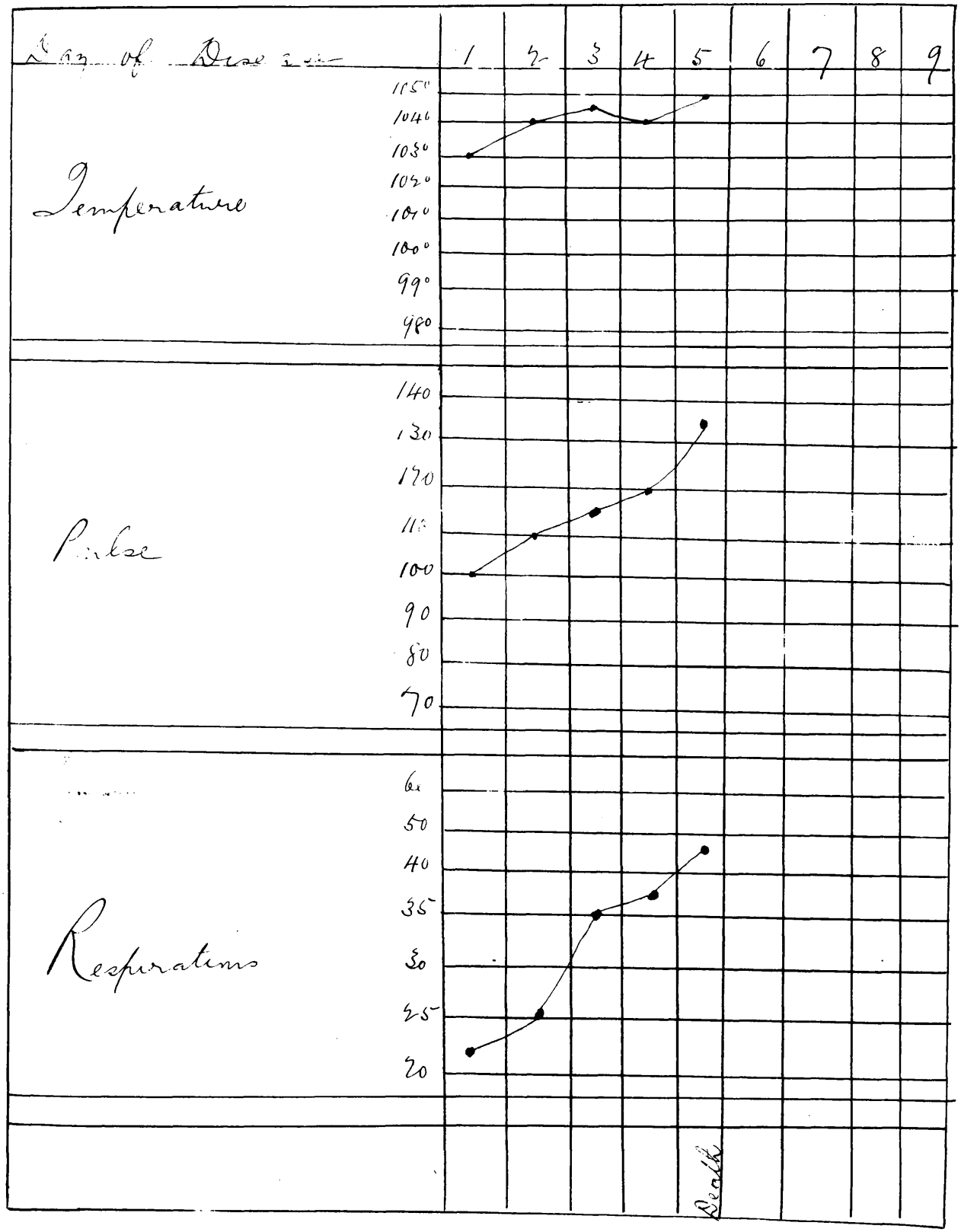
(2) The pulse-respiration ratio of simple pneumonia was absent from the plague form. This will be seen on comparing the two charts I have made out, the one of my case of simple pneumonia and the other made up from the description given by Childe of his typical case of the pneumonic plague form (3) the sputum in the plague form was not at all rusty. It was loose and free and came up with the slightest cough. It was watery looking, and more like serum than mucous, and it was slightly pink, not yellow, or rusty. (4) A very striking fact was that the patient's general condition was out of all proportion to the pulmonary disease revealed on physical examination, and (5) his sputa was teeming with plague bacilli.

Acute Lymphadenitis. - On September 18th, 1900, I was consulted by R. E. aet. 30, housewife, residing at 71 Thistle Street, E. E., concerning a painful swelling in her right axilla. She said that she felt the right axilla tender three days previous, and then a little "lump" which gradually became larger. She had no plague symptoms nor association with plague patients. Her temperature was 100 F. and on examination her right axillary glands were inflamed and enlarged. No other glandular enlargement could be made out. The common cause of inflammation of the axillary glands is the

J. Mc K. Acute Lobar Pneumonia.



Childe Pneumonic Plague.



absorption of some irritative material from wounds of the hand. or fingers, and on close examination a slight wound was detected in the pulp of her middle right finger which she remembered had resulted a few days previously from the prick of a pin while washing the floor. The bubo after fomentation was incised, and the patient became well. Two days later I was called to see her infant girl 10 months old, who had a swelling in her left buttock and another in the region of the left oblique set of glands which run parallel to Ponpart's ligament and are inflamed in affections of the penis, scrotum, perineum, anus, buttock and lower part of abdomen. On examining the swelling in the buttock a little black point was seen which on removal was found to be the distal end of a small splinter of wood that had evidently pierced the skin and cellular tissue for about an inch. The mother said that the child had began to crawl and she remembered removing two days previously a "shelf" of wood that had pierced its buttock while crawling on the floor. The splinter of wood had evidently broken and the part remaining in the skin had caused both swellings. The unusual coincidence of bubbes in two inmates of the same house in a plague street caused me to direct the attention of the sanitary plague experts to the fact, and they agreed in my diagnosis of acute

lymphadenitis with an evident cause, and did not consider it necessary to examine material from the bubboes for the presence or absence of the bacillus pestis.

Acute Parotitis. - On September 20th, 1900, J. M. aet. 25, shoemaker, residing at 210 South Wellington Street, consulted me about a swelling on the right side of his face. It came on gradually two days previously and gave rise to a feeling of tightness in his right jaw on mastication or deglutition. It was only moderately painful and while he was out of sorts he was able to follow his employment. On examination his temperature and all his organs were found to be normal. His right parotid gland was distinctly swollen, the swelling extending in front of, and below, the ear and backwards to the inner border of the sterno-mastoid muscle. The other parotid gland was normal and the only other glandular enlargement that could be made out was that of one of his left axillary glands which was about the size of a bean but not tender nor painful. He had no plague symptoms nor known association with plague cases, but as the parotid gland has been occasionally, though not commonly attacked by the plague bacillus in the Indian plague epidemics, a suspicion of plague was at first entertained but afterwards negatived by the disappearance of the

swelling in three days. The swollen axillary gland remained the same and I was of opinion that it resulted from chronic irritation from slight wounds in the pulps of his left fingers which would be caused in the act of picking up the sharp sprigs that he hammered into the soles of boots. Since then I have had under observation five other cases of mumps but in none of these cases could I detect any axillary glandular enlargement.

Gonorrhoea. - On September 23rd, 1900, I was consulted by X, a boiler-maker, residing at 132 Rose Street, S. S., concerning a painful swelling in his right groin. He said that it had appeared gradually and admitted being under treatment for a "runner" three months previously. On examination there was in his right groin in the region of the obliaue set of glands a painful tense bubo the size of a hen's egg which was the result of glandular inflammation and suppuration from absorption of the specific virus in his uretha. The glands in left groin were slightly enlarged, but not painful. There was no other glandular enlargement and no plague symptoms nor history of association with plague patients. The main points in the differentiation of plague buboes from gonorrhoeal buboes in a man are (1) history of infection.

(2) the comparatively gradual affection of the glands without any rise in temperature or any constitutional disturbance in gonorrhoea, and (3) in the latter condition the absence from the gland of the bacillus pestis.

In a woman besides the points noted above would be the fact that a bubo in the groin is rarely a sequel of gonorrhoea in a female. The primary affection is commonly a vulvo-vaginitis and the glands commonly affected are the vulvo-vaginal or the glands of Bartholin. In my limited experience in cases where I had reason to suspect gonorrhoea in a female, I have never met with the inguinal glands affected nor do I find any mention of their affection in the ordinary surgical text-books or in the text-books on diseases of women. Hence I believe that they are rarely involved in an uncomplicated female gonorrhoea. Apart from the situation of the buboes, in my plague case of Rosina Murphy, a diagnosis of gonorrhoea should have been excluded by the history of sudden onset with nausea, vomiting, delirium, temperature 104 F. early appearance of buboes, and the fact of her living in the infected area and having direct association with plague contacts.

Soft chancre. - On October 5th, 1900, T 23, a joiner residing at 32 Rutherglen Road, S. S., consulted me about little hard painful

"lumps" in each groin. On examination they were found to be inflamed and enlarged inguinal glands and on drawing back the foreskin a typical soft sore was seen on the glans penis near the fraenum. He had no plague symptoms nor association with plague patients and the main points in the differential diagnosis of plague buboes from the buboes of soft chancre are the same that differentiate the former from gonorrhoeal buboes. In soft chancre in females inguinal buboes are not uncommon.

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