Japanese textbook on plague, with, Pathogenic horticulture : in two parts.

Price
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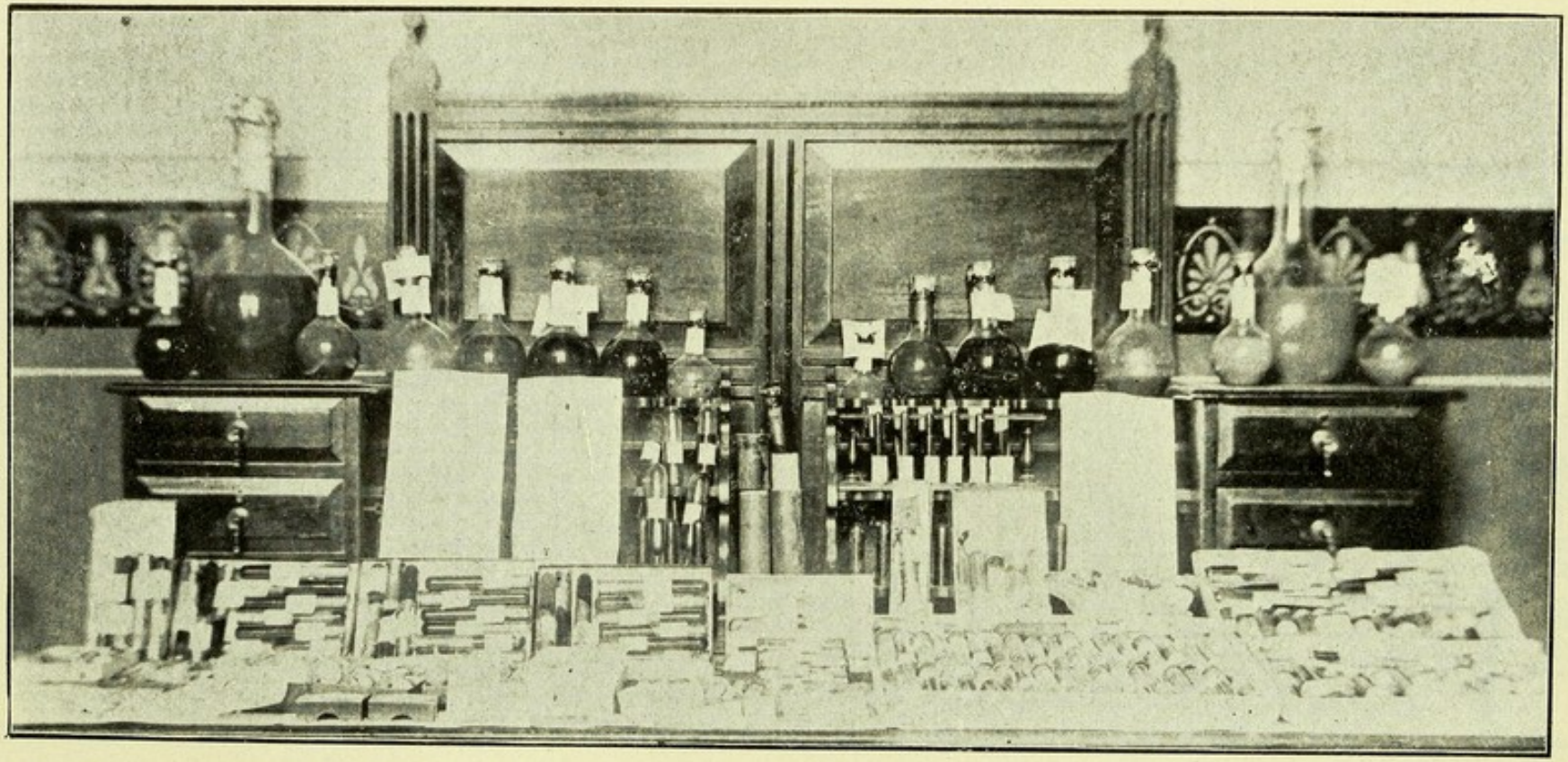
BACTERIOLOGICAL AND HORTICULTURAL EXHIBITION OF HEALTH AND DISEASE.

THE PRINCIPAL MICROBES.



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Manures, Seeds, Vegetable Infusions, Vaccines, Antidotes.

I.

JAPANESE TEXT-BOOK

ON

PLAGUE

WITH

PATHOGENIC HORTICULTURE

IN TWO PARTS.

AUTHORISED EDITION.

II.

A TEXT-BOOK
ON
PLAGUE

BY

DR. TOHIU ISHIGAMI,
Superintendent Bacteriological Institute, Osaka, Japan; formerly Assistant Bacteriologist to Prof. Kitasato.

Revised by Prof. Shibasaburo Kitasato,
Tokyo, Japan.

TRANSLATED, ENLARGED, AND ILLUSTRATED
WITH
PATHOGENIC HORTICULTURE

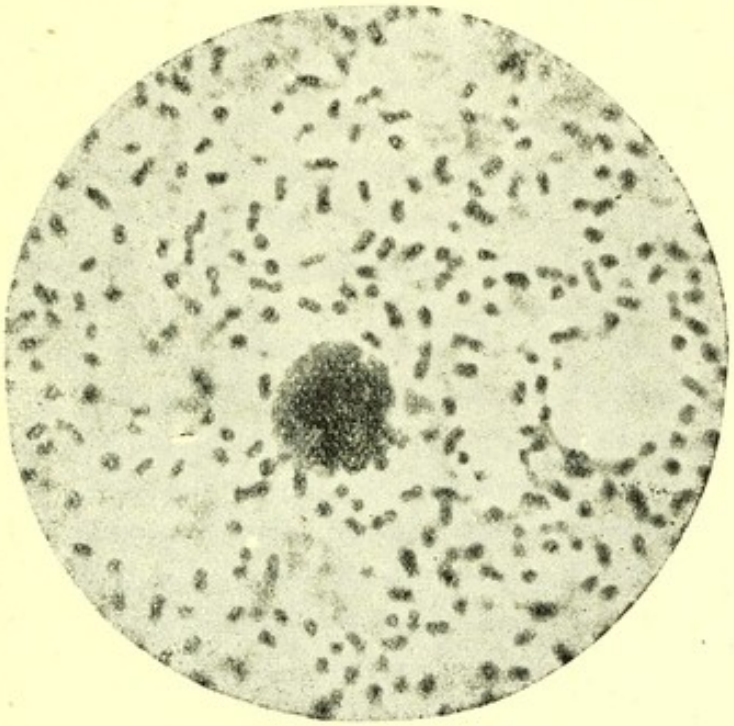
BY

DONALD MACDONALD, M.B.C.M. (Glasg.)
Late Consulting Bacteriologist to the S. Aust. Govt.; Medical Officer-in-Charge of Infectious and Isolation Wards, Bacteriologist, Medical Superintendent (A.), Honorary Pathologist (A.), Resident Medical Officer Adelaide Government Hospital, S. Aust., &c.

WITH ONE HUNDRED AND FIFTY-TWO ILLUSTRATIONS
AND THREE CHROMO-LITHOGRAPHIC PLATES.

ADELAIDE:
Printed by Vardon & Pritchard, Gresham Street.
1905.

THE PLAGUE BACILLUS.



Bacillus Pestis Bubonicæ from a bubo of a Chinese case, Hong Kong. 1896. Cover glass specimen. Fuchsin. X 1000.

[*Kelly.*

AUTHOR'S PREFACE TO THE JAPANESE EDITION.

Last November, when bubonic plague was tending very much to become epidemic in Kobe and Osaka, medical practitioners were not infrequently in the dark as to its proper clinical treatment, since no book had ever been written on the subject.

Having acquired some knowledge of it when I was in Hong Kong in 1894 with Professor Kitasato and Professor Aoyama, I, after hurriedly consulting a few reports on the subject, compiled and published this book which has been revised by my revered teacher, Professor Kitasato.

Since its outbreak at that time, the plague has spread enormously, scourging both places as a malignant epidemic. Even the pneumonic type, hitherto considered as rare, was found in nearly a score of cases. My professional assistance being required by the Osaka Temporary Epidemic Inspection Bureau, I had the opportunity of making observations on several different cases of the disease. These observations are included in this second edition.

AUTHOR.

Osaka, 11th February, 1900.

PREFACE TO THE AUTHORISED TRANSLATION.

Osaka, November 25th, 1901.

My Dear Sir—

Your favor of August 20th duly received. You ask my consent to publishing in English my work on "The Plague," the revised second edition. I deem it a great honor that a physician of this country should be asked to publish his book in your more advanced country. I give my hearty consent to your proposal.

However, please bear in mind that the book was published at the time of the last outbreak of Plague here, in the spur of the moment, so it is not a very complete work. There are some errata too. I wish very much to add a little to the book, and also to make corrections on the results of my subsequent studies on the subject, but, as I am very busy at present, I am unable to do so.

Believe me, yours truly,
(Signed) TOHIU ISHIGAMI.

Dr. D. MacDonald,
Medical Superintendent
Adelaide Government Hospital.

PREFACE TO THE ENGLISH EDITION.

To the Japanese belongs the honor of discovering the Plague Bacillus. It will suffice to mention Prof. Kitasato's name in commending Dr. Ishigami's excellent work on Plague.

The first part of this book was written by Dr. Tohiu Ishigami to meet a growing demand for a book on Plague. That it has not failed in its object is shown by the fact of it having already reached a second revised edition, which is again out of print. I am indebted to Dr. Constance Frost, New Zealand, for help with some proofs. I must ask the indulgence of the reader for any errata or deficiencies of this translation. I trust that they will be found to be neither numerous nor important. Annotations have been inserted for any alterations which I have deemed advisable in the original text.

The author wished very much to add further to the book, but has not been able to do so for want of time, so that I have added Part II. in completion of the work.

It is hoped that this edition will as adequately supply the want of a work of its kind which has long been felt in this country as well as in Japan. Owing to the comprehensive importance of plague, the illustrative enlargement of the work requires no further explanation. I take this opportunity of thanking Dr. J. F. Nelly, Melbourne, Victoria, Australia, for the microphotos of my specimens in this work.

DONALD MACDONALD, M.B., C.M. (Glasg).

Addington House,
86, West Terrace, Adelaide,
South Australia.
December, 1905.

Text of Plate I.

The King of Passion plants, clothed in the purple of the Cæsars.

"We are slumbering poppies,
Lords of Lethe downs,
Some awake, and some asleep,
Sleeping in our crowns,
What perchance our dreams may know,
Let our serious beauty show."

—*Leigh Hunt.*

The King of Pathogenic plants (M. P. aureus) as exquisite roseus Its botanical preservation in elementary biological reagent with good spectroscopic relief (1898). After original oil painting from a specimen of an embryonic metamorphotic microbe plant.

Bacteriaceæ,
Chromaceæ,
Exanthemata,
Rosaceæ.

Formulary XIV.

$$AGJSY^{fcs}_{s\beta}NaCO_3.$$

100 c.c.

A = ascetic 35 c.c.
G = glycerine m iv.
J = jequirity 35 c.c
S = glucose grms. ii.
Y = yelk 30 c.c.

$s\beta$ = serum bouillon ad 100 cc.
f = fluorescein i% m.vi
c = cochineal sol. 4 c.c.
s = saffranine i% m.xii.
$NaCO_3$, 10%. 1 c.c.

Filter, sterilise.

Text of Plate I.

The King of Passion plants, clothed in the purple of the Cæsars.

"We are slumbering poppies,
Lords of Lethe downs,
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Bacteriaceæ,
Chromaceæ,
Exanthemata,
Rosaceæ.

PLATE I.

PATHOGENIC HORTICULTURE SERIES.

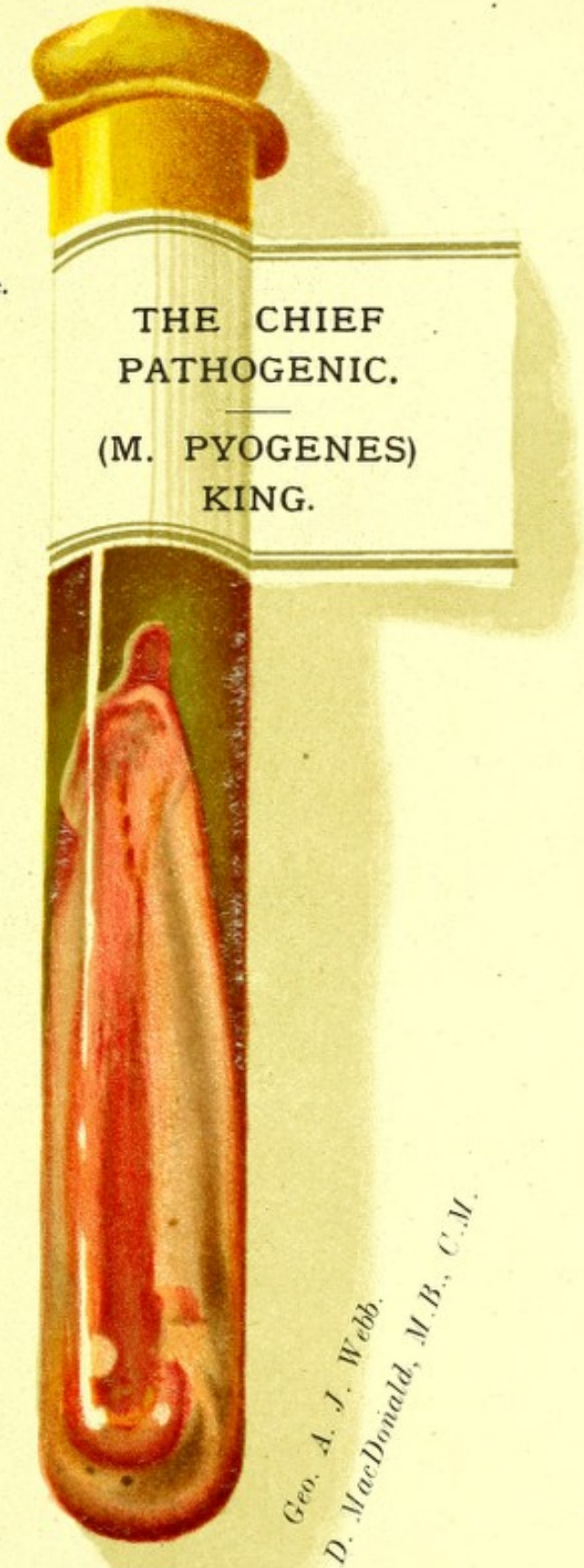
Kaleidoscopic Chromogenesis.

Bacterioscopy.

Biological Reagent C.

Papilionaceous Leguminosæ.

Pyo-phanerophyte.



Geo. A. J. Webb.
D. MacDonald, M.B., C.M.

MICROBE PLANT.

Vardon & Pritchard,
Lithographers,
Gresham Street, Adelaide.

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BUBONIC PLAGUE.

PART I.

THE JAPANESE PLAGUE TEXT.

CHAPTER I.

The Definition of Plague.

In the time of Galen, when the distinction of an epidemic disease was so obscure, whenever an acute epidemic fever prevailed which swept away many lives, it was generally called plague, no matter what variety that disease might be, nor what was its character. Thus, at that time the term plague not only included the present particular plague, but it embodied such as smallpox, measles, diphtheria, and other kindred febrile diseases.

Such a broad appellation was generally used until the eighteenth century, when the progress of medical science and experience had distinguished first, smallpox from this name, and then measles and diphtheria ; so much so that at the present time it has finally become only applicable to a narrow circle of the particular pest. The plague is otherwise called the "Black Death." Before the fourteenth century, when this disease prevailed greatly in Europe, there was no such name as "Black Death." In this period a main feature of the disease was hæmorrhagic ecchymosis of the skin and mucous membranes. The hæmorrhage was similar in nature to that in cases of smallpox, and death was rather sudden, the surface and skin of the whole body presenting a dark purple colour. Hence, it is supposed, the name.

In Great Britain this disease is called bubonic plague. In perusing reports of the Colonial Health Statistics this name

was being continually used. In France it is called Oriental Pest, no doubt on account of the outbreak originating from the Eastern Asia Minor regions. In China, what the Chinese doctors call " Wăn yik," is this disease. In the neighborhood of Hongkong and Canton they call it " Bew shair jēn," denoting snake-shape. Perhaps the phrase signifies the features of the body affected. The subcutaneous veins and lymphatics often become inflamed, and present red tortuous lines. In Formosa it is said that before an outbreak of plague occurs there is sure to be observed a great mortality amongst domestic rats, " Bī she bēng," so called, rat plague.

To analyse plague in accordance with the present scope of medical science, this disease is originally an acute epidemic fever caused by an invasion of the plague bacillus. It principally attacks or invades the surface, as well as the subcutaneous veins and lymphatics. It can also originate in the intestines, lungs, and epidermal structures, producing in each case an original, special, or essential feature of the disease. Finally, it becomes a blood destroying disease, a septicæmia.*

CHAPTER II.

Summarised History of the Plague Scourge.

According to the old record of the Physician Rufus of Ephesus, which was discovered at the beginning of this century, it appears that plague was prevalent before the Christian era, especially about the second century before Christ. The name of this disease also occurs in the writings of Hippocrates. It is said that it was rife in the camps at the time of the campaigns of Peloponnesus and Athens. In these remote times the area where this disease was prevalent was principally restricted to the regions of Asia Minor, Egypt, China, and India, not having then yet invaded Europe.

It was after this, in A.D. 542, in the time of Justinian, Roman Emperor of the East, that the epidemic invaded Europe for the first time. It attacked it from various quarters, one from lower Egypt, one via the northern coast of Africa, one through Palestine and Syria. It then devastated the whole of Europe for a period of fifty years, during which time innumerable lives were decimated. This was the first time it ever prevailed in Europe.

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* Chinese plague is largely septicæmic.

The second time was in the fourteenth century. Opinions differ as to the original starting point of the disease, some attributing it to China, and some considering it to have originated in the neighborhood of the River Ganges. It is difficult to ascertain its exact origin. Still, it is quite evident that the disease was imported from the East. It reached Turkey overland from Western Asia, travelling by way of Asia Minor through the traffic and intercourse between those countries. It came to Italy by sea from the North African coast. Thence the epidemic spread all over Europe, even Greenland in the far north not escaping. It is said that its virulence at this period was so great that the people attributed it to the influence of evil stars, or reckoned it as a direct punishment from God; all awaited their end with clasped hands; and corpses were to be seen scattered about everywhere.

According to Hecker, the victims who succumbed to the scourge were considerably over twenty-five millions or almost one quarter of the entire population of Europe. The name Black Death was given to the disease at this time. It was during the period of 1346-1383 that the epidemic made the greatest ravages, the neighborhood of the Danube in Austria, Upper Italy, England, East Germany, Holland, and Russia being devastated most terribly. It raged and continued into the fifteenth century, but we do not see a trace of its cessation. Its force, however, appeared to be spent in the sixteenth century, though by no means was it extinct. The last half of the seventeenth century (1650) saw the entire disappearance of the epidemic in England. In Sweden and Denmark (1657), West Germany (1667), North France and Switzerland (1668), and East Germany, it gradually became extinct.

Since then until now, Europe has been spared entirely a revisitation of this dreadful scourge; on which account European doctors, considering the disease a medieval one, had almost entirely omitted it from their medical works, giving only a mere outline of its terrible nature in the history of medicine, just sufficient to exercise our historical imagination.

Although the disease ceased to prevail in Europe, yet the eighteenth century saw a small outbreak in Asia Minor and Asiatic Turkey which spread into Russia and part of Prussia, and from Prague into part of Austria. But the epidemic was of a slight nature. In the nineteenth century, about 1878-1879, there occurred small outbreaks at Odessa

on the right bank of the Volga, in the Balkan Peninsula and in Astrakhan. Thither medical men were sent from the European Universities to investigate it.

In India and Southern China there seems year after year to have been no cessation in the prevalence of the plague.

Lacking any authentic records, there is no means of ascertaining its extent and details. That it broke out in 1815 in India and Hindustan is the only fact recorded. It is a positive fact that plague has prevailed in Southern China annually from ancient times. What the Chinese doctors term " Wăn yik " is the plague.

According to treatises on geographical diseases as well as the report of the Hong Kong Government, it is undoubted that this disease is endemic in the Province of Yunnan in Southern China. The disease prevails there annually. The Pakhoi outbreak of 1894 (Dr. Dunn's report) most strongly attracted public attention to the prevalence of the disease in China.

The epidemic was rather a malignant one, but it was of short duration. In 1893 it broke out in Canton, and carried off 140,000 victims. It is not very certain how Canton originally became infected, but most probably it happened, however, from the Yunnan Province through the constant comunication which is kept up between the river and these places. The infection was carried to Hong Kong from Canton through the boat traffic. Hence arose that most notable epidemic which prevailed there in 1894. (In this year the Japanese Government sent out Professors Kitasato and Aoyama.) The infection then spread so far as Pakhoi and Amoy.

The plague epidemic of Formosa in 1896 was the first that was ever known to the outside world, but, according to the missionaries there, it had been well known to the Formosans from olden time. The infection was no doubt imported from Canton to Amoy through the boat traffic. Malignancy is not a feature of the Formosan plague ; but it breaks out annually in several places.

In 1899 it broke out in Newchwang* in North China, and is not yet extinct. The same year, it is reported, witnessed a slight outbreak in Oporto. Excepting the Formosan Colony, Japan was free from plague until 1899, in which year it broke out in Kobe and Osaka for the first time in the annals of Japanese history. The primary source of infection has

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* Plague was still epidemic in 1903 in Newchwang.

not been clearly investigated. The presumption is, however, that it was imported from Newchwang. Knowing of its prevalence in Bombay, the infection might have been imported from India.

(At the end of the Japanese war in 1895 cholera was rife in the Gulf of Pechili, and its importation likewise set up a similar epidemic in Japan. In 1896 and 1897, Bī shc beng, "the rat epidemic," broke out in Foochow, China, when in each year about 40,000 died. 1900 saw epidemics in New Caledonia, Southern Australia, and in Glasgow, Scotland.—D. McD.)

CHAPTER III.

Endemic Regions of Plague.

An investigation into the history of the plague scourge enables us to say that plague has its own original or endemic areas. Wherever it resides it dominates over those regions. As cholera invades other countries from its headquarters, Bengal plague also seems disseminated by some means through communication after an elapse of a fixed number of years. On enquiring where its native quarters might be, we find that up to the present time investigation indicates five endemic areas.

1. The foot of the Himalaya.
2. Asir, in Arabia.
3. Mesopotamia in Asia Minor.
4. Yunnan and Thibet in North China.
5. Kisiba in Western Africa and the neighborhood of Uganda.

These last places were discovered by Robert Koch in *1898 during his travels in Africa.

6. South Africa.
7. Southern Australian shores (sporadic).

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* Plague has since made its appearance in various other places, such as Mauritius, San Francisco, and South America; so that two more areas are added.

CHAPTER IV.

The Biology of the Plague Bacillus.

In 1879, when there was an outbreak of plague in the neighbourhood of the Black Sea Coast, Virchow had already declared his opinion that the disease was of microbic origin. In the same year, at the time of the Astrakhan outbreak, Messrs. Zamelbrot and Ifiwald, in examining the blood of the patients, discovered in the corpuscles and in the serum small round bodies having a highly refractive power. Thinking, however, that these were chylous or lymphatic globules, they did not investigate further; so that their discovery did not throw any light on the etiology of the plague.

Fig. 1.
Ga, colonies of the plague bacillus, from blood of plague case, Hong Kong, 1896, Glycerine Agar.
[*Ernest Gall.*

When the epidemic broke out in Hong Kong in 1894 the Japanese Government sent thither Professors Kitasato and Aoyama to investigate the disease. They commenced their investigations on the 14th of June, 1894. Professor Aoyama was to undertake the diagnosis and clinical part of the investigations, and Professor Kitasato the bacteriological. I assisted the latter in his researches. On the first day Professor Kitasato examined the blood of the heart, spleen, liver, lungs, and swollen lymphatic glands of a corpse dead from plague. He found in them a considerable number of strangely original rod-shaped bacilli. On the same day microscopic examinations of the blood obtained from the finger tips of a critical patient revealed the existence in it of similar bacilli.

Subsequent bacteriological examinations of several patients and corpses demonstrated the presence of the same bacillus in every case. But those existing in the blood were regular in shape and smaller, whilst those in buboes or swollen glands and other organs were irregular in shape, and somewhat larger. Both were found to exhibit bipolar staining, the middle part refusing to retain the dye. The cultures made from both varieties were similar in the shape and appearance of their growth. Therefore, Professor Kitasato was of the opinion that those which existed in the glands and

other organs were pleomorphic involution forms, and, following a fundamental principle of pathology, attached greater importance to those existing in the blood. Hence he conducted a series of cultivations and experiments on animals, the results of which were made public as explaining the cause of the disease.

FIG. 2.

S ^{1}a, culture of the plague bacillus, from spleen of a plague case, post-mortem specimen, stab culture; 37°C., four days old, in Litmus Glucose Agar. [*Ernest Gall.*

A few days after this discovery of Professor Kitasato's, Dr. Yersin of France, sent out from the Pasteur Institute, arrived at Hong Kong via Saigon. Conducting his investigations independently of Professor Kitasato, he studied the bacilli chiefly as met with in the glands and organs, which we had considered to be involution forms.

He reported the results of his investigations to the Pasteur Institute as the cause of the disease. Since then, Professor Aoyama has made a special microscopic study of specimens of organs and glands from the plague corpses which have been brought from Hong Kong. He gave out as the result that the bacillus which exists in the glands, and which is decolourised by Gram's method is an entirely different species to that which exists in the blood, and which is not decolourised by Gram's method.

(Imperial University Report, vol. III., No. 2.)

Following upon this, Mr. Zetnur, having studied cultivations of the plague bacillus obtained from Dr. Metschnikoff, had reported that they correspond with the bacilli which are reported by Yersin to exist in the buboes.

In July, 1896, Mr. Yaowaka Murakami sent from Formosa to the Army Medical College in Tokyo a pure culture obtained from an inguinal bubo of a plague corpse in Formosa. Professor Okada, one of the staff of the College, made a careful investigation of the culture, and reported that it corresponded with the bacillus which Yersin found in the buboes. Since then, Dr. Wilm, Dr. Haffkine in Bombay, Dr. Abel in China, and Hankin of England have all studied the plague bacillus. They are all of the opinion that this bacillus which exists in the buboes is the etiological element of the disease. Professor Ogata, who was sent in 1896 to Formosa by the Tokyo

Imperial University, also considers that this bacillus which exists in the buboes is the true cause of the disease, and he declares that it will not be found in the blood unless in the last stage when septicæmia sets in. Professor Yamakime, one of the research party, likewise analysing the disease, is of the opinion that the bacillus which can be demonstrated in the lymphatics is the primary cause of the disease.

FIG. 3.

Na, colonial culture of the plague bacillus, from the spleen of a plague case, post-mortem specimen; 37° C., a week old, in Nutrient Agar.

[*Ernest Gall.*

Thus, since the bacillus that exists in the buboes was considered to be the pathogenic element of the disease by several investigators, it naturally became our duty and responsibility to fully pursue our former incomplete investigations, so as to definitely determine one way or the other its pathogenesis.

During the unfortunate outbreak, Professor Kitasato at once came over to Kobe to prosecute further studies. His investigations proved no doubt that the bacillus of bubonic plague is identical with the Yersin bacillus which exists in the buboes, and which we had considered to be involution forms during our former investigations. The so-called plague bacillus that is not decolourised by Gram's method assumes, as it were, a second nature during the septicæmia stage of the disease. It first invades the blood as the Yersin bacillus. I believe Professor Kitasato will later on make public the results of this investigation. Later on I will fully describe the Kitasato bacillus and my investigations. The plague bacillus is a rod-shaped bacillus. It possesses the property of variation in size and shape. Its shortest length is 1μ, whilst some of them are four or five times longer. Those especially in old culture media grow to a considerable length. They make one doubt in glancing at them whether they are not some other kind of bacilli. (Vide microphoto figs., Part II.)

Sometimes it metamorphoses, becoming small and thick. It presents the shape and features of a yeast cell. When cultivated on Agar media at 37° C. it grows in chains. Not only so, but if cultivated in bouillon its streptococcal appearances are very conspicuous. However, these streptococcal appearances are seldom in straight lines, but almost always present an acute angle. Some of them are spindle-shaped, the middle

portion being swollen. Those in old culture media or in unsuitable soil resemble algæ or spheres, being egg-shaped. They are often confounded with very fine yeast cells. It stains at both ends with the aniline dyes. There is a tendency on the part of this bacillus to retain, though weakly, the original dye when adopting Gram's method, and unless the solutions are used when freshly made, the two extremities of the bacilli will still retain a little of the original dye. Yet, on the whole, it is decolourised by Gram's method. In appearance it very much resembles the chicken cholera bacillus (Abel).

This bacillus seldom has a capsule.* It never forms spores, no matter what media it is cultivated on, nor what temperature it is kept at.

FIG. 4.

Sγ, colonies of the plague bacillus at horticultural temperature, two months' old, in Glucose Gelatine. [*Ernest Gall.*

On Gelatine media at 22° C. colonies develop in from 24 to 48 hours. They are pure white or yellowish white in colour, and have no tendency to coalesce. They are generally the size of a pin's head. If these colonies are examined under the microscope they first present an ash-white colour, but later on they are white. Their margins are sharp and regular. As the colony develops it becomes granular in appearance. This bacillus does not liquefy the gelatine culture media. In Gelatine stab cultures it produces a dull yellowish white growth along the stab, and on the surface around the puncture it forms a flat investing membranous growth.

On Agar-Agar surface culture media it produces dew drop-ike colonies which, if incubated at blood heat, grow rapidly, and in 48 hours they attain to the size of a small lens-shaped bean. If transplanted often on Agar-Agar slant culture media the colonies finally become of a gelatinous consistence No film is produced in the water of condensation. Its growth on Glycerine Agar culture media is not so fertile as on Peptone Agar. It grows much better on Agar media than on Blood Serum. On potato at 37° C. it grows sparsely as dull ash-white colonies. It grows little on boiled turnips in petri plates (vide Figs. 1-3).

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* Some investigators have demonstrated flagella.

The culture in Bouillon is most characteristic. It does not cloud the liquid, and is like streptococcus pyogenes in this respect, but forms a fine sediment on the walls of the tube, and on several places at the bottom. The Bouillon culture, when shaken, becomes clouded, but it soon clears up again.

It grows well in Alkaline Bouillon to which 2% Peptone and 1 to 2% Gelatine are added. It grows sparsely in milk, which it does not coagulate. The optimum temperature is 37° C. for cultivation, but from 22° C. to 24° C. the growth of the bacillus does not diminish. It grows very slowly at 15° C., but after three or four days colonies develop. Yet, as low as 10° C. to 8° C. colonies can still be observed after six days. This bacillus is both ærobic and anærobic, but if oxygen is excluded the growth is rather slow.

It grows well in culture media containing glucose, but there is no gas formation. There is no production of indol, either in Bouillon or Peptone water.

In neutral Litmus media, after 24 hours, an acid reaction rechanges it into red. The red colour will be observed persisting unaltered after three weeks. (See Fig. 2, p. 7.)

There is no production of a nauseous odour. The bacillus is not chromogenic. It still grows well in an acid media, such as 100 c.c. requiring as much as 10·5 c.c. of a 1% standard alkaline solution to neutralise it.

Cultures kept for 5 or 6 weeks do not perish.

This bacillus is virulently pathogenic. Excepting pigeons and fowls, it is pathogenic to small animals, producing death by septicæmia. According to Dr. Abel's experiments in China.—

Marmots inoculated subcutaneously died in eight days.
Marmots inoculated in abdomen died in three days.
House rats } inoculated hypodermically died in four
Field rats } days.

If plague bacilli are present in the bubo of a patient, and if these are injected directly into the abdomen of Nankin rats (white tame rats) the latter die within six to twenty-four hours. If they are inoculated hypodermically they die in from one to four days.

We have observed animals become infected when fed upon food mixed with the bacilli.

Nankin rats, house rats, marmots, and tame rabbits are very susceptible to infection by this bacillus.

Around the inoculation there is œdema to a limited extent with lymphangeitis.

In 20 hours the animal's hair stands on end. They lie down on one side. Emaciation and septicæmia set in, and the animal dies. If the bacillus is inoculated from marmot to marmot its virulence is intensified. It likewise increases its virulence on its transit through tame rabbits ; but when so treated it is less virulent to Nankin rats. In serial cultures its virulence diminishes, although its vegetation increases.

CHAPTER V.

The Resisting Power of the Plague Bacillus.

The resistance of the bacillus against direct sunlight and scientific chemicals is as follows :—

When material from the bubonic swelling of a plague case is dried on a cover glass at 28° C. to 30° C., and is kept for 36 hours, it will still retain its fertility, but after four days it is sterile.

Drying pure cultures on cover glasses kept at 29° C. to 31° C. kills them after four and a half days (Wilm).

A pure culture spread on the surface of a cover glass and kept in the incubator dies in three hours.

If dried in a room which partially excludes the light it will live from six to nine days.

In drying it is killed quicker by a high than a low temperature. Those spread on a piece of glass or metallic substance perish sooner than those on a piece of cloth, thread, or reel of cotton (Abel).

An investigation has been made into the skins and hides of plague infected districts. When the bacilli are diffused over them it is found that they live for ten days (Abel). Their importation ought to be prohibited.

Referring to the resisting power of this bacillus against sunlight investigation demonstrates as follows :—

The bacilli die after one hour's exposure to sunlight when diffused on the surface of a cover glass. But when kept in a dark room as a control they will not perish even after 24 hours.

An Agar-Agar culture which was thickly spread over a glass plate died after four hours' exposure to the sun (Abel). In a tropical colony such as Hong Kong it will perish after three or four hours' exposure to the sun (Prof. Kitasato).

The resisting power of the bacillus against dry heat :—

Those spread on a cover glass die in one minute at a tem-

perature of 100° C. They are not killed by heating to 75° C. for half an hour nor at 50° C. for an hour.

The research on its resisting power against moist heat is as follows :—

Degrees of Temperature at which it is Killed.	Kitasato	Yersin.	Wilm.	Abel.	Russian Commission. (Toptchiff.)		Bombay Plague Committee.
					Capillary tube.	Culture tube.	
100° C.	23 min	..	10 min.	1 min.			..
80° C.	30 min.	..	20 min	5 min.	..	..	5 minutes
70° C.	..	..	..	10 min.	..	..	10 minutes
60° C.	..	..	..	over 10 min	..	..	10 minutes
58° C.	..	1 hour	1 hour	..	..	8 min.	..
55° C.	..	..	..	..	..	..	10 minutes
54° C.	..	..	..	..	15 min.	30 min.	..
50° C.	..	..	..	1 hour	2 hours	4 hours	..

Those cultivated in Agar, potato, milk, and other culture media if kept in the steam sterilizer perish positively in five minutes.

Concerning research on the resisting power of this bacillus against germicidal chemicals there are as yet not many to record :—

1. Carbolic Acid.

If carbolic acid is added to a Bouillon culture in the proportions of $\frac{1}{2}$ to $\frac{3}{4}$% after an hour has elapsed it still has the power of growth, but it is killed in two hours. It is killed in one hour if, when added, the proportion is 1% (Professor Kitasato).

They are killed in five minutes with a 5% solution of carbolic acid when silk threads are saturated with the bacilli (Wilm).

An Agar culture is killed in 48 hours in 1% carbolic acid. When diffused on a cover glass they die in two hours. An Agar culture died in ten minutes in a 5% carbolic solution, those on a cover glass in five minutes (Abel).

2. Carbol-Sulphuric Acid.

This is a mixture consisting of equal parts of crude carbolic and sulphuric acids.

A 1% solution of this mixture kills an Agar culture in ten minutes.

Those present in pus from bubonic swellings are killed in two minutes.

A 0·2% solution kills an Agar culture in thirty minutes (Abel).

3. Lysol.

A 1% solution of this liquid has the power of killing an Agar culture in thirty minutes. The bacilli of bubonic pus dried on cover glasses are killed in five minutes. A 0·2% solution of this liquid has the property of destroying an Agar culture in a little over 24 minutes (Abel).

4. Creolin.

A 5% solution destroys the plague bacillus in five minutes when silk threads are saturated with them.

5. Chlorine.

A 0·1% solution destroys an Agar culture in ten minutes, and material from bubonic swellings smeared on a cover glass in two minutes. An Agar culture is killed within an hour with a 0·02% solution. The addition of table salt to this solution does not enhance its germicidal power.

The growth of the bacillus is still perceptible in Bouillon to which this solution is added in the proportion of 1:100,000, but it cannot be observed if the proportion is 1:50,000.

6. Lime Water (Calcium Hydrate).

If a 28% solution is added to a Bouillon culture in the proportion of 1% it is killed in two hours. If the slaked lime solution is sprinkled over an Agar culture it looses its propagating power (Abel). Silk threads saturated with bacilli become sterile in ten minutes with a 5% solution of lime water (Wilm).

7. Chloride of Lime.

A 1% solution destroys an Agar culture in thirty minutes. Material from bubonic swellings is destroyed when smeared on cover glasses in five minutes. Its 0·2% solution kills an Agar culture in two hours (Abel).

8. Formalin.

The 44% solution of "Formaldehyde" (1:1% Formalin) destroys a Bouillon culture in three hours. After three hours in a 22% solution it still possesses its fertility (Abel).

9. Soda.

This soda is used for curing animal skins, hence the investigations (Abel).

A 1% solution destroys a Bouillon culture in an hour. After the lapse of two hours the bacilli can still be observed alive in a 0·1% solution. An Agar culture, after having been in a 0·5% solution for 25 hours, still has the power of growth.

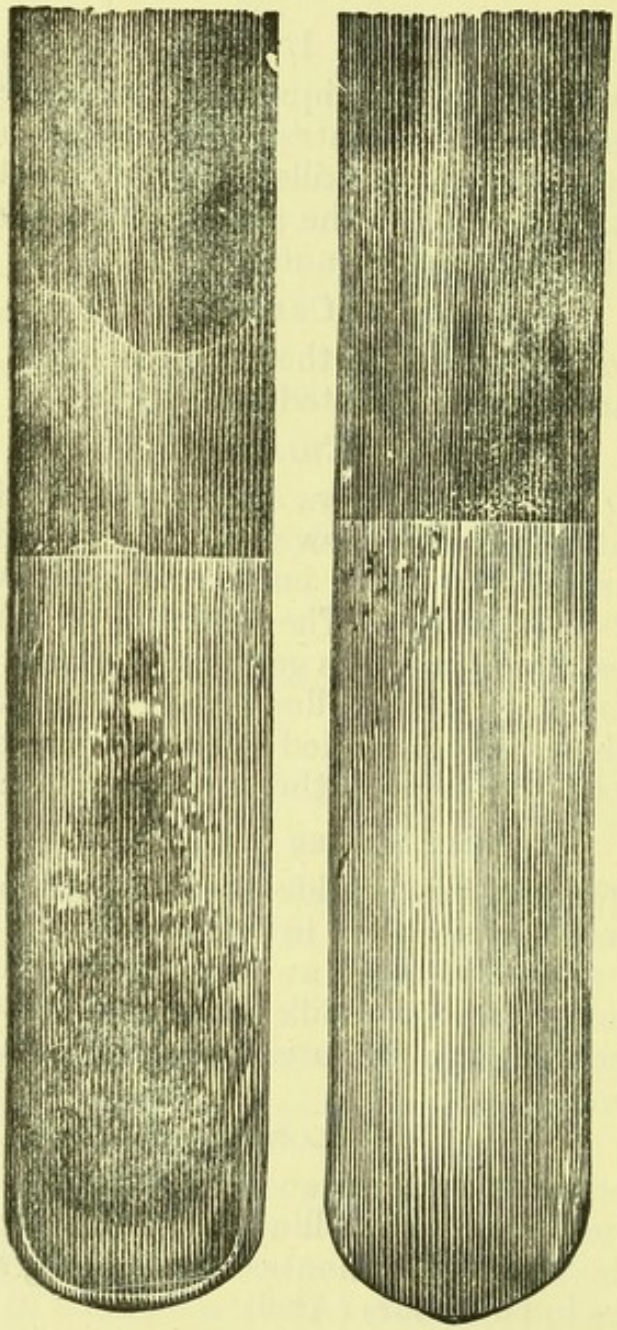


FIG. 5. FIG. 6.

FIG. 5.*—The germicidal value of solyptol vapor; Nutrient Agar cultures of the typhoid bacillus control. [*Gall.*

FIG. 6.—The germicidal value of solyptol vapor on surface cultures (figs. 5 and 6). A 2% solution of solyptol is germicidal, disinfectant, and antiseptic, and this quantitative analysis of mine has been confirmed. (See figs. 7-11.) [*Gall.*

* I am indebted to F. J. Faulding & Co., Adelaide, for permission to insert these illustrations of my work for them on their "Solyptol," an Australian germicide (D.McD).

The tables of the germicidal chemicals of the plague bacillus investigated by Dr. Schultz and others are as follows :—

Germicidal Reagents.	Bouillon Culture.		Piece of Paper.	
Mercuial solution	1:1000	2 minutes		
Alkaline "	1:1000	2 "	1:1000	2 minutes
Phenol	1:2000	2 "	1:2000	30 "
Chloro-phenol	1:50	2 "	1:50	5 "
Formic aldehyde	1:200	2 "	1:200	60 "
Slaked lime	1:50	2 "	1:50	60 "
Chloride of lime	1:100	30 "	1:100	30 "
Alkaline argentic solution	50:1000	30 "	50:1000	10 "
Sulphuric acid	100:1000	10 "	100:100	2 "
Nitric acid	100:1000	10 "	100:100	2 "

Germicidal Reagents.	Proportion of Germicide.	Germicidal Equivalent.
Mercurial solution	1:1000	1:500,000
Alkaline "	1:2000	1:50,000
Phenol "	1:50	1:400
Chloro-phenol	1:200	1:5,000
Formaldehyde	1:50	1:25,000

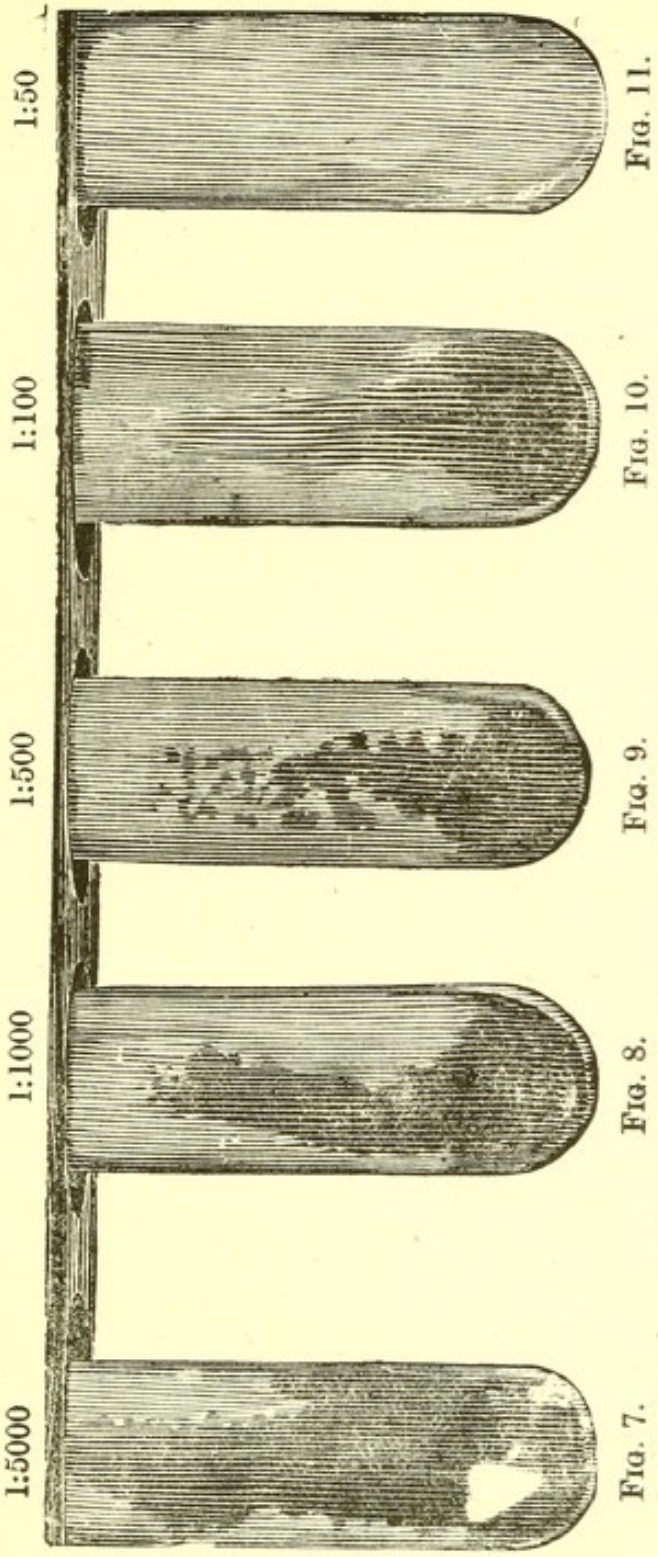
In order to ascertain the germicidal power in a room, experiments were conducted with Bouillon cultures under different conditions in a closely shut up room of 3,177 cubic metres. The strength of the chemicals then employed were : Mercurial solution, 1:2000 (by adding alkaline solution 2000 parts). Phenol, 5%. Chloro-phenol, 1·5%.

Sulphur, phosphorus, and formalin were distilled by Trillart's process.

The time-limit employed was from one and a half to 24 hours. The results are as on the following table :—

Chemicals.	Wrapped in an envelope.	Placed in petri plates.	Covered over.	Hanging down exposed.
Mercurial solution	+ + + +	— — — — — — — —	+ + .. ? ?	— + — — + — — ..
Mercurial solution	+ — — —	— — — — — — — —	— — + ...	— — — — + — + —
Phenol - -	+ — + +	? — ? — ? + ...	? ? + + ...	— — — — + + ? .. ..
Phenol - -	+ + ...	? — — — — —	— — — — — ?	? ? — + — .. .. ..
Chloro-phenol -	+ + + +	— — — — — ? — ..	+ + + + ...	— — + — — ? — ..
Chloro-phenol -	.. ..	— — — — —	+ + + + ...	— — — — — — .. ..
Formaldehyde -	.. ..	— ? — — — — — —	? — — — — ?	— — — — — — — —
Formaldehyde -	+ + ...	? + — — — + — —	— — — — — — — —	— — — — — — — —
Sulphur - -	.. ..	— — —	? + + + ...	— — — — ? .. .. ..
Sulphur - -	.. ..	— ? ? + + + — +	— + + — — + ...	? ? — — — .. .. ..
Phosphorus -	+ + ..	+ + + + + + ?	+ — + + ...	+ — + + + + — —
Phosphorus -	.. ..	— — — + ? + + ?	.. ..	— — .. + ? .. .. ..

Note.—+ denotes no growth. — denotes growth. ? denotes growth of other bacilli.



FIGS. 7-11.

Na, Nutrient Agar surface cultures of the typhoid bacillus, 24 hours old, at blood heat (37° C.), showing the presence and absence of germ life with solyptol solutions from 1:5000 to 1:50. The germicidal value of this antiseptic has also been confirmed for other pathogenic bacilli by (Griffiths, London, 1903) another authority. I found it to be a reliable plague antiseptic and disinfectant in the East (D.McD.).

CHAPTER VI.

The Toxin of the Plague Bacillus.

Those who have investigated the toxin produced by the plague bacillus are Drs. Markl, Babes, Toptchiff, Winisky, and Rustch.

Dr. Markl's investigations are most concise. I describe principally from his investigations.

1. The plague bacillus contains a virulent toxin in the interior of its body which dissolves very slowly in water.

2. A Bouillon culture of the plague bacillus kept at room temperature for a few weeks is highly toxic to small animals, and especially for Nankin rats (tame rats). This toxin exists in the body of the bacillus and its offspring.

3. This toxin has the same effect whether injected into the abdomen of an experimental animal or hypodermically. The animal so treated dies from septicæmia in from six to twenty-four hours with accompanying symptoms.

4. By injecting the toxin into the abdomen of experimental animals gradually increasing the dose, the animal can be rendered immune against the toxin ; but the immunity is not complete.

5. A considerable perturbation or depression of the system takes place by injecting a small quantity of the toxin into a cat, although the symptoms of infection will not be observed. Later, emaciation sets in and the animal dies. The hair falls out, cutaneous gangrene occurs, and the slightest friction produces a wound before death.

6. By injecting a small quantity of this toxin into a cat, and gradually increasing the dose until complete immunity is established, its blood serum becomes anti-toxic ; but it is not anti-microbic, having no germicidal power against the live bacilli.

7. The toxin is manufactured from a Bouillon culture. A precipitate is obtained by adding absolute alcohol.

8. The toxin is very sensitive to the chemicals which are used during its manufacture. It has mucilaginous properties also. It has a tendency to combine not only with alkaline albumen or proteids, but with all the proteoses, and presents the reaction of albumins.

An antitoxic serum could be obtained to produce immunity against the bodies of the bacilli as the substance used for this investigation upon immunization hitherto carried

on became antitoxic by heating. There is no reason against procuring an antitoxin.

He (Dr. Markl) manufactured the toxin by the following process :—By adding ten times the quantity of absolute alcohol to a Bouillon culture and drying the precipitate in oxygen, a white amorphous substance is obtained which dissolves in water. This substance is not pure toxin, but is an albumoid substance. The toxin manufactured from Bouillon cultivated for four weeks is toxic to Nankin rats. 18 Mgs. kill within twenty-four hours.

The organs of a marmot and rabbit which died from plague were washed in glycerine and physiological salt solution. These liquids were subjected to a Chamberlain filter. An infinitesimal quantity of the toxin was discovered in that where glycerine was used.

CHAPTER VII.

The Extra-Corporeal Existence of the Bacillus.

1. Water.

In distilled water plague bacilli could be detected alive for twenty days, in tap water and in well water for sixteen days, in sea water for six days (Wilm). An investigation was conducted into the wells and drains of the infected districts of Formosa, but no conclusive evidence has yet been obtained (Prof. Okada).

Repeated analysis of the well water of infected houses have been made, but the plague bacillus has only been discovered in one instance (Hankin).

2. The Ground and Soil.

Cultivations have been made with the soil from the floor of infected houses, and Nankin rats (white rats) were directly inoculated. Bacilli very closely resembling the plague bacillus have thereby been discovered, but not genuine plague bacillus (Yersin and Hankin).

It will live for two weeks in soil that is deprived of organic matter and in soil that contains organic matter it will live for two months (Glardin). Professor Okada in Formosa examined the soil beneath the floor of infected and non-infected houses for the presence or otherwise of plague bacilli. In the former, he often found positive evidence of the existence of the bacillus.

He says that if such soils should contain much organic matter having a uniform degree of moisture being less exposed to atmospheric influences, such would be the most suitable nidus for the plague bacillus where it could thrive permanently.

The investigations conducted by Mr. Chiyonosuke Yokote so as to ascertain how long the plague bacillus will live in the plague corpse, demonstrates that the corpse contains live bacilli from three to thirty days after burial. The corpses utilised for the purpose of this investigation were those of Nankin rats which had died of the disease. They were put in wooden coffins covered over and buried in moulds of metallic ware. The presence of the bacillus could not be traced in the ground surrounding the coffin boxes.

3. The Dust.

The existence of plague bacilli can be demonstrated by inoculating Nankin rats with the dust from the floor of the room wherein plague patients have lived (Prof. Kitasato at Hong Kong, Yersin, Hankin). But according to the investigations of Professor Okada in Formosa. there was no positive proof of the existence of the plague bacillus in the dust on the floor of infected houses. He, therefore, concluded that since the dust on the floor generally consists of pieces of articles and material in ordinary use which are not as yet very much pulverised. although plague bacilli may mingle with such dust, yet they cannot live long on account of the dust being unable as yet to furnish them with pabulum. But, on the contrary, dust containing such substances as the discharges from rats will long sustain the existence of the bacilli. So in the event of the bacilli mingling with the dust that contains the discharges and evacuations of plague-infected rats as well as those of non-infected rats, it will be a great source of danger.

4. Food and Drink.

The bacilli live from one to three weeks when attached to cooked or raw albuminous foodstuffs such as milk, turnips, white radish, potatoes, apples, and brown bread (Glardin).

5. The Discharges.

The urine contains live bacilli for twenty-two days.

6. Pieces of Cloth and Material.

The bacilli live for forty days when attached to a piece of hemp cloth, paper, and such materials.

CHAPTER VIII.

The Relation to Insects and Rats.

Flies, mosquitoes, fleas, ants, bugs, rats, and mice are closely connected with plague epidemics, as the investigations of Professor Kitasato, Yersin, Ogata, and others will show. These insects invade plague-infected rats and disseminate infection through sucking their infected blood. Should these insects come in contact with a human body they produce infection by inoculation through their sting or bite.

Professor Okada's investigations in Formosa demonstrate that insects, especially flies, become infected and die as soon as they have tasted a liquid containing plague bacilli. The bodies of those flies contain an innumerable number of plague bacilli, so that if a rat should eat those dead flies the rat in turn becomes infected; and when that rat dies in its nest the other rats will prey upon its corpse as they invariably do; then an epidemic will break out amongst them, and many dead rats will be observed.

His investigations, therefore, assert that the insects first become infected with plague, and the spread of the disease amongst rats follows through eating such insects. Also, since the discharges of such insects contain a considerable number of the plague bacilli, there is every opportunity for rats and human beings to be infected with the same.

CHAPTER IX.

The Portals Through which the Bacillus Invades the Human Body.

As described in the preceding chapter, the plague bacillus invades the human body through mingling with the following agencies :—

A. The discharges of insects, rats, and mice.

B. The sting and bite of insects.

C. The discharges of infected patients.

D. The dirt in the crevices of infected patients' fingers (Okada).

E. The underclothing.

F. Food and dust.

There are three principal portals, viz.: Wounds, the respiratory system, and the digestive system.

1. The Infection Through the Digestive System is clearly demonstrated by experiments on animals which instantly become infected when fed upon food mixed with the plague bacilli (Ogata). Intestinal plague is proof of the infection by the digestive system; however, such infection appears to be rather rare. The part of the digestive tract which is very liable to be invaded is the parotid gland (Okada). As the discharges of insects contain a great number of the plague bacilli, there is every opportunity of infection through the food upon which insects, such as flies congregate, more so is it the case since the bacilli can exist on foodstuffs for a very long time indeed.

2. The Respiratory System.

There are several different theories concerning the invasion of the respiratory system by the bacilli adhering to dust and floating particles in the air.

The theory of air infection is generally rejected. Experiments on animals negative infection through any circulating dust (Naisel).

3. The Wound.

The principal portal through which plague bacilli invade the system is a wound of the mucous surfaces and of the skin. This is the principal reason why the disease generally attacks the poorer classes, the laborers and mechanics.

Some time ago it was declared that this bacillus invaded the system through wounds of the surface (Greesingel), but since then, the investigations of several scientific men confirm this fact. The investigations in Hong Kong and Formosa will also establish the fact that this disease was far more prevalent amongst the poor Chinese who go about bare footed than the British or Japanese soldiers who wear boots and stockings. The latter, too, were very seldom affected during the epidemics (Aoyama, Okada).

An invasion through wounds of the feet attacks first the femoral glands, whilst those of the fingers and hands attack the axillary glands.

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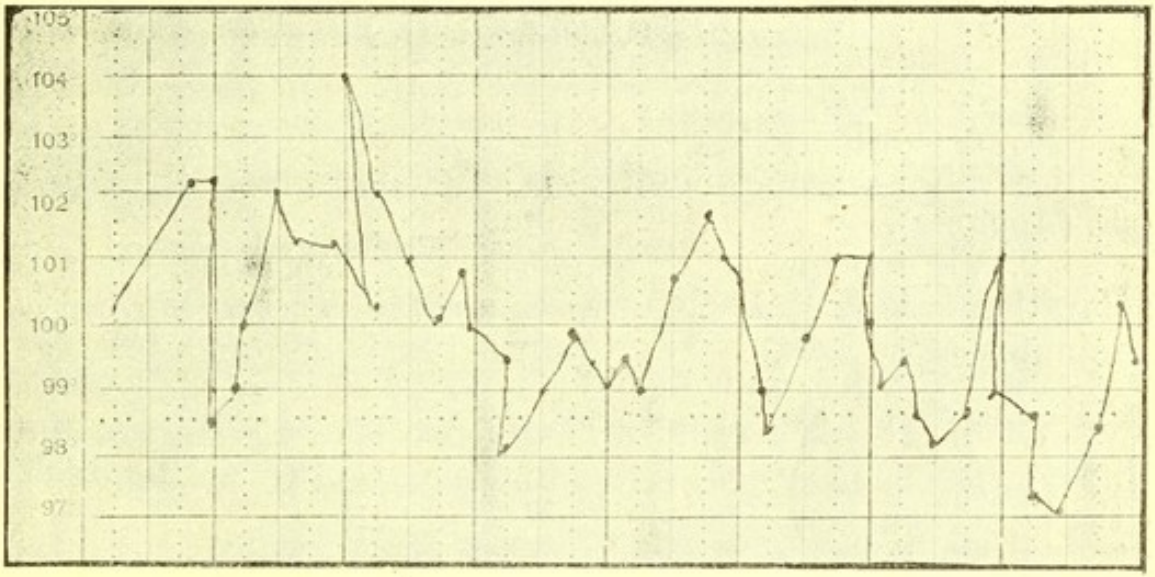
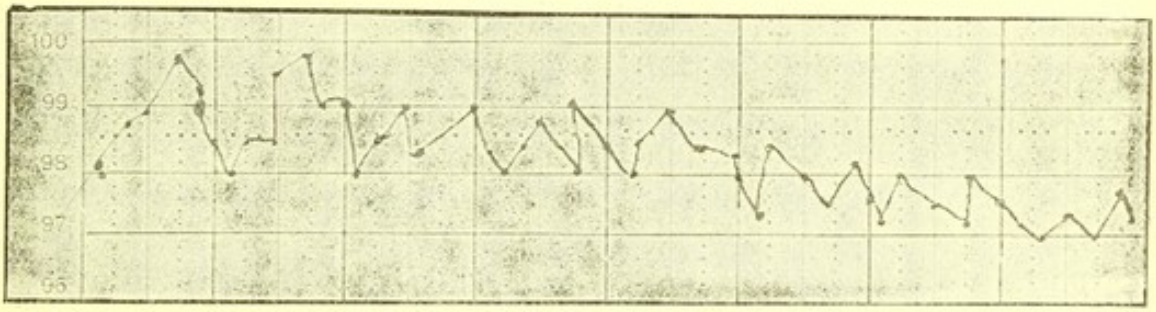
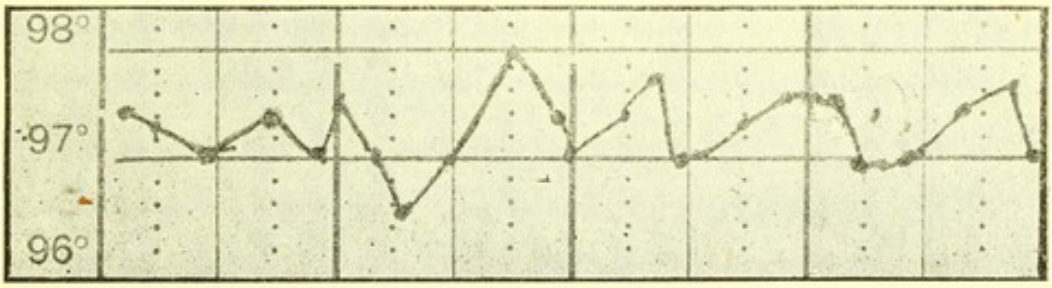
FIG. 12 (i.).—H.S., age 22; male. Bubonic. Indian.
2nd June, 1896. Admitted 6.30 p.m.



10th June. FIG. 12 (ii.)—H.S. (continued).

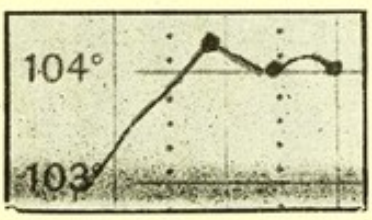


18th June. FIG. 12 (iii.)—H.S. (continued). Recovered.



I.

16th May, 1896.



Admitted 1.30 p.m.

Died on 16th May, 1896, at 10.30 p.m.

FIG. 13.—G.B., age 26; male. Bubonic. Indian.

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* Vide Part II., Chapter I.

CHAPTER X.

Practical Pathological Analysis.

A. Macroscopic Analysis of the Corpse.

1. A considerable rise in the temperature of the body is noticed after death, sometimes it amounts to more than 107° F. (Aoyama).

2. Vis mortua or rigor mortis is conspicuous, and its duration is long. It commences quickly even within thirty minutes after death. Sometimes the hands and feet are violently jerked, and it makes one doubt whether the body is yet dead. Such a rapid production of the vis mortua and its conspicuous features is due to desiccation of the tendons, the blood being deprived of liquids.

3. The signs of death are very prominent. They are often observed all over the body.

4. The blood is usually of a dark red colour.

5. The heart in most cases is dilated, particularly the right ventricle. The pericardium has often hæmorrhagic spots, but nothing is observed on the pleura. There is no trace of endocarditis, nor is a considerable increase in the pericardial fluid observed. The cavities are always dilated; as in the beri-beri heart, there are large blood clots and fluid blood. There is little dilatation of the left ventricle which is often empty. The chordæ tendineæ are often pale in colour, and there is fatty degeneration of the cardiac muscle.

6. The Lungs and Pleura.—The pleural membrane is always healthy. There is sometimes, although not often, fluid in the pleural cavity. This is generally observed in cases with axillary buboes which are primarily attacked, and always on the same side as the bubo. There is no hæmorrhage into the pleural cavity (Aoyama).

The lungs are always hyperæmic and œdematous. This is more marked especially in the lower lobes. Sometimes the lungs are markedly œdematous, but not often. They float in water. In many cases blood mixed with froth can be pressed out from the cut surface. The trachea is generally normal in most cases. Sometimes it is filled with blood fluid. The mucous membrane is often congested alone or presents a cyanotic appearance. It may be a general feature of plague that the tracheal and thoracic glands are not very liable to infection.

7. The spleen is always enlarged often to four or five times its normal size, soft, and easily torn. The splenic

parenchyma is hyperæmic. Hæmorrhages are observed under the capsule in the glandular parenchyma. The cut surface often presents an unusual number of fine white points.

8. KIDNEY.—The kidneys in most cases are slightly enlarged and very hyperæmic. The pyramids are conspicuous. The capsule is not adherent. The parenchyma presents fatty degeneration. The mucous membrane of the pelvis is generally more or less hæmorrhagic, but no hæmorrhages occur in the parenchyma.

9. The liver is always enlarged and hyperæmic. The parenchyma is often increased. Slight hæmorrhages often present themselves under the capsule.

10. STOMACH.—The stomach is often very much shrunken and crimpled. There is degeneration of the mucous membrane which is ash-white in colour (gastritis), or it presents catarrhal features. The stomach is often digested in cases that have expired some time. Hæmorrhagic maculæ are observed in some places. Small gastric ulcers are sometimes observed. Such ulcers might have been produced by the hæmorrhages becoming eroded (Aoyama).

11. The small intestines are always hyperæmic, presenting catarrhal features. The mucous secretion is increased; and small hæmorrhages are often observed. The solitary glands are always more or less tumid. They are more so in the lower part of the intestine which are sometimes the size of a pea. As in the case of typhus, Pyers patches are hyperæmic, and always present an indented appearance.

12. The large intestine. It has a catarrhal appearance, and sometimes exhibits slight hæmorrhages. The agminated glands are often enlarged.

13. THE MESENTERIC GLANDS AND THE PERITONEUM.—The mesenteric glands are always enlarged to the size of a pea or almond, presenting a pink colour or a greenish white tinge. Extensive hæmorrhages are often observed in the peritoneum.

14. BLADDER.—The bladder in most cases is shrunken, and sometimes punctate hæmorrhages are observed in the mucous membrane.

15. THE PHARYNX in most cases presents a dark red colour; cyanosis is often visible. The thyroid gland is sometimes atrophied, and its cut surface is congested.

16. The œsophagus presents catarrhal appearances, and its mucous membrane is cyanotic. The cervical glands are enlarged, and the œsophagus on the same side presents well-marked œdema.

17. The salivary gland is normal. The salivary gland

is even healthy in cases where the submaxillary glands are involved.

18. The mediastinal thoracic glands. Suppuration of these glands has been observed, but Prof. Aoyama denies this.

19. The dura mater is always hyperæmic. The pia mater is congested and œdematous. Sometimes, although rarely, there is degeneration of the blood vessels endarteritis. Professor Aoyama declares that he has observed a case termed "acute cerebral œdema."

The cerebro-spinal fluid is very much increased. There is often œdema of the brain, and numerous puncta crurenta. Hæmorrhagic maculæ are sometimes observed in the spine. (Prof. Aoyama).

20. Spine.—The appearances in the spinal membranes are similar to those of the brain. The arachnoid is specially tumid and hyperæmic. The cerebro-spinal column is normal. Sometimes the spine undergoes degeneration and softening.

21. The Lymphatic Glands.—The lymphatic glands present peculiar abnormalities in this disease. The femoral or axillary glands enlarge according to the portals through which infection has taken place. The cut surface of the gland always presents dark red infective macular appearances, sometimes ash-white with a reddish tinge. In appearance it resembles the spleen of anæmia. It varies in its consistence. The gland is often pulpy, but sometimes friable. The boundary between the medullary and cortical tissue is not distinct, and the cellular is indistinct.

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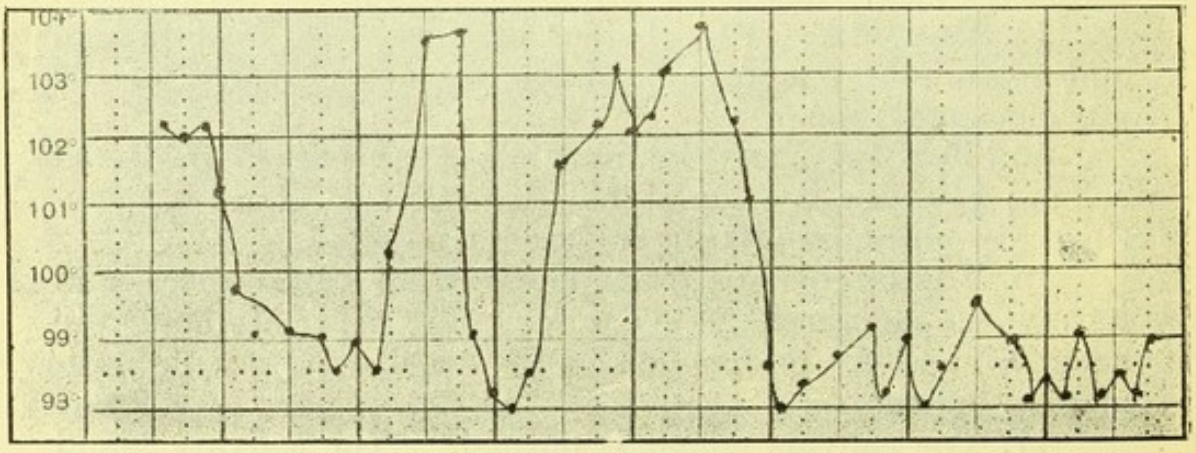
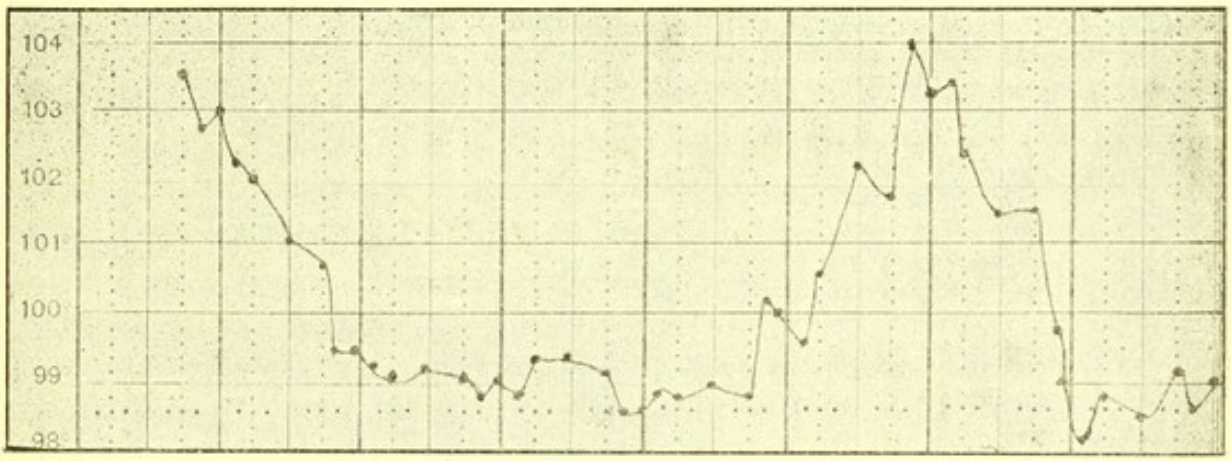
Fig. 14 (i.).—R.P., age 13; male. Bubonic. Indian. 18th June, 1896.

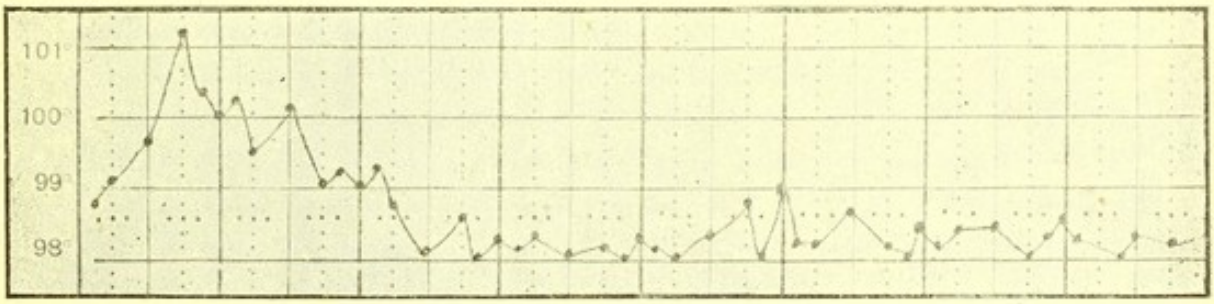


20th June. FIG. 14 (ii.).—R.P. (continued).

5th July. FIG. 14 (iii.).—R.P. (continued).



13th July. FIG. 14 (iv.).—R.P. (continued).



Discharged 23rd July, 1896.

B. THE MICROSCOPIC ANALYSIS.

1. THE BLOOD.—The red blood corpuscles are generally unaltered in quantity; but the leucocytes are considerably increased. The polynuclear variety of leucocytes is principally increased, and the bipartite nuclear leucocytes. Though the mononuclear leucocytes are increased in small quantities, yet the cosinophiles do not multiply.

The blood plates are usually increased. The calcula-

tions made by Prof. Aoyama in Hong Kong of the blood cells of a few patients are as follows (in 1 cubic millimetre) :—

No. 1	Red,	4,400,000	White,	120,000
No. 2	"	7,600,000	"	200,000
No. 3	"	8,180,000	"	110,000
No 4	"	6,700,000	"	20,000

2. The cardiac muscular fibres are indistinct and fatty.

3. The Lungs.—Hæmorrhagic enfarcts are observed in the lungs in most cases. In pneumonic plague there is an infiltration of the smaller bronchioles which has an ætiological influence. The disease process attacks both lungs in irregular patches, varying from the size of a pea to that of the hand. They are surrounded by a hæmorrhagic ring of a dark colour. Large numbers of the bacilli are present in the blood vessels.

4. Spleen.—The capillaries are dilated, and contain numerous bacilli. In the pulpy cellular tissue of the spleen there is a disintegration of the fibrous tissue often observed. Metastatic infective enfarcts and foci are visible. Such foci are produced by bacillary emboli in the capillaries.

5. Kidneys.—There is a thickening of the capsule of the glumeruli which are obscure and granular, the nuclei of which have no power to absorb the dye. The interior of the uriniferous tuberles is full of granular and hyaline tube casts. The glumeruli are increased in size in several places. There are ruptures in the capsule, or round cell infiltrations of Bowman's membrane having an infective nature. Sometimes the uriniferous tubules are dilated with hyaline casts. The interstitial parenchyma is often slightly increased. Sometimes an infective hyperplasia is observed. A round cell infiltration around the Malpighian body is particularly conspicuous. The capillaries in the interstitial parenchyma are always dilated. There are large quantities of the bacilli present in the interstitial parenchyma and in the interior of the Malpighian bodies.

6. Liver.—The hepatic cells are obscure and hypertrophied. The nuclei are generally destroyed. The interlobular capillaries are always dilated and hyperæmic. An infective round cell infiltration is often observed in the interlobular zone. Professor Kitasato's so-called "second nature" bacilli are present in the interlobular structures as well as the hepatic lobules.

In cases that have had jaundice during their illness the hepatic tissue is greenish yellow in colour. The gall bladder is observed to be filled with bile mixed with blood.

7. The Stomach.—The gastric epithelial cells are ob-

scured, presenting cloudy swelling. Sometimes marked proliferation of the gastric cells are observed.

8. THE SMALL INTESTINES.—The cells or the solitary glands are multiplied. They have no circular defined boundaries, but form in shape irregular bodies. The nuclei of the epithelial cells of the mucous membrane are often increased. The plague bacilli are found in small quantities in the proliferating cells of the solitary glands.

9. THE LYMPHATIC GLANDS.—The swollen lymphatic gland presents in its first stage an increase in the size of the individual lymphoid cells and a dilatation of the lymph channels. A small quantity of the plague bacilli are present under the capsule in the lymph sinuses. In this stage there has already been a bacillary metastatic infection of the gland parenchyma which exports red and white blood corpuscles. The bacilli are contained in this infective secretion of the gland. As the disease progresses the infection of the interior of the gland will increase, and the red and white corpuscles will invade the lymph sinuses and dilate them. The number of the bacilli, having greatly increased in the lymph sinuses under the capsule, present the characters almost of a colony. When the bacilli have progressively invaded the afferent lymphatic vessel and the lymph sinuses, the walls of the vessel undergo degenerative changes. There is an infiltration of the walls of the vessel with white and red corpuscles. and the structure of the walls of the vessel becomes impaired, The fibrous tissue separates, and finally each layer cannot be distinguished. Owing to such processes going on in the wall of the vessel rupture occurs, and a considerable hæmorrhage takes place in the structures around. This is the reason why the gland presents peculiar processes through the plague bacillus. The gland will finally perish with an infiltration of red and white corpuscles, and rod-bacilli. The original structural organization of the gland becomes more and more obscure. The plague bacilli inside the gland then survive outside in the cellular tissues around. (Vide Part II., Chapter III. for method.)

CHAPTER XI.

THE SYMPTOMS OF PLAGUE.

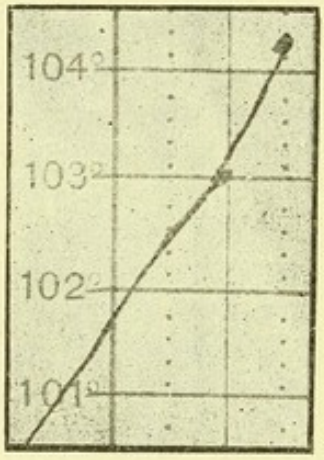
The symptoms of plague vary according to the part of the body affected with the disease.

It is divided into five varieties.

1. Bubonic plague.
2. Pneumonic plague.
3. Intestinal plague.
4. Inoculative plague.
5. Minor plague or pestis minor.

KENNEDYTOWN HOSPITAL.

FIG. 15.—F.C., age 18; male. Bubonic. Chinese, Ambulant.
17th March, 1896. Admitted 10 p.m.



Discharged on 17th March, 1896.

No. 1. BUBONIC PLAGUE.

The disease principally attacks young and middle age males and very rarely attacks infants or the aged. It is because the young or middle-aged are more liable to external wounds than infants or old people. Prof. Aoyama's investigations in Hong Kong were as follows :—

Males, 62.40 per cent.	Boys, 8.92 per cent.
Females, 19.23 per cent.	Girls, 9.45 per cent.

Professor Yamakime's investigations in Formosa were as follows :—

Ages.		Ages.	
11-20	9.50%	41-50	14.3%
21-30	46·0%	51-60	2.03%
31-40	25·6%	over 50	1.6%

Males	92.06%
Females	7.94%
Manual labourers	65.08%
Mental labourers	20.63%
Undefined	14.29%

THE LATENT STAGE.—The multiplication of the plague bacillus is bacteriologically very rapid, so much so that the

latent stage of the disease is accordingly very short. According to Professor Aoyama's investigation in Hong Kong it was from two to seven days, and that of Dr. Lowson's the average was nine days.

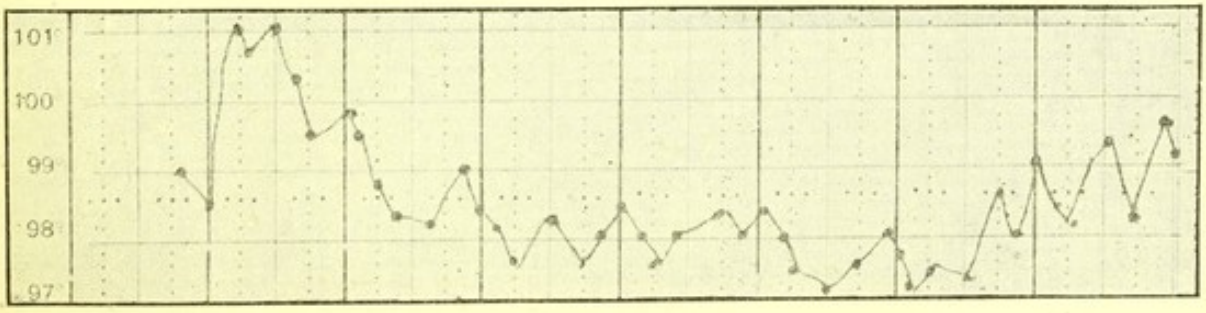
THE INCUBATION PERIOD.—This disease has no clearly defined incubation period. The temperature ushers in after a rigor or chill, accompanied with painful grandular swellings. However, sometimes prior to the rise in the temperature painful grandular swellings will first originate. If an incubation does at all exist its duration is from a few hours to two or three days. The patient complains of feeling languid, and of being tired and exhausted. The patient suffers from headache, nausea, loss of appetite, and giddiness. As in other acute fibrile diseases there are a few who complain of pains in the limbs and across the small of the back. Those who are very sensitive and pay personal attention to their health are cognisant at the beginning of the change of their constitution before the grandular swellings become very paintul; but the lower laboring classes do not notice anything until the adenitis causes very acute pain.

After the initial stage of the disease, the order in which the special symptoms peculiar to this disease appear is very variable. The temperature generally rises after a rigor or chill. Sometimes it rises suddenly. A febrile condition prevails for a short time. The temperature rapidly rises to 102° F., 104° F. or more, and then the temperature remains at this. (Vide Figs. 12-17.)

The temperature in a mild case is generally below 102° F., and the progress of the disease is comparatively slow. It is unusual for the patient to complain of pain in the enlarged gland soon after the fever sets in. However, sometimes, though rarely, the bubo maturates a considerable time afterwards. The bubo appears on the sixth day after the fever sets in (Aoyama).

KENNEDYTOWN HOSPITAL.

FIG. 17 (i.).—C.M., age 47; male. Bubonic. Chinese. 14th April, 1896.



Calomel.

FIG. 17 (ii.).—C.M. (continued).

22nd April, 1896.

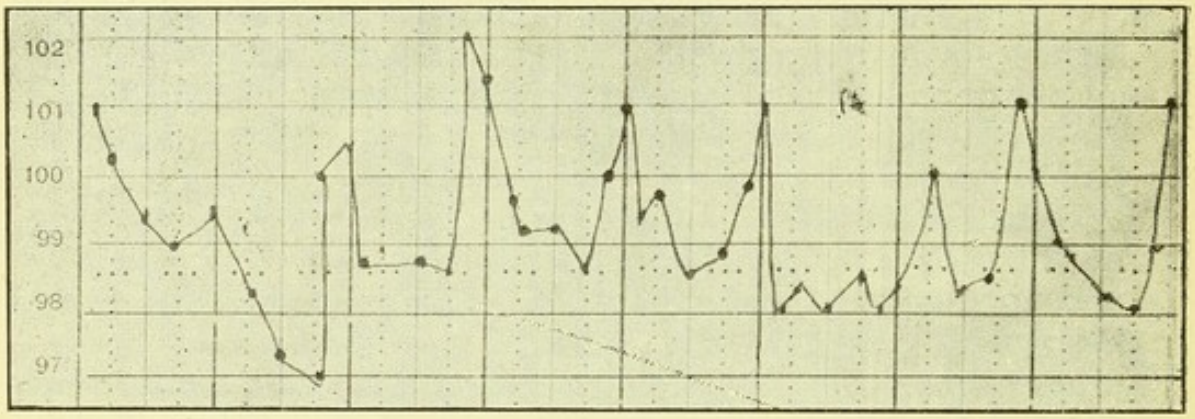


FIG. 17 (iii.).—C.M. (continued).

16th May, 1896.

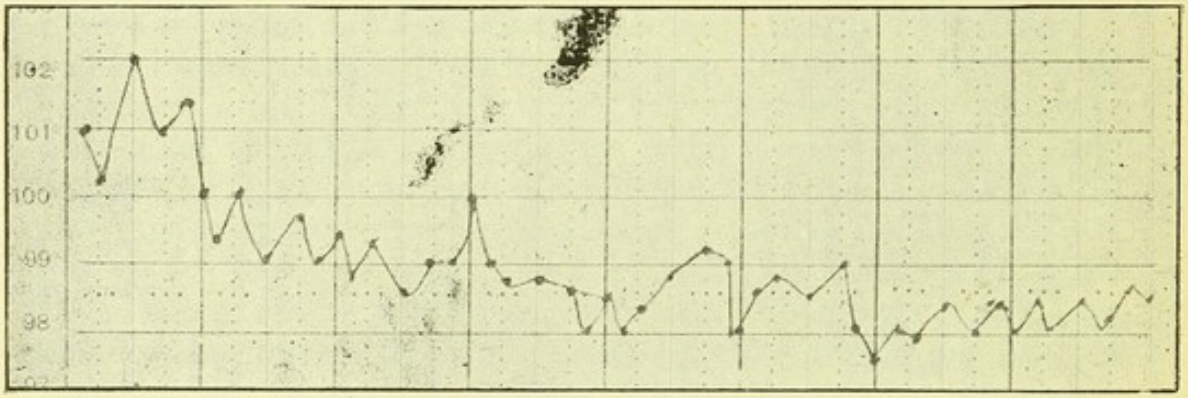


FIG. 17 (iv.).—C.M. (continued).

24th May, 1896.

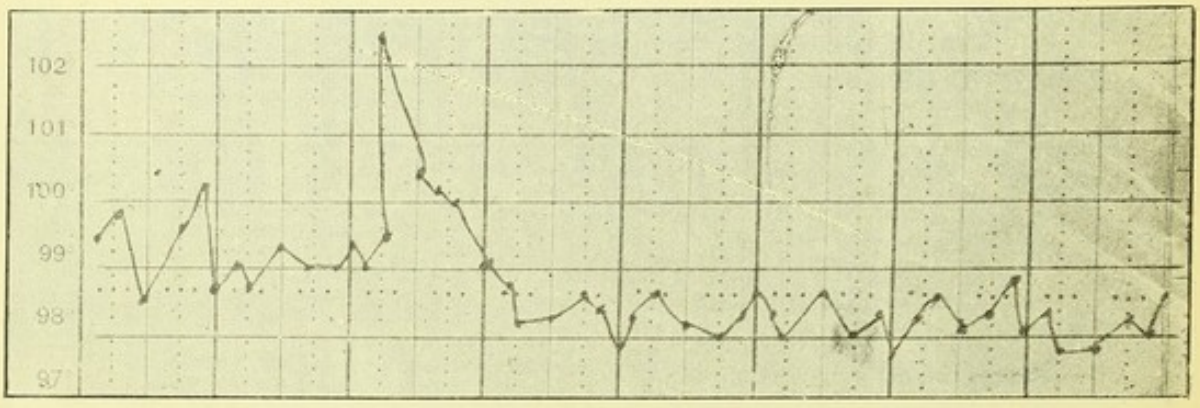


FIG. 16 (v.).—C.M. (continued).

16th May, 1896.

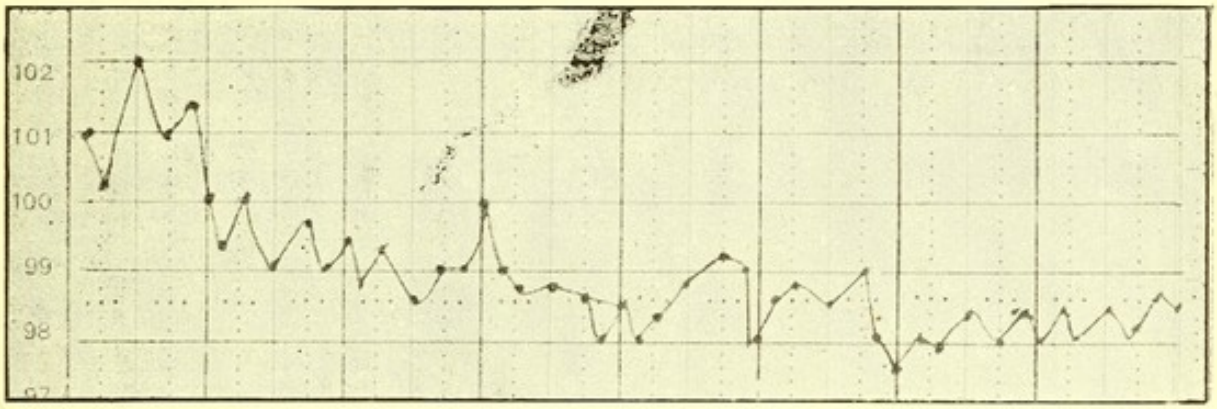


FIG. 16 (vi.).—C.M. (continued).

24th May.

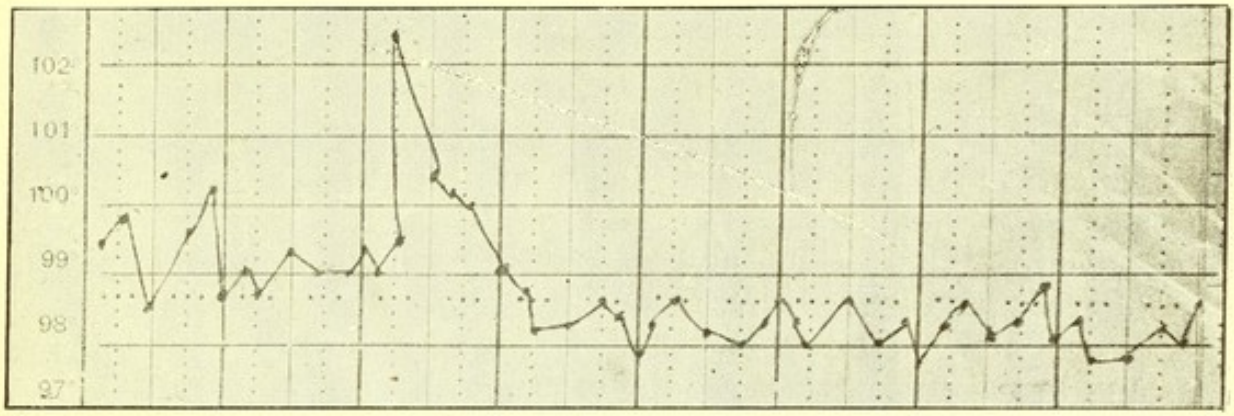
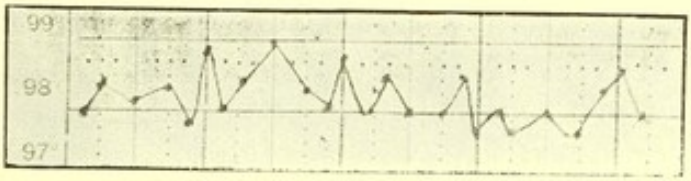


FIG. 16 (vii.).—C.M. (continued).

1st June



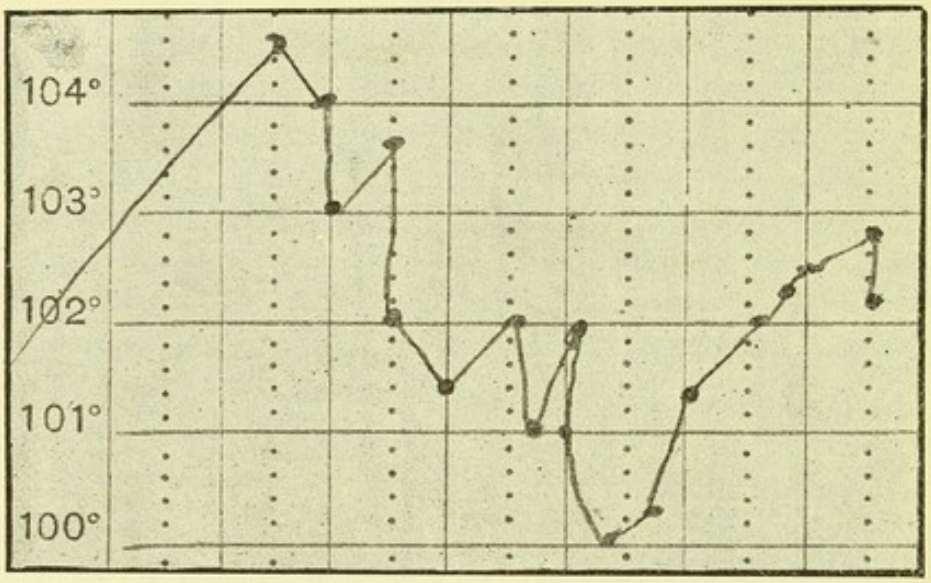
Discharged on 24th June, 1896.

A Malignant Case.—The patient complains of headache and giddiness. The appetite is lost. Although there is nausea and vomiting, this generally ceases after a short time. Acute œdema of the brain sets in early. Nausea and vomiting sometimes continue throughout the various stages of the disease. The substance vomited is watery, a mixture of bile or a blood mixture resembling coffee ground.

KENNEDYTOWN HOSPITAL.

FIG. 17.—L.I., age 38; male. Bubonic. Chinese.

12th May, 1896. 13th. 14th. 15th.



Died on 15th May, 1896, at 4 p m.

From the beginning there is marked prostration. Sometimes the patient is unable to stand up or walk straight very soon after the fever sets in.

He tosses about in bed, or lies on his side complaining of great pain ; but thirst is not intense.

Sometimes he complains of pains in the hips and loins, and in the epigastrium (at the beginning).

Retching is very rare. If it does occur it is very troublesome. Diarrhœa is observed in the initial stage, or in the early part of the second stage, but later on the evacuations are normal. Still, sometimes diarrhœa persists throughout the whole disease.

Cerebral symptoms are evident in a malignant case. Very soon after the fever sets in the patient becomes delirious and talks incessantly day and night. But such symptoms generally develop after the second day, and in a mild case the patient talks during the night only. The patient's countenance is at first only flushed, but later on it presents typhoid features, being without expression or emotion. The eyes are sharp, and the conjunctival membranes are hyperæmic. Prof. Yamakime considers that this hyperæmia of the conjunctival membranes is a constant symptom of this disease. The pharynx is of a dark red colour, and is hyperæmic. The tonsils are often enlarged. The tongue at the first is moist and covered with an ash-white fur, later on it dries up and becomes typhoidal. The dorsum of the tongue is coated

white. A greenish red colour of the tongue is said by some to be a special symptom of this disease. Prof. Aoyama affirms that he did not see any such cases. The skin and surface of the body are hot and dry. In the febrile stage respiration becomes shallow and hurried. Dyspnœa is present in those cases that have enlarged cervical glands pressing on the larnyx, and also during the final stage of the disease in œdema of the lungs. Further, dyspnœa will be present when hæmorrhage occurs in the spine near the respiratory centre without any apparent cause during the clinical examination.

The pulse is between 90 and 120. It is usually full and expansile, presenting dicrotism. It becomes irregular when the patient is approaching his end.

HEART.—A systolic bruit is audible over the mitral and pulmonary areas. It is directly transmitted to the right and left thereof.

LUNGS.—The lungs are healthy in some cases; but crepitations are audible when the end is approaching owing to œdema of the lungs. Some present pneumonic symptoms from the beginning of the disease. Dr. Lowson says that he has seen a few cases of hæmoptysis.

SPLEEN.—The spleen enlarges on the second or third day of the disease, and is sometimes palpable. Sometimes, it enlarges to the extent of 2 or 3 centimetres beyond the costal margin. The liver is also enlarged and palpable.

URINE.—The urine is cloudy and of a dark red colour. It contains albumen, and gives a slight indican reaction. It does not give the diazo-reaction. On this point it differs from intestinal typhoid. Microscopically, it contains granular and hyaline tube casts. Very often there is also blood in the urine.

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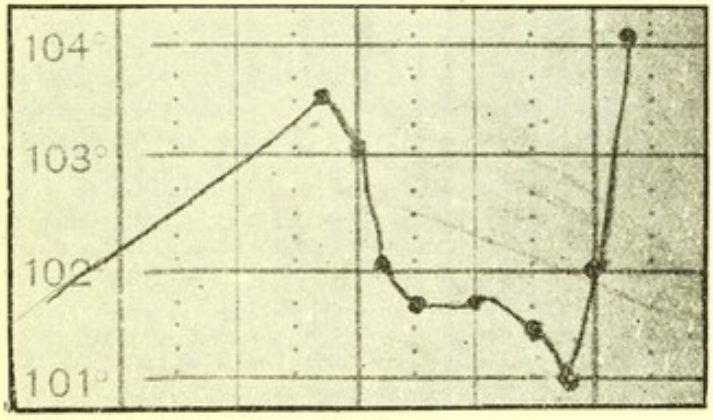
FIG. 18.—T.K.I., age 52; male. Bubonic. Chinese.



Died on 6th May, 1896, at 6.15 a.m

The temperature rises rapidly as in pneumonia. It rises over 104° F., and remains at this from two to four days. In convalescence the lysis will be accompanied with perspiration, or a simple remission of the temperature takes place. Sometimes, although rarely, the fever is irregular and persistent. In those cases that the urine contains albumen and tube casts there is no œdema present. Prof. Aoyama observed hæmorrhagic spots in cases in Hong Kong.

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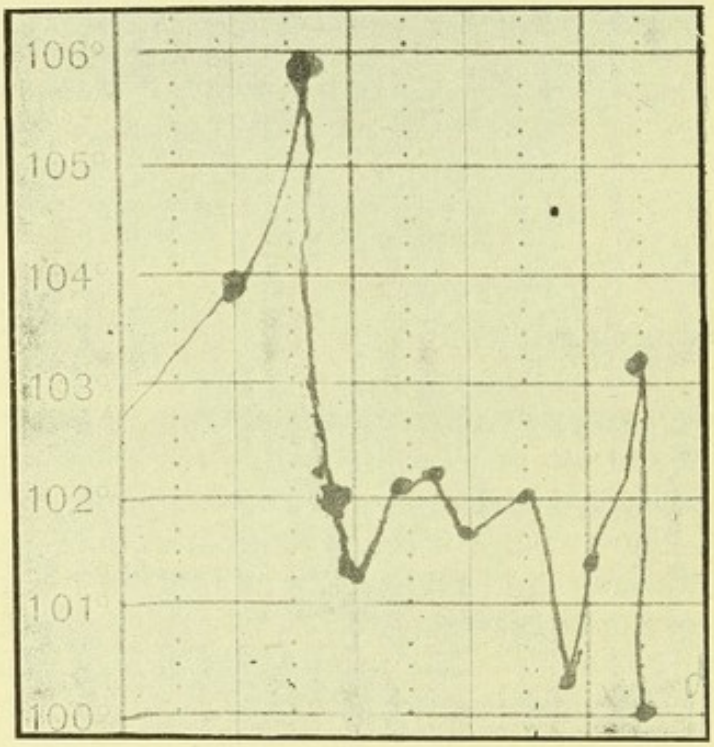
FIG. 19.—X., age about 30; male. Bubonic axillary, left. Chinese. 12th May, 1896.



Died on 14th May, 1896, at 8.30 a.m.

There is no impairment of the sense of hearing. The cerebral symptoms are talking and delirium; but convulsions are very rare.

The bubo is the special symptom of this disease. It develops prior to, simultaneously, or after the fever sets in. After one or two days the affected gland swells up as large as an egg. The pain intensifies as the swelling increases, and the movements of the limb are impaired. Sometimes the hip cannot be extended when the femoral glands are involved Owing to the intense pain, sleep is disturbed, and the patient moans and cries out loudly. But sometimes there are cases that have no pain. This is elucidated during the clinical examination.

The bubonic swelling is very characteristic in this disease. There is first of all a lymphangeitis of the afferent lymphatics. When the portal through which invasion has occurred is in the lower limb, the internal femoral glands inflame first of all, then the infection extends to those over Poupart's ligament, then to the axillary glands on the same side. The cervical and sub-maxillary glands will also be successively affected. Its course of progression is always on the same side. It never goes over to the other side (Aoyama). Its origination in the ulnar or popliteal gland is very rare. It originates in the femoral glands about two or three centimetres below Poupart's ligament, which first enlarge. Then those above them follow. The invasion of the external glands is rare. When the axillary glands are affected the glands in the joint in the immediate vicinity are also observed to be enlarged.

As the gland inflames extensive inflammation of the cellular tissue around develops, so much so that the gland and subcutaneous tissue cannot be moved. Owing to the inflammatory process involving the gland and surrounding tissues, the structures are hard and tense to the touch, being rough and in masses. Later on the skin becomes red and prominent. The pain is intensified by pressure owing to the marked implication of the cellular tissue around. The buboes either undergo resolution or suppuration. Resolution usually occurs in mild cases. Suppuration takes place either in malignant or mild cases. Resolution proceeds very slowly sometimes taking about two months. Suppuration occurs in the third stage of the disease. It is observed from the eighth to the fourteenth day of the disease. Its appearances are similar to an ordinary abscess.

Death.—In very malignant cases death ensues before the bubo has developed. Death is due to failure of the cardiac action. The patients never become unconscious until death. Some die within two days, but such is a rare occurrence. Death generally occurs between the second and eighth day. The average is on the fourth day. As suppuration generally occurs after the tenth day death usually ensues prior to the suppuration of the bubo.

Second Stage.—If the patient survives the disease its influence mitigates, and he is tided over to the third stage, which is mostly of a remissive character. The fever rapidly abates accompanied by perspiration ; but sometimes the remission of the fever is very slow and protracted. At the end of the first or the beginning of the second week it is usual in malignant cases for the disease to subside in the third stage.

In a mild case sometimes the disease subsides in two or three days from the commencement of the illness.

The Third Stage.—Suppuration of the buboes is very common. Buboes present features of maturation between the eighth and fourteenth day. Suppuration of the bubo is viewed by some as a good omen, but such is not the case. It only indirectly points out the fact that the disease is in transition from the critical stage.

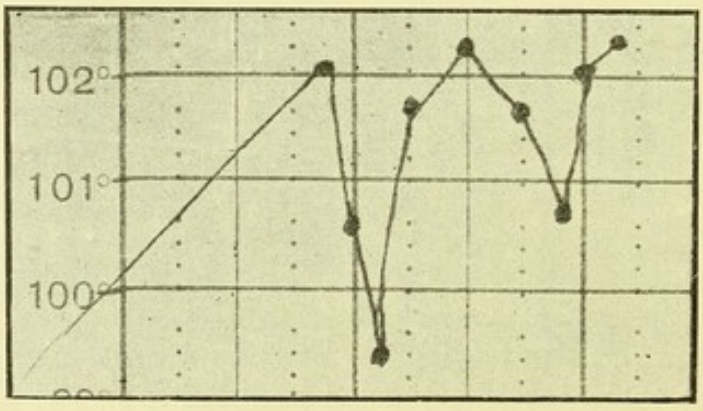
The cerebral symptoms subside in the third stage, and the mental condition becomes bright and clear. But the headache still persists, and the memory is weak still. The pains subside. The body is considerably emaciated, and the countenance is pale. The tongue is moist and its fur has gradually disappeared. The pharynx is hyperæmic, but subsiding. The appetite gradually recovers. Some cannot stand on account of weakness in the limbs. The splenic and hepatic enlargements will dissipate. The pulse is still a little rapid, and the urine still contains albumen. The patient gradually passes into the convalescent stage—in the third stage—if the suppurative process does not extend, nor any concomitant disease is present.

The Fourth Stage.—The commencement of this stage varies according to the nature of the disease, but it is from the end of the first to the beginning of the fourth week. When the appetite will improve the tongue becomes moist and red. The pulse will diminish to 80 or 90. Respiration is quiet, and sleep is sound. The strength in the limbs and the memory have not yet quite recovered.

When suppuration is extensive, recovery is slow. Sometimes suppuration persists for a month or more. When a bubo does not suppurate it gradually resolves. After two or three months it entirely disappears.

KENNEDYTOWN HOSPITAL.

FIG. 20.—L.K., age 22; male. Cervical. Chinese. 8th May, 1896.

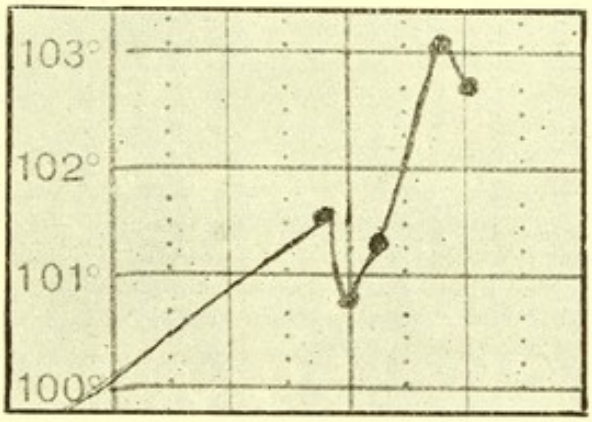


Died on 10th May, 1896, at 7 a.m.

Calomel.

KENNEDYTOWN HOSPITAL.

FIG. 21.—N.T.M., age 31; female. Bubonic. Chinese.
5th May, 1896.



Calomel.
Died on 6th May, 1896, at 1.30 p.m.

CONCOMITANT DISEASES.

1. Nephritis is often present in malignant cases, usually on the third or fourth day. The urine is of a dark red colour, and contains albumen, granular, and hyaline tube casts. Œdema of the body is very rare ; it never causes suppression of the urine (Aoyama).

2. LYMPHATIC GLANDULAR ABSCESSES.—Boils. Purulent enlargements of several of the lymphatic glands are present. Pyæmic abscesses of the internal organs are sometimes observed.

3. JAUNDICE.—Catarrh of the gall bladder is produced by the extension of the inflammatory process to the stomach and intestines, hence the jaundice is of a catarrhal nature.

Dilatation of the capillaries around the gall ducts also produce pressure on the ducts which soon produces jaundice (Aoyama).

4. Pleurisy is often produced when the axillary glands on the same side of the chest are affected.

5. Pneumonia is often conjointly produced.

6. Tracheitis occurs on the third or fourth day, but never extends very far.

7. Parotitis has been observed.

8. Retching is sometimes very troublesome.

9. Melæna is often observed due to acute enteritis.

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FIG. 22 (i.).—T.O., age 22; male. Bubonic. Chinese.
28th April, 1896. 1st May.

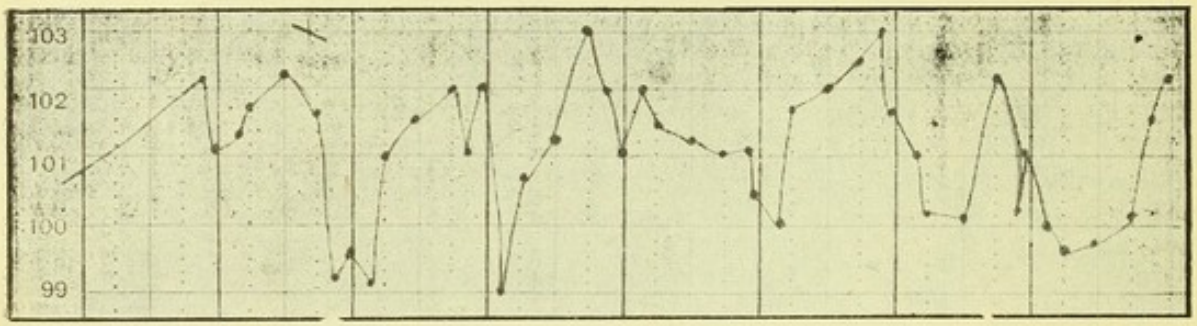
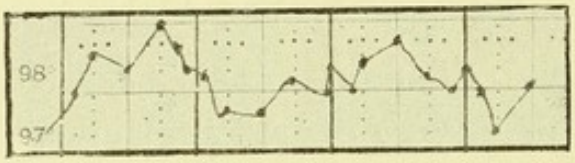


FIG. 22 (ii.).—T.O. (continued).
8th May.



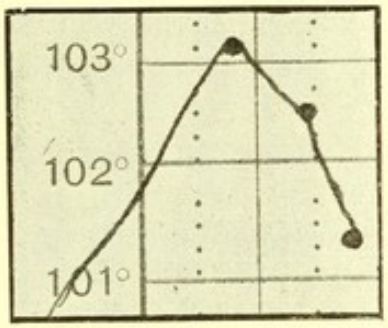
Discharged

No. 2. PNEUMONIC PLAGUE

This disease presents the symptoms of common croupous pneumonia. It first begins with a chill or rigor. The skin becomes red, nausea, and vomiting supervene. As in the case of a critical pneumonia, there is coughing, expectoration. and pains in the chest. The respiration becomes hurried, Sometimes it is 70 or more per minute. Then dyspnœa supervenes. During auscultation crepitations are audible. There is impairment of the percussion note over one of the lobes which may be found extending into another lobe. The sputum which is expectorated from the capillary bronchi is usually bloody. The patients usually succumb on the third day through exhaustion or dyspnœa. When the case is not specially observed it appears at a glance as if it were one of croupous pneumonia, and has often been treated as such. Besides the plague bacillus the sputum contains Fränkel's pneumococcus. Sometimes the disease suddenly attacks those who have lung trouble, which forms a nucleus for pneumonic plague.

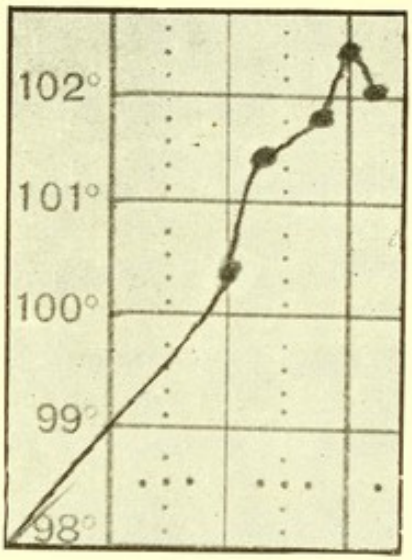
KENNEDYTOWN HOSPITAL.

Fig. 23.—C.H., age 36; male.
Bubonic. Chinese.
19th May, 1896.



Died on 19th May, 1896, at 10 p.m.

Fig. 24.—H.Y., age 79; female.
Bubonic. Chinese.
10th May, 1896.



Died on 11th May, 1896, at 5 a.m.

No. 3. Intestinal Plague.

In animal experiments it is observed that intestinal plague can be produced by feeding animals on foodstuffs mixed with the plague bacillus. Lesions are present in the intestines and spleen resembling typhus. Primary ulcers can be observed in various parts of the mucous membranes of the stomach and intestines (Winisky). It is a fact that this disease existing in the human being was investigated by Dr. Wilm in Hong Kong in 1896. Whilst first confined to the walls of the stomach and intestines, and to the solitary glands in the immediate vicinity, it is only a simple gastric and intestinal catarrh. There is no more than diarrhœa with pains in the abdomen. Mucous and bloody secretions are present; but when the plague bacillus invades the circulation then it suddenly produces critical symptoms, and the patient at the outset will succumb to septicæmia within three days. The spleen enlarges very rapidly, and is tender to pressure which is a special symptom. Therefore, diarrhœa accompanied with bloody evacuations and fever are prevalent during epidemic of plague; the cause of such symptoms being unknown, it will be essential to treat such as doubtful cases of plague. The evacuations of such patients suffering from this disease contain large quantities of the plague bacilli.

No. 4. Inoculative Plague.

This variety presents particularly special symptoms. The patient feels, on the part of the skin affected, burning dains or an itchy sensation, and later on there appear whitish spots the size of a lentil seed. The skin around presents a deep red colour, being inflamed. Then a small pustule or eruption the size of a seed appears on the part affected. The outline of the affected area, on being observed, presents dark red lines. When the watery pustule bursts a dark spot of ulceration will be visible, the base of which desiccates gradually. It gradually eroded the surrounding tissue. The physical symptoms are very mild. Excepting the corrosive nature of the sore its prognosis is good. But during its progress lymphangeitis occurs, and a bubo results—bubonic plague. Sometimes during the course of the disease a phagedænic ulcer is observed.

No. 5. Pestis Minor or mild cases of plague may prevail prior to or after an epidemic of the malignant variety of plague. It is often observed in Bombay, India. It has no initial stage of fever and such symptoms, but the glands appear to be directly swollen which either undergo suppuration or resolution. The patient recovers. Whether pestis minor is caused by the genuine plague bacillus or not is not definitely settled yet. Pestis minor prevailed in Mesopotamia in 1867-77, and in Calcutta in 1895-96.

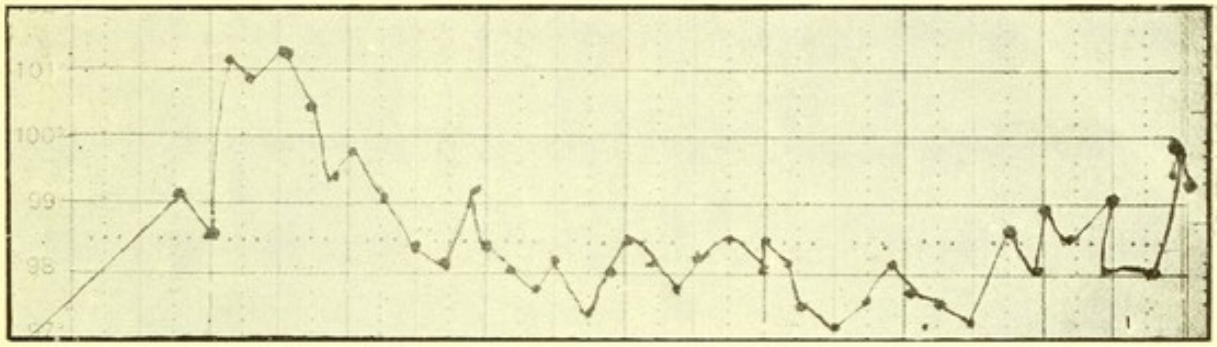
SEQUELÆ.

Those which belong to subsequent ailments after recovery are as follows :—

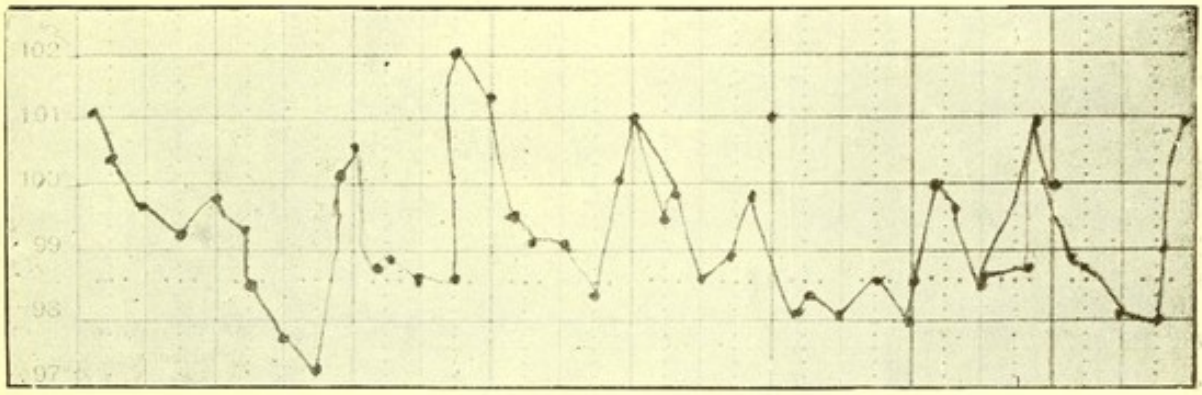
1. Paralysis.
2. Permanent cardiac injury.
3. Torpidity of the circulation.
4. Paræsthesia of the neck.
5. Paralysis of the vocal cords.
6. Anæsthesia.
7. Aphasia.
8. Deafness.
9. Hysteria.
10. Gangrene of the lung.

KENNEDYTOWN HOSPITAL.

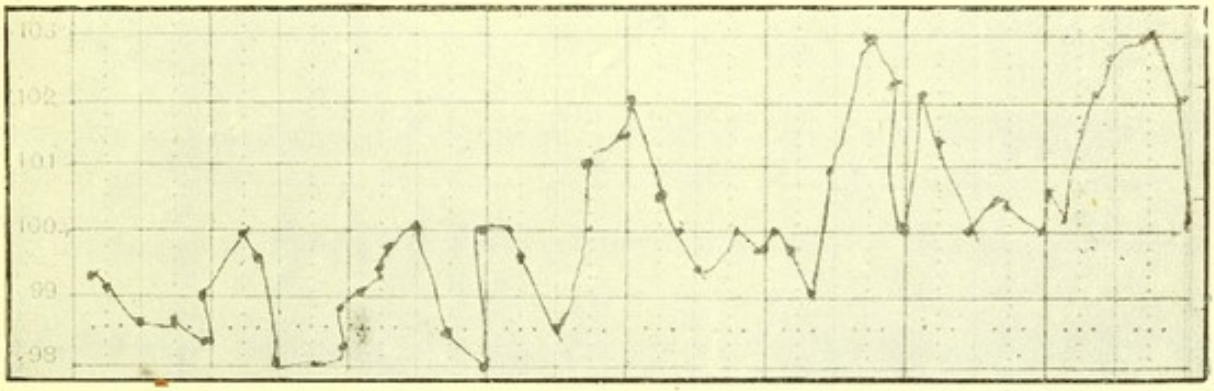
FIG. 25 (i.).—C.M., age 47; male. Left and Right Femoral, Bronchitic. Chinese. 14th April, 1896.



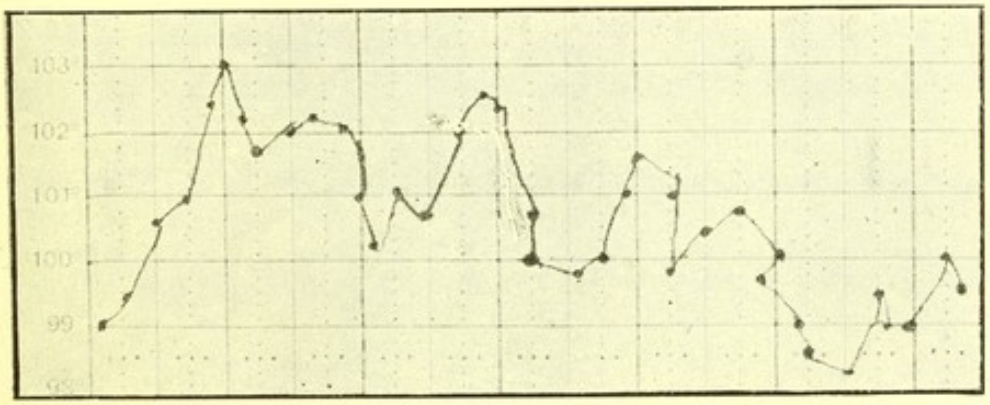
22nd April. FIG. 25 (ii.).—C.M. (continued).



30th April. 1st May. FIG. 25 (iii.).—C.M. (continued).



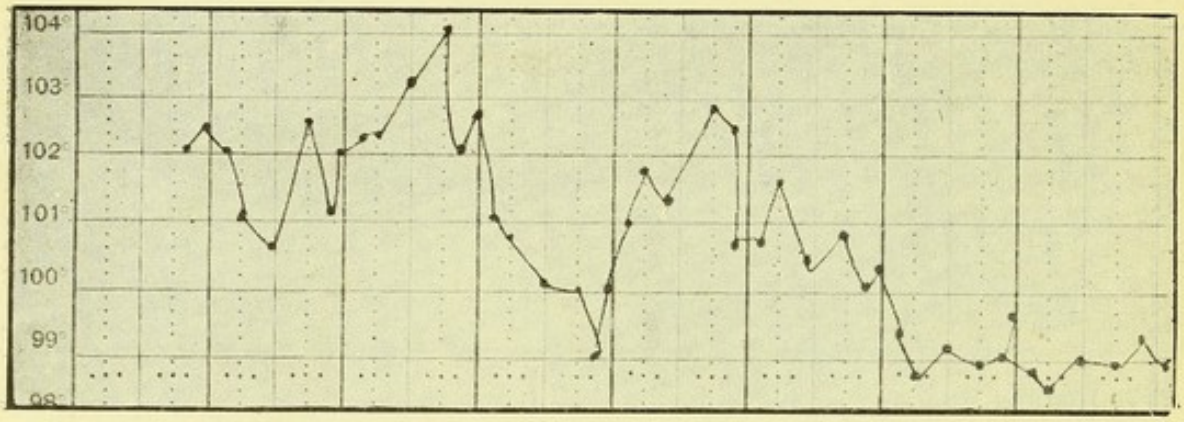
8th May. FIG. 25 (iv.).—C.M. (continued).



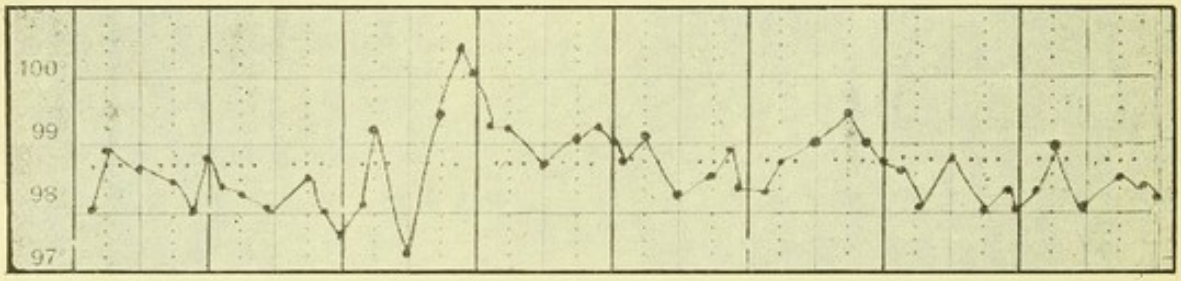
Recovery.

KENNEDYTOWN HOSPITAL.

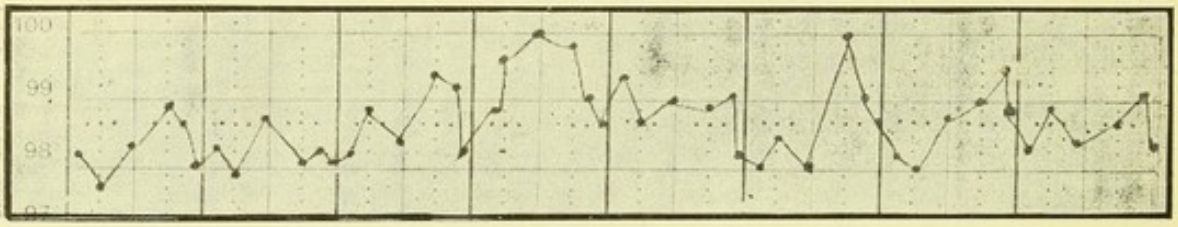
26th May, 1896. FIG. 26 (i.).—L.T., age 44; male. Bubonic. Chinese.



3rd June. FIG. 26 (ii.).—L.T. (contnued).



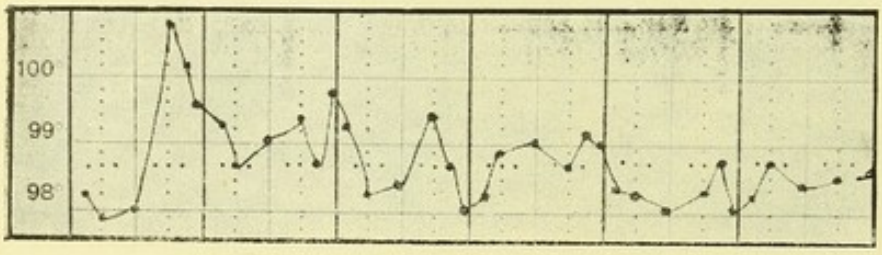
11th June. FIG. 26 (iii.).—L.T. (continued).



19th June. FIG. 26 (iv.).—L.T. (continued).

FIG. 26 (v.).—L.T. (continued).

27th June. 1st July.



Discharged 16th July, 1896.

CHAPTER XII.

The Prognosis of Plague.

The prognosis of pestis minor is good, but in a case with a melancholic disposition the prospects of recovery are generally not so bright. In a very acute case it is decidedly worse.

Of the bubonic variety the femoral has the best, and the axillary comes next, but the condition of the patient whose spinal glands are attacked is very bad.

The prognosis of pneumonic plague is decidedly bad, and there is little prospect of recovery.

The condition of the heart is of primary importance in judging of the patient's prognosis. The one that has a strong and regular pulse has a much brighter prospect of recovery than the other with a weak and irregular pulse.

Death generally follows from the sixth to the tenth day of the disease—the limits. Those who survive for ten days have some slight hope of recovery.

The acute cerebral type of the disease is of the worst possible prognosis, and will positively succumb. According to the investigations of Professor Yamakime in Formosa the mortality of the patients who had the cerebral type of the disease was 39·2%, and of those having no cerebral symptoms was 9·8%. He also investigated the prognosis of regional bubonic plague. The results are as follows :—

Femoral bubonic mortality ..	61·9%
Axillary	80·0%
Submaxillary and cervical	71·3%

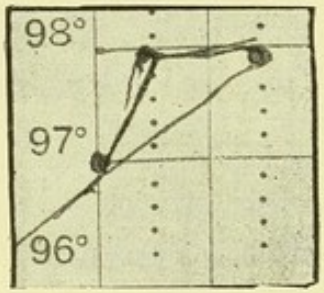
The mortality of plague is very high. Dr. Lowson's investigations in Hong Kong at the time of the epidemic in 1894 are as follows :—

Of 2,619	Chinese	93·4%
11	Europeans	18·0%
10	Japanese	60·0%
13	Indians	7·7%

During the time of the Bombay epidemic it was 95 to 99%. Professor Ogata's investigations in Formosa 56·1%. The average mortality in Hong Kong in 1896 was 85·0% according to Dr. Wilm's investigations.

KENNEDYTOWN HOSPITAL.

FIG. 27.—Y.; age, about 15; male. Septicæmic. Chinese.
14th May, 1896.



Died on 14th May, 1896, at 11.45 p.m.

CHAPTER XIII.

THE DIAGNOSIS OF PLAGUE.

The diagnosis of plague is not always simple. A mild case, or pestis minor, is very easily mistaken for venereal disease or simple infective adenitis. It is difficult to distinguish pneumonic plague from croupous pneumonia. Intestinal plague is readily confounded with intestinal ulceration or intestinal typhus. It is hard to distinguish plague in infants from simple meningitis. In a case of bubonic plague presenting clear symptoms the disease cannot be mistaken for any other. The high temperature, the marked degree of prostration, the sharp appearance of the patient's countenance, the painful swelling of a certain gland, and suffusion of the conjunctival membranes are the symptoms which can be easily recognised at once as plague. Besides, the glandular swelling or bubo has special characteristic features. The surrounding area is tense, and not easily moved. It is not smooth, but hard and nodular, which are its peculiarities.

The disease cannot be definitely diagnosed as a positive case until the particular bacillus of the disease is positively found. Therefore, the sputum in pneumonic cases is to be submitted to an examination, and in the intestinal variety the evacuations are to be examined. In a case of bubonic plague it is necessary to make a microscopical examination of the glandular contents. A bacteriological examination of the blood and urine is necessary in the final stage of the disease when septicæmia has set in.

An ordinary Pravaz syringe is used for obtaining the contents of a bubo. Wash the instrument first, taking antiseptic precautions, in a five per cent. solution of carbolic acid, or in absolute alcohol. Then wash it in 0·6% sterilised physiological salt solution or 0·5% carbolic acid solution. The instrument is to be applied in the usual way to puncture the gland, and the contents are drawn out slowly but steadily. However, when the gland is not very much involved, suppuration not having yet occurred, this simple procedure will not give good results, as it will not absorb the glandular contents. So that it is best in such cases after inserting the needle to withdraw it. Insert it again in different directions, and move it to and fro, so as to tear up the glandular substance. Then proceed to draw out the contents. In a difficult case. it is better to excise a piece of the gland.

In the intestinal variety, according to the usual method, the material for examination is collected from the evacuations, in pneumonic plague, from the sputum and from the watery secretion or discharge in the case of inoculative plague.

A needle selected specially long is used on the cadaver. When diagnosing the disease it is plunged into the bubo, heart or spleen, and the contents thus obtained are examined.

In examining a bubo or corpse spread the material on a cover glass with a platinum wire in obtaining material from the heart, spleen, urine, fæces, sputum, or secretion. Dry it in the atmosphere, and pass it two or three times through the flame to harden. Stain with fuchsine or methylene blue, and examine it under the microscope. Then the particular bacilli can be detected. Use Gram's method in case of any doubt. Stain the material first with aniline water gentian violet, and wash with a mixture composed of iodine one part, iodide of potassium two parts, and water 300 parts, and then with pure alcohol to distinguish them from the pyogenic bacilli (Vide Plates I., II.) contained in the gland, which are not decolourised by the process. The pyogenic streptococci produce extensive inflammation of the veins and lymphatics.

The streptococcus pyogenes in the living body do not invariably retain their chain-shaped appearance, but it is usually present as a diplococcus. Consequently a tyro might have a mistaken notion that the plague bacillus is a diplococcus, about which great care must be exercised.

Fränkel's pneumo-bacillus is generally found symbiotically with the plague bacillus in the sputum. The microscopic appearances of those two bacilli sometimes cannot de-

termine the one from the other. Therefore, it is necessary to distinguish them by Gram's process. Fränkel's bacillus is not decolourised by the process. Sometimes, though not often, the sputum contains Friedlander's pneumo-bacillus as well, but the latter can be easily distinguished from the former, as it is larger in shape, and its capsule is conspicuous.

In examining the evacuations for the plague bacillus, numerous other bacilli having similar features and appearances common to the plague bacillus are encountered in the fæces. These are the bacillus coli communis which exist in the intestines, and the typhoid bacillus. Although these bacilli present staining properties, the plague bacilli, being decolourised in its centrum, whilst the two former retain the dye in their centra, yet the end of such a method of bacteriological diagnosis can only be attained with the very best microscope used, and in the hands of the most experienced and skilful bacteriologists. Again, the plague bacillus possesses no motility which is common to the others.

In the event of these examinations leaving any room for doubt, conduct experiments on animals. Make a microscopic examination of the material collected from the blood of the heart, and of the contents of the spleen of the animals inoculated. The relative appearances in the staining, together with the clinical and pathological phenomena of the animals inoculated will enable us to judge correctly of the existence of the disease. Besides, bacteriological diagnosis can be arrived at by cultivation experiments. For each bacillus possesses its own peculiar features. To diagnose it from meningitis in infants puncture the spleen with a hypodermic syringe with antiseptic precautions, or perform lumbar puncture of the cerebro-spinal fluid in order to obtain the fluid for the microscopic detection of the plague bacillus.

This method of diagnosis is essential, as there are no suitable glandular enlargements in infants who suffer with plague and they die with symptoms of meningitis.

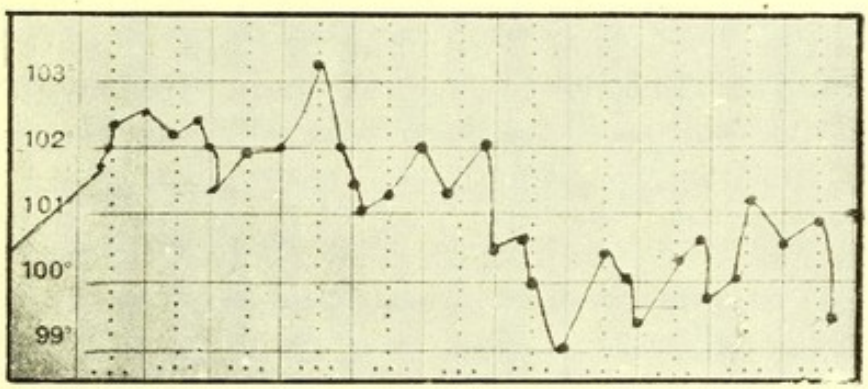
It appears in diagnosing this disease that Widal's reaction is not essential. According to the researches of several bacteriologists, it is said that this reaction is not well marked during the first stage of the disease, or in mild cases.

According to Zabolotny's investigations, there is no reaction during the first two weeks. It first appears during the third week, and presents a well-marked agglutinative reaction. Considering also that other investigators have confirmed the above results, the serum diagnosis by agglutination appears to be of little value in the diagnosis of this disease.

Besides, there is very great difficulty experienced when conducting the agglutinative reaction, as the plague bacillus produces clouding of the Bouillon media, and is non-motile.

KENNEDYTOWN HOSPITAL.

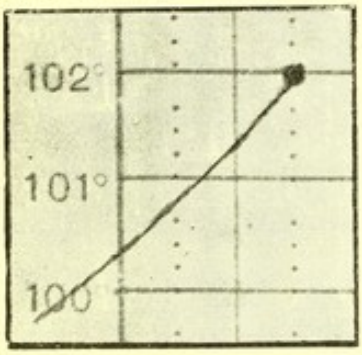
FIG. 28.—T.W.C., age 50; male. Bubonic. Chinese.
5th May, 1896.



Died on 15th May, 1896, at 10.45 p.m.

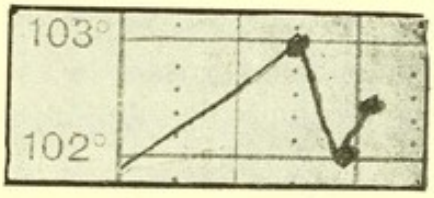
KENNEDYTOWN HOSPITAL.

FIG. 29.—X.Z., age about 22; male.
Bubonic. Chinese.
14th May, 1896.



Died on 14th May, 1896, at 11.10 p.m.

FIG. 30.—C.T., age 24; male.
Bubonic. Chinese.
14th May, 1896.



Died on 15th May, 1896, at 12.45 a.m.

CHAPTER XIV.

THE PROPHYLAXIS OF PLAGUE.

Treatises on the remedy for plague are divided into preventive and remedial measures.

No. 1. THE PREVENTIVE MEASURES.

The preventive measures are of two kinds—the national or public, and the individual or personal.

A. National Prevention.

To prevent the invasion of plague or to exterminate the disease when once it has obtained a foothold is of the utmost importance. Also, it is of the utmost importance to encourage such national preventive measures. In tracing the history of plague, it is obvious that the malignant epidemic has often been prevented through the administration of proper preventive measures. One example may be cited. In 1815, when the epidemic had broken out in the port of Noya, in the province of Bari, in lower Italy, military patrols were posted around the town. A double trench was made around the place. A bridge was constructed over the trenches that could be removed when not in use; and thus the place was isolated entirely, with the exception of allowing the daily necessaries to pass in. The only articles that were allowed to pass out were letters which were dipped in vinegar before being delivered. Guns were mounted at the entrance to the town, and soldiers guarded the trenches around. Martial law was proclaimed, and anyone approaching the entrance or trenches was liable to be shot. One hundred and ninety-two houses were burned down by the authorities. Such strict measures as these prevented the epidemic from spreading into the whole of lower Italy, or even into the whole of Europe. In 1879 the Russian Government had blockaded the Astrakhan district with soldiers when the epidemic prevailed. A second military cordon was posted around the district. A third set of blockading lines was also posted around the villages and streets. The only communication allowed was by telegram. Such astringent measures had also a salutary effect, and the epidemic did not spread.

National preventive measures are the quarantine of vessels, the inspection of trains, and isolation (if fresh line). In order to combat the disease, cleansing, fumigation, rat extermination, the supervision and inspection of residents are to be carried out in an isolated district.

B. The Quarantine of Vessels.

All vessels from an infected, or via an infected port, must be quarantined whether the incubation period has expired or not. Inspect the vessel in a suitable place outside the harbour, and land the passengers and their effects in the quarantine station. Having fumigated the wearing apparel and the personal effects of the passengers, to whom a bath must be given, place the passengers in an isolated room for

seven days, during which time necessary supervision must be made. At the end of the prescribed time, if the passengers are found to be healthy, then pratique is to be granted. Send the health officer on board to look for any dead rats.

Even in the event of the passengers being healthy, and no dead rat being found, the cargo must not be permitted to land. If necessity arises for landing the cargo, it must be subjected to strict fumigation. When a vessel has been granted pratique as described, and intends leaving for another port, it need not be subjected to similar treatment again when arriving at the next port. Perfectly equipped modern quarantine stations must be established in various ports in order to efficiently disinfect the passengers' wearing apparel and their luggage. Affairs must be conducted according to the measures adopted by the temporary epidemic inspection bureau of the Army Department in 1894 to '95, as during the time of the China war, and all vessels bound from plague-infected ports must be subject to strict quarantine regulations.

2. Inspection of Trains.

A complete quarantine of trains cannot be effectively tarried out. However, in case an infected patient is discovered in a place, it is best to proclaim such a place infected, and isolate it.

3. Isolation.

If isolation is properly conducted it is not difficult to prevent the disease from spreading. But effective isolation is not only to prevent traffic and the communication of human beings alone ; the traffic of rats must also be prevented. So that the area of isolation will consequently be wider, and the expense heavy. By every possible means of diagnosis it is better to ascertain the number of patients affected at the outset, and carry out strict isolation on the spot. A net must be drawn around the infected house and its neighborhood to prevent the rat traffic ; and rat hunting must be instituted. The expense of the residents in isolation must be borne by the public fund or the national treasury. Complete fumigation must be carried out in the district that has become infected.

Steps must be taken to burn down the interior of the house ; for the germs of the disease will be diffused by rats in the dust, ceiling, and in the rooms, and underground. Such steps, as are taken in the fumigation of cholera or dysenteric houses, are not effectual. Then measures ought to be con-

sidered for destroying or burning the houses. The houses within the isolated area ought to be visited, and the residents examined for fresh infection.

The symptoms to be observed during inspection when visiting are as follows :—

1. The particular kind of countenance.
2. Whether there is swelling of the glands in the armpit, thigh, femoral region, neck, or under the jaw ; and, if any, whether they are painful to pressure or not.
3. Whether the temperature is febrile or not.

In examining enlarged glands, medical men must use both hands when there are any swellings in these three parts, viz., the centre of the armpit, the side of the chest, and the sides of the arm. The persons examined ought to hold up their arms vertically. Press those parts, and observe if any pain is felt, taking care to see if any signs of pain are visible in their faces. In examining the inguinal and femoral regions it is necessary that they lie on their back, but if these regions are swollen and painful they will feel pain when walking and going upstairs. Make the persons examined walk or go up three or four steps, about two feet each, and watch their movements. When examining, if a doubtful case is tound, conduct bacteriological examinations of the contents fof the glands, secretions, spleen or cerebro-spinal fluid as mentioned in the chapter on diagnosis.

After the inspection medical men are requested to sign the visiting inspection book so as to be responsible.

It is of great importance to exterminate the rats in places where plague has broken out. There is as yet not any complete system in rat hunting, but arsenic and phosphorus can be used under supervision. Loffler's bacillus typhi murium can be used with less danger. A culture is mixed with food. This bacillus is quite harmless to human beings. If one mouse or rat becomes infected and dies, an epidemic will prevail amongst them. A whole tribe of rodents in the infected area may thus be destroyed. It has been used with good results in Thessaly.* If a dead rat is found send it to a recognised laboratory for examination, so as to determine the existence or otherwise of the plague bacillus. If the plague bacillus is found, isolate the place where the dead rat was found. Steps must be taken to cleanse the houses, and to commence rat hunting. In sending a dead rat wrap

* Various rat-destroying cultures of bacilli have been isolated by different investigators ; vide Part II. (D. MacD.)

it up in a cloth that has been saturated in some germicidal fluid. Care must be taken to prevent vermin from escaping into its surroundings. Make a post mortem examination of the rat, and make cultivations from the spleen and blood of the heart. If it is infected with plague, the plague bacilli will be detected with the microscope. When the rat has undergone putrefaction so as to contain the bacilli of putrefaction in the blood of the heart and in the spleen which makes its diagnosis impossible, dissolve the contents of the spleen or the blood of the heart in distilled water, and inject it into the abdomen of Nankin rats (tame rats). If the plague bacillus is present, the Nankin rat will die within 24 hours; and an analysis of the corpse will determine whether the bacillus is positively present or not.

Observe that in the organs of decayed rats there are other bacilli having similar characteristics to the plague bacillus. In case there is any difficulty in recognising it, then conduct experiments on animals as already described. Also, the plague bacillus in a putrid rat often loses its virulence or dies owing to the symbiosis of the bacilli of putrefaction, and sometimes the experimental infection of animals is ineffectual.

"Notices to householders," signed by John J. Francis, dated 24th May, 1894, and the regulations adopted at a special meeting of the Board of Health with reference to cleansing and fumigation on the 31st May, 1894, signed Edward A. Lamb as the proclamation of the Hong Kong Government during the epidemic of plague in 1894, will be found in the proceedings of that colony.

*The following is the method of dealing with outbreaks of bubonic fever (plague) by Dr. Francis W. Clark, Medical Officer of Health, Hong Kong, dated June, 1900.

European cases are reported and are treated by registered medical practitioners, and are only removed to the hospital at the request of the medical attendant. The following method of procedure applies practically to Asiatics only.

1. NOTIFICATION.—All cases of infectious disease should be reported at once to the nearest police station or to the Sanitary Board, or to the Medical Officer of Health. This is compulsory on "all persons knowing or having reason to believe that any person has been attacked by or is suffering from" bubonic plague, cholera, or small-pox (bye-law 17, Ordinance 15 of 1894); but it is universally evaded by the Chinese and even by the Chinese "doctors."

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* Dr. MacD.

The penalty for its evasion is $25.

2. Detection of the Sick.—In the absence of notification this can only be affected by means of house to house visits. The Sanitary Board has power to institute such house to house visits in any district in which the disease may prevail, and must define the limits of such district (Bye-law 25, Ordinance 15 of 1894).

The City of Victoria has been declared infected, and four European officers of the Board, with two Chinese constables, are at present engaged in house to house visiting in No. 2 Health District, while a Chinese doctor trained in Western medicine is making similar visits to No. 1 Health District, and two European constables and a Chinese constable are making visits in No. 9 Health District. Their hours of duty are from 5-8 a.m. and 2-5 pm., and a copy of the instructions which have been issued to these officers is attached. The villages on the Kowloon Peninsula have also been declared infected, and six European officers, with three Chinese constables are engaged in house to house visits in Yaumati, Mongkoktsui, Taikoktsui, and Hunghom.

3. Removal of the Sick.—Ambulances for the removal of the sick are kept at the various police stations, at the Canton Wharf, and at the Board's matsheds at Praya East, Taipingshan, Yaumati, and Hunghom, and sick persons are removed in these ambulances to the Tung Wah Hospital on the application to the Sanitary Board, the Medical Officer of Health, or the police, and are there examined by a Chinese doctor trained in Western medicine, and are either drafted at once by him to the Plague Hospital or detained under observation in case of doubt. In making any such application, care should be taken to state distinctly whether the patient is alive or dead, for in the case of dead bodies, a dead-box is forwarded for the removal. The ambulance is attended by a Chinese constable, who conveys the details concerning the case to the hospital authorities.

Heavy wooden boxes, with rubber washers fitted to the lids, are used for the removal of dead bodies to the Government Mortuary ; these are kept at the various police stations and at the Board's matsheds and information concerning the death is forwarded on a card attached to the body.

4. Treatment of the Sick.—This is entirely in the hands of the Medical Department. The Government Hospital at Kennedy Town is supplemented by a series of Matshed Hospitals, also at Kennedy Town, which are managed by the

authorities of the Tung Wah Hospital, but are under the supervision of the Medical Department.

5. Disinfection of Infected Premises.—This is carried out by a European officer, assisted by eight coloured foremen, a Chinese forman, and a varying number of coolies. As soon as it is known that a case of the disease has occurred at any house, a Chinese constable is sent from the nearest police station to detain all persons found therein (Bye-law 22, Ordinance 15 of 1894), and the officer in charge of the disinfection proceeds to the house to ascertain how many persons are detained there. He then procures, either from the matshed at Praya East or from the disinfecting station, as many suits of Government clothing as are needed for the persons so detained, and, having thus provided these persons with clothing, he removes their own clothing, bedding, curtains, and carpets, to the Steam Disinfecting Station, the clothing being tied up in sheets dipped in a soltion of Jeyes' fluid and conveyed through the streets in baskets; persons who are able to obtain new or clean clothing from some uninfected premises are, however, not detained after they have discarded their infected clothing and handed it to the Inspector for Disinfection. New goods, silk clothing which has not been recently worn, furs, and leather goods are not removed to the Steam Disinfector, but must as a general rule remain on the premises till they have been fumigated. When the clothing, &c., is returned (in the course of some two hours) from the disinfecting station, the persons who have been detained are required to put on their own clothing, and must then leave the premises for some 5 or 6 hours while it is disinfected and cleansed. The Government clothing is returned to the disinfecting station to be steamed before it is again used. The people so displaced from their homes are at liberty to make use of the Board's matshed shelters until the processes of disinfection of the premises are complete.

The disinfection of the premises consists in the spraying of the walls with a solution of perchloride of mercury (1 in 1,000) or fumigation with free chlorine obtained by the addftion of diluted sulphuric acid to chlorinated lime (1 quart of a 1 in 8 solution of the acid to each lb. of the chlorinated lime. Floors and furniture are then scrubbed with solution of Jeyes' fluid, and the walls are then lime-washed, chlorinated lime being added to the lime-wash in the proportion of ½ lb. to the gallon.

6. Burial of the Dead.—This is carried out under the superintendence of one of the Board's Officers, all bodies

being buried at the Kennedy Town Plague Cemetery, unless a special permit has been granted for burial elsewhere.

7. General Sanitary Precautions.—Chlorinated lime is supplied to all the public latrines for use in the bucket, and the officers of the Board are instructed to see that it is freely used.

A reward of two cents. per head has been offered since January 16th for every rat brought to an officer of the Board, and some 25,000 rats have by this means been collected and destroyed.

On the 1st of January, 1899, a report was read before the meeting held to discuss the prevention of plague in Samarkand, in Russia, and the following were the preventive measures adopted by the Russian Government to exterminate plague during the Samarkand outbreak, and to be proclaimed to the public :—

They isolated the village called Anzob in the Samarkand District.

1. 56 physicians and 15 nurses and 46 epidemic inspectors were dispatched from St. Petersburg and Kiev.

2. Three doctors who had studied the disease in India, nurses, and experienced fumigators were also dispatched.

3. 25 bood (1 bood = 16,380 Kilos.) of germicidal chemicals and fumigating apparatus were exported.

4. 2,500 doz. of Drs. Haffkine and Yersin's vaccine were ordered.

5. Orders were issued to all the physicians in the principal towns to be in readiness to start for the infected district when called upon to do so.

6. Soldiers were sent out to assist in all the operations.

7. Epidemic inspection officials were established along the roads between the infected district and the other principal towns.

8. The inhabitants of Anzob have been provided with provisions and fuel for the coming winter, and with white seal skins.

9. The department is to aid the orphans of patients.

10. The number of medical men are to be increased on the Transcaspian railway, and to keep surveillance over the passengers and traffic.

Personal Prophylaxis.

Concerning individual precautionary measures care is to be exercised in keeping the body and the dwellings clean. Plenty of light and fresh air must be admitted into rooms. Great care is to be taken so as not to receive any wounds. If

a wound is inflicted, bathe the part directly with 5 per cent. carbolic acid, and use an ointment or collodion. When in haste fat may be plastered over the part. Labourers should wear gloves and socks. Food and drink must be boiled. Use lids over any utensils that contain food or drink.

During an epidemic do not, if possible, visit plague infected houses. As a preventive, inoculation with Dr. Haffkine's vaccine is used. It consists of a Bouillon culture of the plague bacilli which have been heated for one hour at 70° C. Its merits are not yet quite known. Dr. Yersin has also investigated an anti-plague serum injection.

No. 2. Remedial Measures.

The remedies for this disease are simply symptomatic and prophylactic. At the beginning of the epidemic in Hong Kong cathartic remedies, particularly liquorice and alkaline purgatives, were used (Cantlie). It is most essential to administer stimulants and stimulating remedies. Brandy and egg flip is most suitable.

Prof. Aoyama considers that an injection of carbolic acid has been proved to be more or less meritorious when injected into the bubo. Prof. Yamakime commends early extraction of the affected gland.

According to Shiga's investigations in the North of Formosa last year, early extraction of the gland presents very favorable results. The mortality was reduced to 50%.

In the cerebral type ice, cold water applications and cold baths should be administered, For sleeplessness, a 0·0006 gm. (gr. $\frac{1}{100}$) hypodermic injection of hyoscine is very good (Cantlie). Mustard sinapisms to the epigastrium and also ice to suck are used to allay vomiting.

When the bubo maturates, incise it, and as widely as possible.

Dr. Yersin has experimented with his anti-plague serum in Hong Kong and Amoy. Active cultures were used. A small quantity of the bacilli were injected into a horse, gradually increasing the dose until immunity was established ; then the blood was drawn off. This serum was first tried on twenty-three persons in Amoy, and on three in Canton ; none of them died, and the results were considered as favorable. Those who were experimented on in Amoy resulted in a mortality of 7·6%. The quantity of serum used was 20 to 90 cubic centimetres, which was administered when divided into small doses. The earlier the injection the better were the results. He afterwards experimented upon 50 persons with serum obtained from the horse in Cochin China which had

been rendered immune by repeated injections of sterilised cultures of the plague bacilli, resulting in a mortality of 50%.

The mortality on the first day after the injection was						12%.
"	"	"	second	"	"	35%.
"	"	"	third	"	"	50%.
"	"	"	fourth	"	"	66%.
"	"	"	fifth	"	"	100%.

A greater quantity of the serum is required if the injection is given late on in the disease.

Supposing 30 cubic centimetres are required on the first day, it will require four or five times of that quantity on the second day.

According to the Russian Commissioners serum therapy is of no value in pneumonic plague. The Austrian Bacteriological Commissioners also assert that serum therapy is ineffectual.

On the whole, although the remedial measures of plague are of two kinds, prophylaxis and sera-therapeutics, yet at any rate the early extraction of the gland is one of the most essential remedial procedures, as it exterminates the very incubator of infection.

The opinion that gangrene or septicæmia might ensue through the operation of extraction is, I think, groundless. Of course, the operation must be performed with every possible care to avoid such ; and every antiseptic precaution must be taken.

On Practical Nursing.

The treatment of these patients do not differ from those in other general epidemics, but there are some points to be observed and regarded in this disease.

1. It is of utmost importance to renew and to fumigate the bed clothing and wearing apparel, as the infection is disseminated throughout the bubonic discharges, the fæces, urine, sputum, tears, saliva, and all such secretions and excretions. It is diffused on wearing apparel and bed clothes. Infection still exists during the convalescent stage, hence convalescent patients are still infectious, and this must not be overlooked.

2. There is the infection through flies, fleas, and mosquitoes. Use mosquito nets ; pay particular attention to the advent of these insects.

3. Infection can be transmitted through a very tiny puncture or wound, such as mosquito or flea bites ; such as the naked eye cannot discern. Be not careless of any tiny wound or abrasion. Submerge the hands in some antiseptic

fluid whenever they touch the patient's body, their secretions, or wearing apparel.

Nurses must attend the patients in rotation during fixed hours. They must not sleep and remain in the same room with the patients.

They must, of course, wear caps, germicidal garments, stockings, and slippers when on duty. They must change their entire wearing apparel, and all, besides their germicidal garments. All must be fumigated when off duty.

The exposed parts of the body must be bathed with a 1:1000 perchloride solution for germicidal purposes. Have a bath then, and retire, wearing fresh garments that have been fumigated.

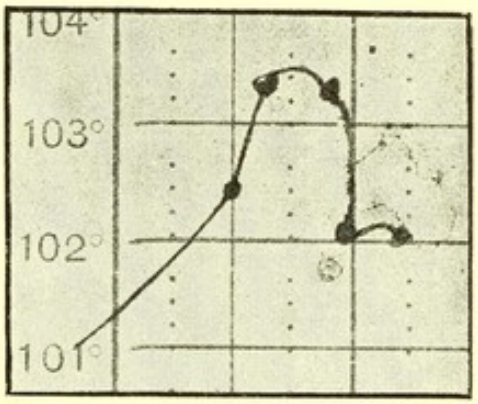
They must not eat, drink, or smoke* in the patient's ward.

The nurses, whilst attending to plague patients, must not be allowed under any circumstances to enter into other wards where other patients are kept.

All who approach pneumonic plague patients must wear respirators, or else cover their mouths and nostrils with cotton wool. A pair of spectacles must also be worn.

KENNEDYTOWN HOSPITAL.

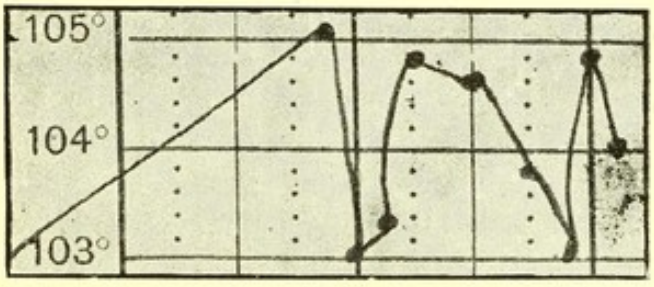
FIG. 31.—M.S.Y., age 16; male. Bubonic. Chinese.
14th May, 1896.



Died on 15th May, 1896, at 4.15 a.m.

KENNEDYTOWN HOSPITAL.

FIG. 32.—N.L F., age 30; male. Bubonic. Chinese.
4th May, 1896.



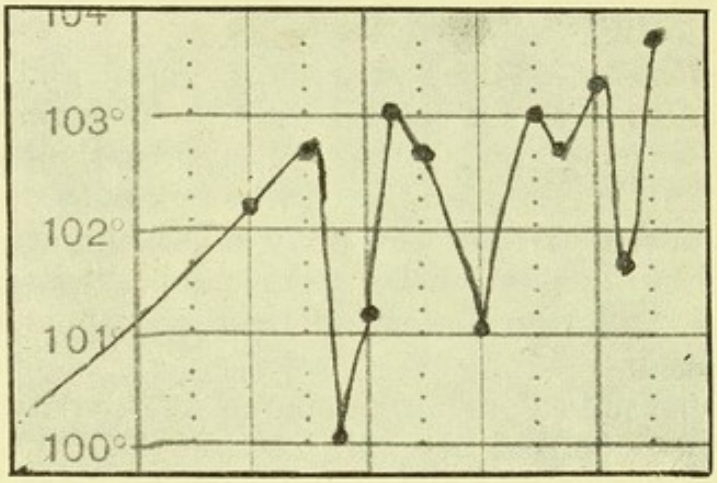
Died on 6th May, 1896, at 6 a.m.

Calomel.

* Male nurses are also employed in Japan (D. MacD.)

KENNEDYTOWN HOSPITAL.

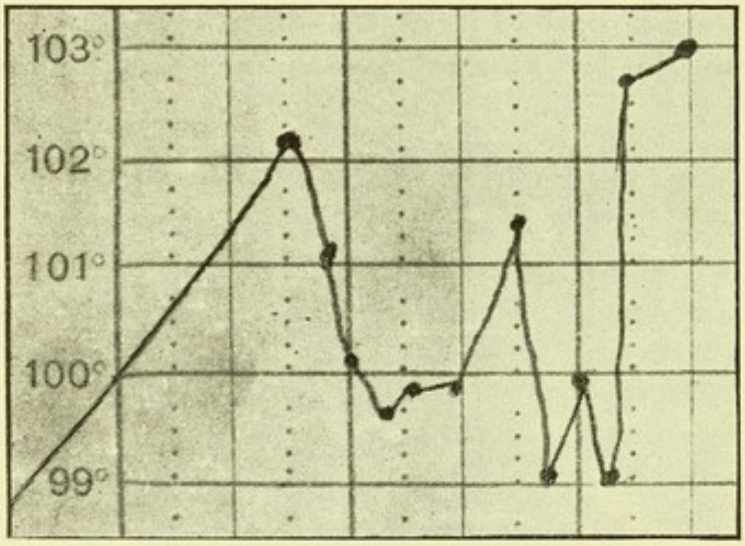
FIG. 33.—O.S., age 24 ; male. Femoral Bubonic. Chinese.
5th May, 1896.



Died on 7th May, 1896, at 9.30 a.m.

KENNEDYTOWN HOSPITAL.

FIG. 34.—S.K., age 24 ; male. Bubonic. Chinese.
12th April, 1896.



Died on 14th May, 1896, at 1.45 p.m

CHAPTER XV.

THE EPIDEMIOLOGY OF PLAGUE IN JAPAN.

Quite recently plague patients have sometimes been discovered amongst the passengers of vessels which had called at an infected port and which were bound for Nagasaki, Kobe, or Yokohama. However, a case of this disease was never discovered on land until the fifteenth of November, 1899, when news from Hiroshima heralded a true case of plague

in the death of Matsugoro Sawada, aged 23, a resident of Yokohama, at an hotel in Hiroshima. This was the first case of plague that was ever heard of in Japan. This man had left Ke-lung, in Formosa, on board the "Omimaru." He left the steamer at Moji, went on to Tokuyama, and took the train to Hiroshima. This being the first case that had ever happened in Japan, the news stirred the public; and the authorities began to move in the preparation of precautionary measures. At this time another case of plague was discovered in the person of K. Yamamoto, age 13, in the employ of a rice and timber merchant living in a place called Fukiai, the eastern extremity of the port of Kobe. This lad had had a swelling in the inner right femoral region since the sixth of November which gave him considerable pain, and he became feverish. He died on the 8th of November. The doctor who attended the lad reported him as a doubtful case of plague.

The examination proved the presence of the plague bacillus in the glandular swellings, and in the organs. This was the first case in Kobe, and the second that happened in Japan.

The second case of plague in Kobe ending fatally was a man named M. Hirai, age 32, a cart driver who lived near the house of the previous patient (No. 1). His left femoral gland was swollen. His temperature had been rising since the third of November, and he died on the 11th. The presence of the plague bacillus was proven in this case. The third case was a female, O. Moto Shimozi, age 34, a cotton operator who lived in the same district. She had a small purulent sore, like a carbuncle on the outside of the chest in the left mammary region since the beginning of November, which had broken. On the eighth of November a painful swelling or bubo appeared in the left axillary region. The whole of this part of the body became inflamed, and she died on the 11th. The plague bacillus had been found in the gland, and in the purulent matter.

These three cases already referred to all lived in the same district—Tukiai—of the city. At the time the disease appeared to be confined to that portion of the city. However, on the 11th November another patient was discovered in a place quite remote; and, as the list will show, the disease had spread and disseminated all over the city of Kobe. The total number of patients affected were 33, and the epidemic ceased at the end of December.

As to the origin of the disease in the eastern extremity of the city of Kobe-Tukiai, there is evidently no cause explaining how infection had taken place. But a careful enquiry into

the matter presents the following hypothesis that the first patient, K. Yamamoto, was not actually the first as was supposed. There were two doubtful cases in the district prior to his death. One of them was Y. Adachi, age 13, employed as a dealer in " ships' refuse," who had been ailing since the 19th October, and died on the 25th of the same month. The other one was K. Kawai, age 48 years, employed as a dealer in the same business, " ships' refuse," in close proximity. He had died on the 4th November after having been ailing since the 30th December.

Although both those cases died with femoral and inguinal buboes after a febrile condition, yet the cause of death was not mentioned on the death cetificate. But as there was no suspicion of suicide or foul play attached to them, they were allowed to be cremated. The reports we had received from the parties who had personally known the deceased were sufficient to enable us to form the opinion that they died of plague. Not only so, but the subsequent patients had a certain relation to them, as regards the method of infection. If these two cases were actually true cases of plague, as they appear to be, then the invasion of plague to the Fukiai portion of the city of Kobe must have taken place in the end of October. It was the lack of practical medical information on this disease that had prevented us from discovering it, and upon the receipt of the telegram from Hiroshima, which specially arrested the attention of medical men as well as the public, we were enabled to diagnose K. Yamamoto by scientific methods on the first plague case.

A similar incident may be recalled during the epidemic in Oporto, at the beginning of which the patients were being treated for fevers of unknown origin. (Curiously enough similar instances happened later in Glasgow, Great Britain, and Australia.—D. MacD.)

To investigate how and when the infection was introduced into Kobe were important questions, consequently the authorities at Hiogo and Prof. Kitasato had carried on an exhaustive enquiry from the commencement. Afterwards Professors Ogata and Nakahara came over to Kobe to investigate it.

Their opinions differ.

It is believed by Prof. Nakahara that the infection was introduced on different occasions through patients who had not been discovered, and through rats and cargo from Formosa, where plague is endemic, and not through the " ships' refuse," which is gathered and received by the Kioyeki Com-

pany who land it, nor through the medium of cotton imported from Bombay.

However, Kitasato inclines to the belief that the cause of infection was the "ships' refuse" which was landed. Thus the bacilli found their way amongst them. He asserts that vessels which arrive from Bombay and other infected ports carry away with them infected rats which infect others, and their secretions containing the plague bacilli will be mixed with the dust and refuse on board the vessels. I agree and concur with the opinion of the professor. The infection no doubt has been introduced through "ships' refuse," and the following hypothetical cases may be cited.

There are three dealers in ship's refuse in Fukiai. Of these there are the two employers of the two suspected cases which had been cremated as the unknown disease already referred to (Y. Adachi, 13 years old, and K. Kawai, 48 years old). They carry home on carts what they buy, and sort it out for sale at their houses.

The so-called "ships' refuse" consists of rags, old cotton, sweepings of rice and grain that is generally found in the bottom of the ship's hold, wherein the bacillus has a suitable media, where the secretions of infected rats and insects are apt to collect.

The bacilli attached to the cargo under ordinary circumstances may have been inert before some port was reached; but the bacilli mixing with the dust containing the excretions of rats at the bottom of the ship's hold will live for a long time.

The relations of the first and the other two unknown cases as regards the ship's refuse are as follows :—

1. The unknown suspected case of Y. Adachi was an employee of one of the dealers, and was daily occupied in carting the ship's refuse and assorting it. His illness began on the 19th October, and he died on the 25th of October.

2. The other unknown suspected case, K. Kawai, was also employed by a dealer in the same line as the other, carting and assorting the refuse. He became ill on the 30th October, and died on the 4th of November.

3. The first patient's house, K. Yamamoto's, is separated from the first employee's house (Y. Adachi) by a narrow road. The two houses are almost next door to each other. He often went to the latter's house, and played in the refuse. He was a thirteen-year-old boy.

4. The second patient, M. Hirai, lived nearly opposite to the house occupied by the employer of K. Kawai (the second

suspected case). The store where this employer kept his refuse is two or three doors from where the patient Hirai lived.

He was often employed in carting the refuse as his occupation was a cart driver.

5. The third patient, O. Mato Shimozi, lived at a comparative distance from the others. On the 19th October she bought $40 worth of refuse cotton from Y. Adachi's employer which was gathered from the S.S. "Kagoshimamaru," and she had beaten the cotton. She was a cotton operator.

Her illness began in the beginning of November. The S.S. "Kagoshimamaru" left Bombay on the 10th September, and arrived in Kobe on the 7th October.

Of course, after a few patients are discovered during an epidemic, the infection will disseminate through several agencies. The ætiology of the infection of the other cases cannot, of course, be traced, nor is it a matter of surprise to see the epidemic break out in distant parts. The first plague rat was found dead in Kobe in the house of I. Sugawara (the fifth patient).

A List of Plague Patients in Kobe.

Commencement of disease.	Recovery or otherwise.	Days in Hospital.	Nature of disease.	Address.	Occupation.	Name.	Remarks.
1899. 6th Nov.	1899. Died 8th Nov.	0	Right femoral	—	Employed at rice merchant.	Y. Yamamoto. Age, 13.	Lived next door to refuse dealer.
3rd Nov.	Died 11th Nov.	0	Left femoral	—	Cart driver.	M. Hirai	Used to cart the ship's refuse. Two dead rats found near his place! &c.

CHAPTER XVI.

The Epidemiology of Plague in Osaka, Japan.

As soon as it was discovered that plague was presenting epidemic features a Temporary Epidemic Inspection Bureau being established in Osaka, every possible means were adopted and carried out to prevent its introduction. All steamers, vessels, and trains were under the Bureau's surveillance. Throughout the city cleansing and fumigation was instituted. The Board of Health delivered lectures on the subject in various places of the city so as to arouse public attention. Notwithstanding these measures which were being adopted, a

plague patient was discovered in the west section on the 20th November, 1899. This was a girl, age 11 years old, called O. Fusa Hashimoto. On the 18th November she came home from school complaining of headache, a tired feeling, and went to bed. She had rigors, fever, and thirst, but the family, thinking that it was a cold, did not call in the doctor. On the 19th a painful swelling appeared in the right axillary region. A doctor was called in for the first time on the 20th, who reported it as plague. The Inspecting Officer visited the house, and examined the contents of the swollen glands, and found plague bacilli. She was immediately sent away to the plague hospital, and the family were despatched to the isolation station. The house, as well as the neighborhood, the school, and the girls who attended were isolated. The class mates of the girl were kept under inspection by the health authorities. At the same time a bacteriological laboratory was fitted up in a portion of the Temporary Inspection Bureau, theworking of which was entrusted to the medical practitioners who had gone through a special course of study in the Infectious Disease Research Institute. A daily inspection of the entire city and its inhabitants was organised and carried out in conjunction with the Osaka Board of Health. On the 29th November a telegram reached Osaka from Wakayama announcing the death of a plague case, a nurse girl, O Kiwa Yamasaki, age 17 years. She was nursing a child called Hidetane Yamada in Osaka (who afterwards died of plague) until the 27th, when she left her employer on account of illness. She took a steamer on the 28th for her home in Wakayama at 4 p.m., and she died on board the steamer at 1 a.m. the next morning. It is said that she had gone to the closet three or four times as if she had diarrhœa. The corpse was that of a well nourished girl, and it is reported that she had a swelling in the armpit the size of a duck's egg. The people that saw her off affirmed that, although she walked on board the steamer with a steady gait, she presented at the time of embarkation an appearance of more or less marked prostration. There is some suspicion that she died 9 hours after embarkation. Seeing that the day when she left Osaka the weather was rough and stormy, septicæmia may have set in through the tossing of the boat, resulting in paralysis of the heart. Experimentation and other circmustances appear to confirm this conjecture. Death is greatly accelerated in plague by the movements of the body.

On the 30th November another patient was found, called M. Ozaki, employer of the nurse girl. This was the third

patient. He died after being admitted into the plague hospital on the 4th December. His internal femoral and inguinal glands were enlarged. The child whom the second patient, O. Kiwa, nursed became infected on the third of December. (The symptoms and details of this child will be especially discussed in the chapter on intestinal plague.)

Then this epidemic disseminated to other parts of the city. It invaded the Linen Wearing Company at the end of December, and produced there a sad state of affairs—several cases being of pneumonia plague, the details of which will be described in another part. The epidemic ceased to exist in the middle of January, 1900. The total number of cases infected were 44.

The ætiology of this outbreak could not be traced in Osaka, which is a lamentable fact, although vigorous measures had been adopted.

The father of the first patient, O. Fusa Hashimoto, is an operator in the Heian Cotton Company of Kyoto. The girl visited him before the infection, and stayed at Saito about 40 days. She came home to Osaka five or six days prior to her illness. The Inspecting Medical Officer was sent out to the Cotton Co. to make investigations with a view to see if the girl's infection had been caused through the Cotton Company's imports. An investigation revealed the fact that not only was the cotton used of American and Egyptian manufacture, but that they had not recently imported any cotton from an infected district such as Bombay. In point of fact, the girl had never come in contact with the cotton. She only now and then brought her father's lunch to the factory when she was in Kyoto.

It is evident that the infection did not take place in Kyoto, seeing that nobody connected with the company became infected, nor had any suspicious case happened in the neighbourhood, nor were any dead rats discovered.

The grandfather, with whom the girl lived in Osaka, is a carrier of imported sugar from other districts. Although there is a possibility of infection through her grandfather's wearing apparel which came in contact with the sugar bags, still the hypothesis is not valid, as none of the carriers in this line of business had been infected. It is true, moreover, that two sons of those carriers were infected, but only afterwards.

These cases were affected when the epidemic had taken a good foothold in the neighbourhood of the patients affected.

The family of Jimbei Yamada furnished three cases of plague. He is a confectioner by trade, and also deals in

waste cotton. The waste cotton is such as the combings from the Cotton Company's machinery, and those which come from broken bales of cotton, imported from foreign countries, also such soiled cotton as that which may, during transport, become detached from broken bales. He preserved this waste cotton in a shed. Although no dead rats were discovered there, still some were found right opposite amongst waste cotton in the neighborhood. Those dead rats contained plague bacilli. It will be correct to conjecture that at this time the rats around Jimbei Yamada's place were already infected. It was on the 2nd December that the plague bacilli were positively demonstrated in the rats.

A relative of *the first patient* lives next door to this Yamada. The *girl, O. Fusa, the first patient*, often went to this relative, and visited the Yamada family through the gate where those waste cotton goods were stored, and she had often played with the nurse girl, O. Kiwa, the second patient. *The third patient, M. Ozaki*, being Yamada's employee, used to go to the store usually barefooted, or employed himself in cleaning the yard around the store. *The fourth patient*, a child two years old, grandson of Jimbei Yamada, was the child whom the second patient nursed. There is a strong presumption that the first and second patients were infected through the upper limbs. Both had buboes in the axillary region. The third case was through the lower limb. The internal femoral and inguinal glands were swollen.

The fourth appeared as if infection had taken place through the alimentary system, seeing that the intestines and brain membranes were chiefly affected.

It may, therefore, be concluded that the introduction of infection to Yamada's store came through the medium of waste cotton, especially that variety which comes from vessels.

A good opportunity was afforded during this epidemic of witnessing the acute stage of pneumonic plague. On the 18th December, 1899, an operator, O. Masa Adachi, belonging to the Linen Wearing Company, became ill. The symptoms were those of acute pneumonia. She died on the 21st. The doctor had diagnosed it as a case of pneumonia. Tracing its after-effects this case was undoubtedly one of pure and simple pneumonic plague.

Her father, Rehei Adachi, and her mother, O. Fuji, died from pneumonic plague through this suspicious case. Four or five operators, the mates of O. Masa, were next affected. Pneumonic plague then proclaimed itself in O. Masa's brother,

Yasaburo, her sister O. Ume, and her lodger Koyoshi. Dr. Wakabayashi the physician who attended O. Masa's parents. next became affected. Dr. Baba, the medical officer who often visited the family, suffered from pneumonic plague almost about the same time and died. The infection was transmitted to his wife, O. Také from Dr. Baba. From Dr. Wakabayashi, Dr. Yamanaka, and Mrs. Wakabayashi, and her male servant were all infected with pneumonic plague. They all died. The infection was next transmitted from Dr. Yamanaka to his wife, Kameko, and his mother.

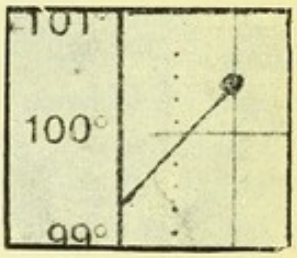
Thus the virulence of the infection can be imagined. Attendant physicians and nurses are very liable to succumb to pneumonic plague, as was observed in the death of Dr. Müller, the Austrian member of the Plague Commission, his attendant, and nurse. The superintendent of the Plague Hospital in Formosa, his medical assistant, and nurse all died from pneumonic plague.

KENNEDYTOWN HOSPITAL.

FIG. 35.—L.F., age 36; male. Bubonic. Chinese.
Admitted at 7.45 p.m.
28th May, 1896. 9th.

Calomel.
Discharged on 24th June, 1896.

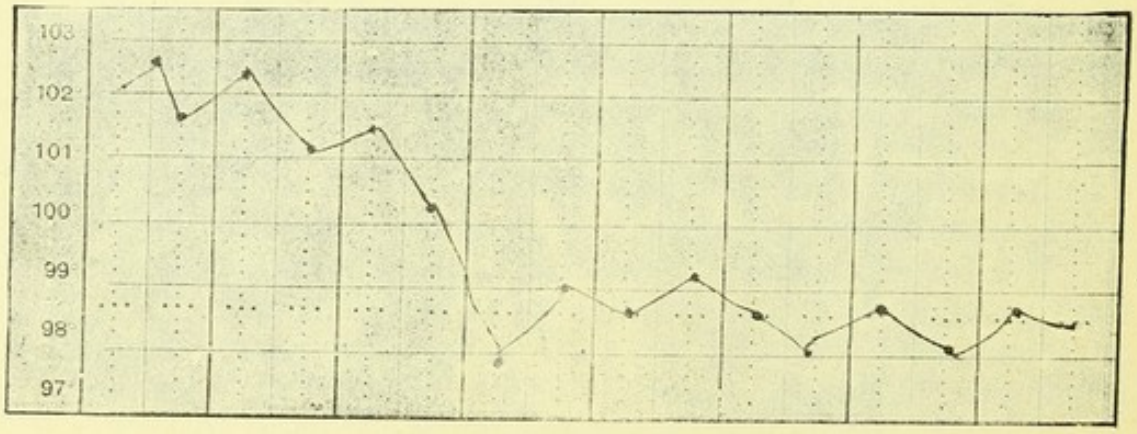
FIG. 36.—T.Y.C., age 61; female. Bubonic. Chinese.
22nd May, 1896.
Admitted at 10 a.m.



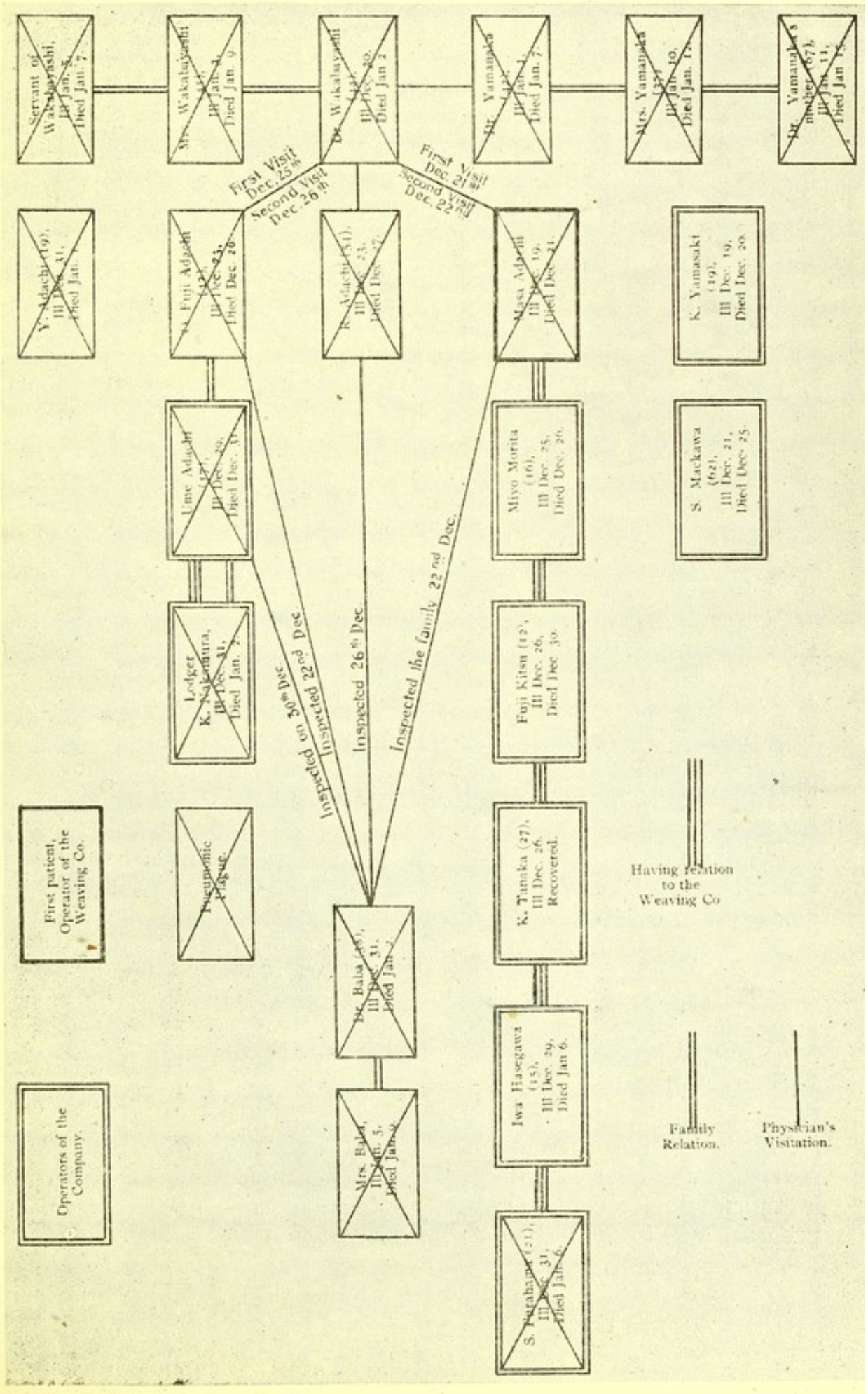
Died on 22nd May, 1896, at 1.30 p.m.

KENNEDYTOWN HOSPITAL.

FIG. 37.—T.K., age —; male- Bubonic. Chinese.
12th September, 1896. 19th.



The epidemiology of pneumonic plague may be tabulated as follows :—



CHAPTER XVII.

Infective Cutaneous Plague.

1. Microscopic Appearances

The characteristics of plague originating in the epidermal and subcutaneous tissues are similar in appearance to splenic carbuncle. A hard nodule circular about eight centimetres in diameter or oblong in shape is formed in the epidermal and subcutaneous tissues, which are elevated a little above the surrounding skin. It has a hard and elevated brawny margin, so that in appearance the central part of the nodule is comparatively depressed. At first the margin presents, as in the case of burns, a vesicular appearance. The contents of the vesicle consist of a reddish yellow exudation which gradually becomes turbid. Later on this watery vesicle will burst and discharge its liquid contents. The scarf skin will shrivel up and desiccate, and also present a dry gangrenous appearance of a surface sore. The vesicular exudate contains an enormous quantity of the plague bacilli. The desiccated scarf skin peels off and the true skin appears. It then has the appearance of an open wound or sore, the surface of which is very characteristic. It presents a mottled appearance, having bright red and yellow points intermingling. It is observed when cutting into it that these are small necrotic ulcers in the cellular and subcutaneous tissues. They are a few centimetres apart, having the appearance of fat, and contain yellowish inspissated purulent matter. There are numerous punctate or linear hæmorrhages in several places. The picture is most characteristic, and the array of colors is exquisite.

This characteristic appearance gradually diminishes toward the margin of the wound presenting only a yellowish colour with œdema.

2. Its Pathology and History.

In studying under the microscope the histology of cutaneous plague, not only are hæmorrhagic areas observed in the epidermical and subcutaneous structures, but there is extensive ulceration of the cellular tissue and round cell infiltration which is of the polynuclear variety as in erysipelatous cellulitis. The plague bacilli are clustered together in several parts of the tissue. The subcutaneous cellular tissue is morphologically disorganised through the inflammatory process, and in some parts the ulcerative process has eroded

and destroyed the structures entirely. The nuclei of the white blood corpuscles are often found free in the purulent secretion undergoing granular degeneration. The sweat ducts and the walls of the capillary blood vessels are disorganised and present, morphologically, occlusion and degeneration which are characteristic appearances. The subcutaneous cellular tissue, around the carbuncle where ulceration is far less marked, has also become morphologically disorganised, and has very fine granular bodies. Each tissue is separated by coagulation necrosis, the necrotic tissue containing a considerable number of granular corpuscles. One of the characteristic features of cutaneous plague is that fibrous tissue cannot be observed notwithstanding the fact that the epidermal structures and subcutaneous tissue are extensively ulcerated and infiltrated with hæmorrhagic material. On the whole the integumentary structures are loculated, and resemble the appearances observed in the skin of small pox and splenic carbuncle. Whilst the vesicle is yet intact a loculation of the epidermal cells like a fan is observed, the interior of the loculi containing liquid.

3. The Symptoms of Cutaneous Plague.

The local symptoms of this variety of plague are described under the microscopic appearances. The constitutional symptoms are similar to those of bubonic plague which begins with an incubation period consisting of rigors, marked prostration, pains in the limbs and in the joints. The temperature rises suddenly. In that part of the skin that feels painful, a tense hard swelling will first appear. A vesicle rapidly develops, the margin of which is very tense and hard. An actual vesicle is formed and extensive lymphangeitis develops in its immediate vicinity. It then goes through the various stages of the disease as in bubonic plague.

There is as yet no accurate statistics of the pathological regional anatomy of the plague carbuncle. According to the Bombay Commissioners one appeared near the umbilicus in one case. In another case one occurred on the inner femoral region.

The investigations in Japan during the epidemic of 1899-1900 are as follows :—

One was present in the abdomen in the right hypochondriac region under the umbilicus (Constable Yase, Kobe) ; one, on the left external mammary region in a female ; two, on the hip in an adult and a female infant. Some idea may be obtained from the clinical history of Constable Yase which is reproduced in

another part. The ætiology of cutaneous plague has not been clearly investigated yet. It may be produced by scratching the skin with infected fingers (the fingers being infected with the bacilli), or it may originate from an abrasion which comes in contact with some media capable of transmitting the bacilli such as wearing apparel. It will be a difficult matter to explain why the bacilli infect an abrasion of the body instead of being absorbed by the lymphatic system and locating themselves in a more suitable locality such as the lympathic glands and producing a bubo, but first to confine themselves to the cutaneous structures: seeing that the bacilli, when inoculated into an animal that has had its hair shaved, are still locally diffused at the point of inoculation producing cutaneous plague. It may be similar in the human being when comparatively large quantities of the bacilli adhere to the abrasion, whilst a portion may be absorbed into the system, but the remainder multiply locally, producing cutaneous plague. On the whole, when cutaneous plague originates in the human being, it is generally where the lymphatics are very sparse; or in a region where, through a change of some kind, absorption is generally obstructed as for example occurs in the healing process. The bacilli may be able to produce plague in this locality when all the necessary requirements are present. Some examples have already been described illustrating why the carbuncle does not originate in those parts which are tender, but which are covered with wearing apparel, boots and stockings causing interference in localization.

There is, as it were, a secondary subcutaneous plague This is due to extensive ulceration around the lymphatic gland which has absorbed the infection from the cutaneous carbuncle. If the ulceration around the bubo is very extensive from the first, precluding the glandular swelling from being felt, it will be very difficult to diagnose the case whether it is a primary or secondary one.

4. The Clinical History of a Cutaneous Plague Patient.

Chiosaburo Yase, age 22 years, constable of Hiogoken.

Commencement of illness, 5th December, 1899, admitted into the hospital on the 9th December.

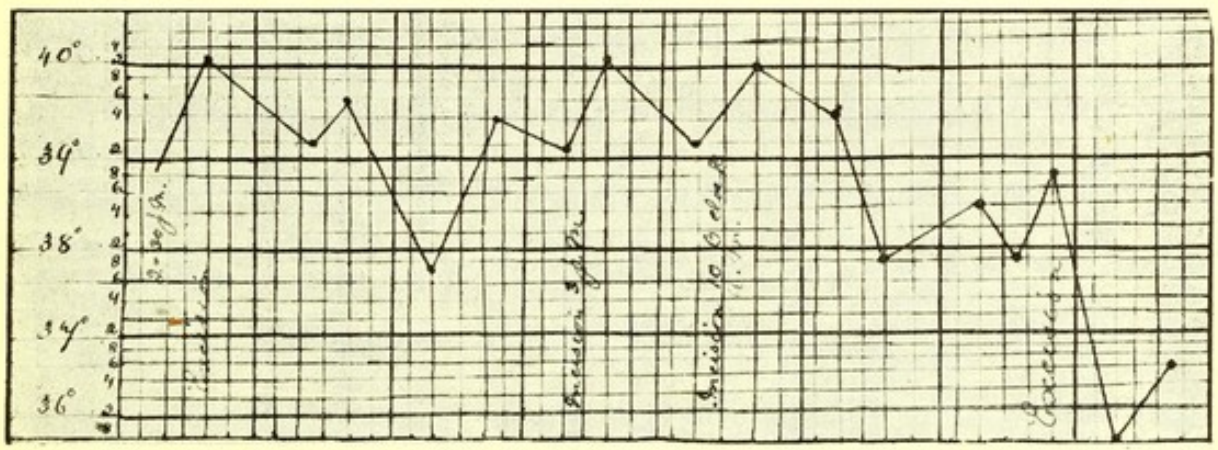
Previous History: He was healthy and strong, and had never been ill before. Since the middle of November, 1899, he was on duty in the Fukiai plague infected district, and attended to the isolation. After the isolation was over he resumed his ordinary duty on the 5th December, 1899.

STATUS PRÆSENS: When he resumed his ordinary duty on the 5th December a nodule the size of an apple appeared in the skin under the umbilicus on the right side of the abdomen. Since 7 p.m. he had felt pain and tenderness. Later on as the nodule increased in size the right inguinal glands became swollen to the size of a pigeon egg. He was feverish, and felt languid and tired. He went to the Kobe Hospital to be examined, and on the 9th December, at 2.30 p.m., he was admitted into the Higashiyama Hospital.

THE SYMPTOMS ON ADMISSION: His body was fairly nourished, being rich in fat. The countenance presented a more or less prostrated appearance, still there was very little hyperæmia of the conjunctive, and no headache; and the tongue was covered with a white fur. The appetite was normal. There were no physical signs in the respiratory and circulatory systems. The spleen was slightly enlarged. Throbbing pain was felt in the carbuncle, which was about the size of the fist under the umbilicus on the right side. There was a tense inflammatory swelling palpable over Poupart's ligament in the right inguinal region. No pain was felt; and in other respects there were no other local symptoms. The temperature was 39·4° C. On the same day five or six portions of the right inguinal bubo were excised at 3 p.m.

FIG. 38.—Chiosaburo Yase, age 22. Cutaneous. Japanese.

9th December, 1899.



10th December, 9 a.m. No alteration in his condition. The appetite is good. The respiratory sounds are impaired. Cough and expectoration are present. The abdomen is tumid all over. The surface of the wound is clean. No signs of ulceration are present. The desiccated part of the carbuncle was excised.

9.30 p.m. Pulse 96. Temperature 38·9° C. The tongue is thickly covered with a white fur, whilst the tip is clean. A worm has been vomited.

11th December. The temperature in the morning 37·9° C. Appetite good. Slept well last night. The pains have disappeared in the affected region. No increase in the abdominal tumidity. The sponging of the body is continued.

9 a.m. No further change. Temperature 39·6° C. Pulse voluminous. Well marked carotid pulsation on the right side. The respiratory sounds are weak ; and cough is present.

12th December. No pain in the surface of the wound, and its margin is not tense, but there is ulcerative erosion of the carbuncle, and the floor of the ulcer is matted.

The abdomen is tense between the primary and secondary bubo, which had been excised at 3 p.m.

13*th December*. Two or three rigors since last night ; none have occurred since. No change in his condition. Temperature 39° C. Pulse 118. Appetite still unimpaired. The physical signs of the right lung remain unaltered. Marked excitability. Hyperæmic conjunctivitis has developed since last night. On pressing the left femoral glands pain is elicited. Made an incision and excised several of the enlarged glands ; since then he feels much better.

14th December, 9 a.m. Condition unaltered. The tongue is white ; hyperæmia of the conjunctival membranes. The appetite is rather poor. A glandular swelling is observed in the right cervical region. There is an absence of the respiratory sound in the upper lobe of the right lung. A glycerine injection to promote evacuation was administered, and with the desired effect. There is no pain in the surface of the wound, but the upper portion exhibits a red blush. A gangrenous bed sore over the sacrum has developed.

15th December, 9 a.m. There is no alteration in his mental condition, but he is a little duller. The white fur on the tongue has cleared up. Appetite is normal. There is no alteration of the respiratory and cardiac sounds. The abdomen is tender all over the tense area. The red blush around the surface of the wound has disappeared, and there is no evidence of its spreading again.

16th December. A little dull mentally, though the condition is unaltered. The appetite, tongue, respiratory and cardiac sounds are as they were yesterday. The progress of the wounds has been favorable. As the glandular swelling

increased in the left cervical region it was excised at 10 a.m. One the size of a large bean, and one a little smaller were extracted from the cervical region. The operation was over at twelve noon. After the operation the temperature was 37·3° C. ; pulse 68 ; respiration 18. As there were symptoms of exhaustion, camphor was ordered to be taken.

17th December. Although inclined to sleep, the condition of the patient is bright and cheerful. No other ominous symptoms have developed. The progress of the surface wounds is good.

18th December. The progress finally is good. The temperature since the 16th has been 37° C. The surface wounds are healing by first intention, and are becoming smaller. By the end of January, 1900, all the symptoms having subsided, the patient recovered. The only remnant was a slight blush on the face.

On the whole this patient did not develop any cerebral symptoms. All through the various stages of the disease he took his nourishment well. A fact worthy of mention is that there was a remission of the temperature after each operation. He always felt better after each operation.

The medicines administered from the commencement were stimulants and alkaline lemonade. Cold bath treatment was not resorted to even for once. The diet consisted of milk, eggs, and soup.

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Fig. 39 (i.)—W.Y., age 53; male. Bubonic. Chinese.

6th May, 1896.

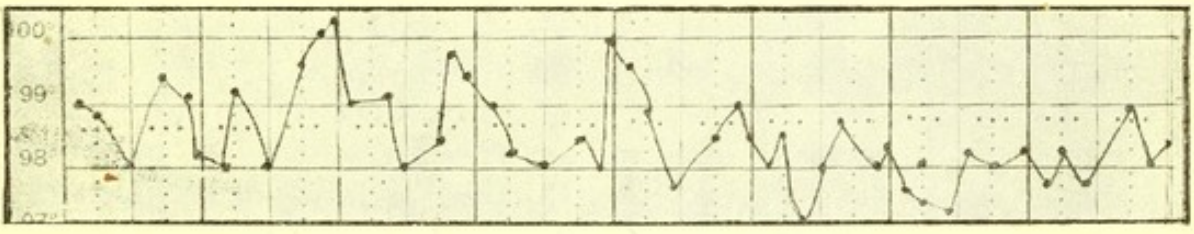


Fig. 39 (ii.)—W.Y. (continued).

14th May. 21st May.

Discharged 6th July, 1896.

CHAPTER XVIII.

Pneumonic Plague.

According to such historians as Hecker, Hirsch, Lucretius, and Davidson there may be during a plague epidemic an affection of the lungs with or without glandular enlargements, accompanied with pain in the chest, cough, hæmoptysis, and hæmorrhagic sputum, that becomes fatal within three or four days.

Dr. Tsugio Horinouchi, who specially studied pneumonic plague while he was in the hospital in North Formosa, has termed the disease *Primary Pneumonic Plague*, and treats it as an independent disease. He says that out of 180 patients in North Formosa eight had hæmoptysis or hæmorrhagic sputum. During the epidemic of 1896-'97 in North Formosa there was one case to almost every hundred patients.

Dr. Matsumoto, the Army Surgeon, became infected with pneumonic plague in 1897 whilst conducting investigations during the plague epidemic in North Formosa. He had pain in the chest, hæmorrhagic expectoration, and died. Dr. Kondo, his medical attendant, an army surgeon, also met his fatal end presenting similar symptoms to the other. It is asserted that they both presented similar symptoms, and contracted pneumonic plague. The plague bacilli were discovered in the sputum, so that the disease must have been *Primary Pneumonic Plague*. The experience of Osaka demonstrates that the medical attendants and nurses are very liable to become infected with the disease. The infection is acute and virulent.

It is evident from practical observation during the recent epidemic that infection takes place through inhaling the fine particles of expectoration during the act of coughing. The portal of infection is the mucous membrane of the respiratory system. The sputum, when examined under the microscope, contains numerous plague bacilli, which are present almost as a pure culture. But the reports of Professors Aoyama, Yamakime, Ogata, Okada, and the Bombay Plague Investigation Committee demonstrate on the contrary that infection may not take place through the medium of the floating particles of dust in the air.

The Post Mortem Appearances.

In primary pneumonic plague, an aggregation of pneumonic patches are characteristic, and bronchitis is present. One or two areas, or the whole lung may sometimes become infected. On the cut surface any of the areas affected can

always be distinguished. The secondary pneumonia of plague can be distinguished by observing that the part affected is superficial. The part of the lung that is affected with pneumonic plague is very characteristic in appearance and cannot be mistaken for ordinary pneumonia. On opening the chest, one of the conspicuous features is the peculiar colour, and the lesions are visible through the pleura. The corresponding part of the pulmonary pleura has lost its lustre slightly, and in colour is bright red. The lesion presents innumerable hæmorrhagic puncta, and is covered with a yellow layer of lymph. The lung is bright red in colour. In the demarcated areas there are fine yellow points visible. On cutting into this, and examining it under the microscope, the pulmonary alveoli are found to be dilated and engorged with large quantities of plague bacilli and blood cells. The interstitial alveolar tissue is hypertrophied and fibrinous, which can be stained well with eosin, in which there is a slight infiltrating leucocytosis, and red blood corpuscles are found. This picture is also characteristic of the disease.

In the lesion an innumerable number of plague bacilli are present, besides Fränkel's pneumo-bacillus, which is mixed with them. The lesion differs from croupous pneumonia in that there is no fibrin present in the pulmonary alveoli with the hæmorrhage nor in the secretion. This fact I have observed in a case under my notice, and found it to be correct.

Clinical History.

As regards the incubation period there has been no definite record, but during the epidemic in Osaka some definite data was obtained.

Patients.	Date of Infection.	Commencement of Disease.
Kame Yamanaka ..	6th January, 1900. Nursed husband.	9th January, 1900. Very bad rigors.
Dr. Baba	26th December, 1899. Inspected the patient (Adachi).	30th December, 1899. Rigor, feeling tired.
Mrs. Dr. Baba ..	31st December, 1899. Nursed husband.	3rd January, 1900. Rigor, fever.
Dr. Yamanaka ..	31st December, 1899. Attended on Dr. Wakabayashi.	4th January, 1900. Rigor.
Dr. Wakabayashi ..	26th December, 1899. Attended on Adachi.	30th December, 1899. Rigor.
S. Nakamura	30th December, 1899. Approached patients.	5th January, 1900. Rigor.
Mrs. Dr. Wakabayashi	30th December, 1899. Nursed husband.	3rd January, 1900. Rigor.
Mrs. Yamanaka ..	10th January, 1900. Nursed son.	10th January, 1900. Rigor.

In studying the foregoing table, the incubation period is from 4 to 6 days. In most cases it appears to be four days. It almost corresponds with the Bombay investigations.

Dr. Sticker, of Germany, in writing on the symptoms of plague describes it as follows :—

" Pneumonic Plague, having definite prodromal symtoms, presents the symptoms of catarrhal pneumonia. The patients cough sputum containing numerous plague bacilli. In most cases they die after three days through the virulence of the disease. Under certain circumstances it produces far greater prostration than in hypostatic pneumonia. In the sputum streptococci and the influenza bacillus are sometimes mixed with the plague bacillus. The plague bacillus thrives in chronic lung affections which form a nidus suitable for the development of the plague bacillus. The prognosis is very bad, and the disease is fatal."

The preliminary stage usually begins with a rigor, languid feeling, pain in the loins or hips, pains in the limbs and headache. In most instances rigors are present. Then the temperature rises and reaches 102° F.

Pains in the chest, cough, and expectoration supervene.

At the beginning the sputum is tenaceous and frothy, as in bronchitis, but it soon becomes streaked with blood. The sputum is hæmorrhagic from the beginning, presenting dark red streaks ; but very soon it becomes bright red ; and later on in most cases it is liquid, being nearly all blood. The temperature rises to 104° F., and remains at this. The respiration becomes laborious, and pains are complained of in the chest. There are very rarely any glandular swellings observable or palpable on the outside of the body in pneumonic plague. In most instances there are no cerebral symptoms till nearing death.

The physical diagnosis is usually similar to bronchitis or croupous pneumonia, presenting dull percussion areas. Bronchial rules, crepitations, and bronchial breathing are audible. At first the respiratory murmur is always harsh. The physical symptoms of the chest are trifling in comparison with the constitutional symptoms. The progress of the disease is quick and rapid.

Dr. Müller asserts that the expectoration of pneumonic plague patients does not contain fibrin. This is of diagnostic importance as compared with croupous pneumonia. So that if Weigert's method of staining the sputum is used the two diseases can be differentiated.

I have confirmed Dr. Müller's statement when examining

the sputum of two cases in Osaka. Marked prostration rapidly ensues. The face becomes emaciated, the mouth and lips cyanosed; the expectoration will become hæmorrhagic. Dyspnœa and sonorous respiration develop. Cardiac failure sets in, and the patient dies. The various stages are very rapid, from three to five days. The last stage is the worst. During the Osaka epidemic, of all the cases that contracted the disease not one recovered.

The Clinical History.

Dr. Teiichi Baba, age 28, Epidemic Inspector.

Previous History: Naturally healthy; had never been ill in bed before. Being corpulent for a few years, shortness of breath developed if he walked quickly; had been ailing for four years with chronic bronchitis; had had cough and expectoration now and then, but had never been off duty. He was a physician in the Momoyama Hospital since 1896. Total abstainer.

Infection: While discharging his duty as an epidemic inspector, he visited the pneumonic plague patients, R. Adachi and his wife, on the 26th December, 1899. During inspection he felt a particle of expectoration striking his right cheek from R. Adachi. He disinfected himself with antiseptic solution, and gargled his mouth thoroughly with a germicide. The infection appears to have taken place at this time.

The Incubation Period: He did not feel anything wrong on the 27th and 28th December. On the 29th he felt unwell. He had frequent rigors; coughing and expectoration increased. Appetite normal. Nothing unusual about his health.

The Preliminary Stage: 30th December. Tired feeling and oppression in the head; rigors frequent; no inclination to do anything; poor appetite. Being busy with the business of the epidemic, he came home late at night after attending to his duties. This night he felt very much exhausted, and did not sleep well.

On the 31st December he suffered from rigors, marked prostration, and severe headache, and pains in all his joints. Temperature 39·3° C. Pulse 120. Excitability became increased, the countenance haggard, and the conjunctival membranes hyperæmic. The surface of the body became dry and hot.

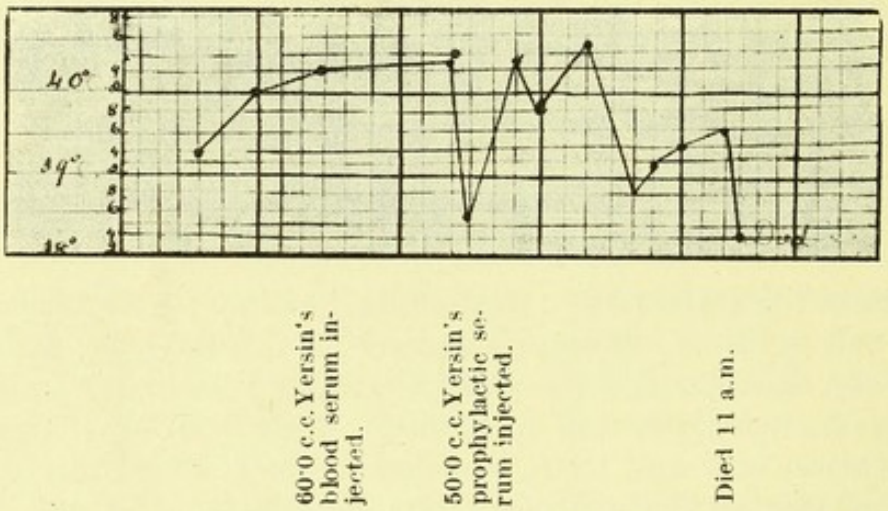
Thick moist white fur over the tongue; no appetite. Thirst and constipation present. The abdomen and spleen slightly enlarged.

PNEUMONIC PLAGUE.

Dr. Tei Ichi Baba, age 38.

31st December, 1899. 1st January, 1900. 2nd January.

Centigrade. M. E. M. E. M.



Persistent cough, acute pain in the chest. Large quantites of reddish tenacious expectoration ; sputum mixed with blood.

Physical Examination of the Chest : In addition to symptoms of bronchitis in both lungs, the upper portion of the right lung was dull on percussion, and on auscultation the respiratory sounds were here impaired. He expectorated a considerable amount of sputum. The quantity of urine was diminished. It was pale yellow in colour, slightly alkaline in reaction.

Sp. Gr. 1020. No alteration in its chemical constituents is found, and no bacilli were detected in it. In the afternoon 60 c.c of Yersin's prophylactic serum was injected. Stimulants and expectorants were administered ; and the patient was sent to the Momoyama Hospital in the evening.

1st January, 1900. The symptoms after admission were complex. Temperature 40·3° C. The face was pallid, and the expression of countenance worried. The tongue was covered with a moist white fur. There was thirst. The mental condition was clear, but not bright. Pulse increased to 130 ; cough ; hæmorrhagic mucous sputum. He did not complain much of his chest condition ; 50 c.c of the anti-plague serum was injected.

2nd January. The pulse has been very feeble since last night, and cannot be counted. The cough and expectoration are greatly increased. The sputum contains blood and pus. The respiration is hurried, and the face is becoming cyanosed. Persistent vomiting ; since yesterday diarrhœa has been pre-

sent. The mental condition is good; no muttering. He did not sleep at all last night. The appetite has entirely disappeared, and intense thirst is present. He complains of very severe pain, and cannot describe it. He tossed about in bed, and died at 11 a.m.

Clinical History.

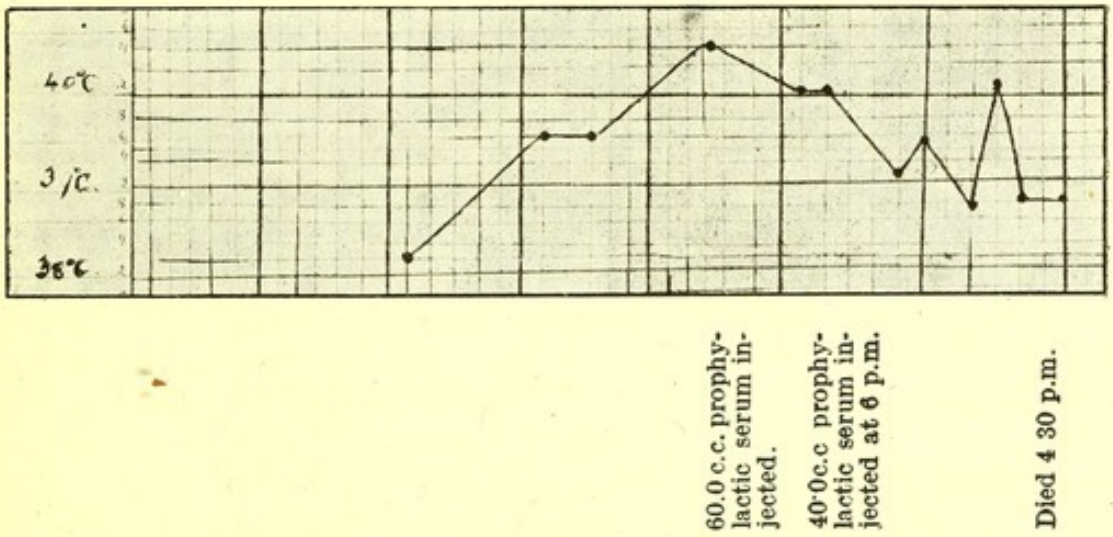
Dr. Wakabayashi, age 44.

Habitually healthy. He has never been laid up with any illness before.

On the 26th December, 1899, he examined the pneumonic plague patient, R. Adachi and his wife. He examined them again in the evening with the inspector, Dr. Baba. On the 30th December he had a rigor, and felt tired all over. He had headache and dragging pains in his limbs. Thinking that he had contracted plague, he ordered the family away, and went to bed. He could not sleep. At 2 a.m. the temperature was 38·2° C. Pulse 110. The following mixture was made up and taken in effervescence till the morning:— Tartaric acid gr. XLV., bicarbonate of soda gr. XXX.

PNEUMONIC PLAGUE.

Dr. Wakabayashi, age 44,



31st December, 1 p.m. In examining Dr. D. Hirata records: The face is pale and haggard, and there is hyperæmia of the conjunctival membranes. The surface of the body is dry, and cutis anserina is present. The tongue is covered with a white fur; very thirsty. No considerable alteration is present in the chest, excepting impaired respiratory sounds at the base of the right lung. Occasional cough and ex-

pectoration of dark purulent sputum. Temperature 39·6° C. Pulse 120. No change in the mental condition, but he is inclined to sleep. After talking he soon goes to sleep, sometimes snoring. The patient complained mostly of pains in the loins and sacrum. An injection was administered twice for the bowels, and with the desired effect.

6 p.m. Condition becoming serious ; blood is mixed with the sputum. Pulse and temperature are the same as in the morning ; nausea ; no appetite.

1st January, 7 a.m. His condition is becoming worse. The face is becoming emaciated. The mind is not clear, but he replies correctly to questions. Temperature 40·4° C., pulse 120. Camphor, red wine, stimulants, and medicine administered. Received a report from the bacteriological laboratory stating that plague bacilli had been discovered in the sputum.

Noon. Injected 60 c.c of the anti-plague serum, and sent him to the hospital.

After admission complications set in. The eyelids presented a purple colour ; the hæmorrhagic expectoration increases ; and now and again there was hæmatemesis. Respiration was hurried and sonorous. Watery catarrhal evacuations passed twice by the bowels.

6 p.m. Injected 40 c.c. of prophylactic serum.

2nd January. Emaciation has set in. The eyelids and limbs present a cyanotic appearance. Purpuric spots have appeared all over the body. He does not suffer much, and is very much inclined to sleep.

Afternoon. The sonorous respiration has increased. He died at 4.30 p.m.

This patient was aware that he was suffering from plague, and warned his family and friends of the deadly nature of the infection. After his admission into the hospital, he told Dr. Hirata that he expected the end, and did not grudge his life whenever duty called for it. He thought that his infection had taken place through inhaling the plague patient's breath ; so, whoever approaches, particularly medical men, pneumonic plague cases should cover their mouth and nostrils with some germicidal material to be inhaled from time to time as a preventive.

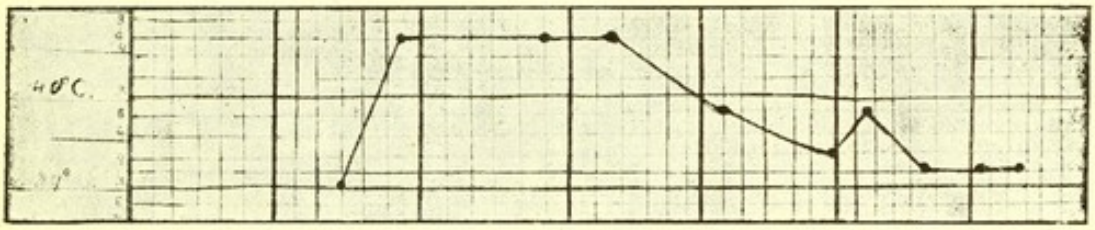
The best mode of preventing infection is to wear a thoroughly efficient respirator, and to wear a pair of glasses also. It will be well to plug the patient's nostrils and cover the mouth with "gauze" soaked with carbolic acid or perchloride solution to prevent the expectoration from spreading out.

In the event of examining a patient without a respirator, wash the face with a germicide, and thoroughly gargle the mouth and throat with it. Inhaling germicidal vapors may be of some value. In order to observe whether the inhalation of such germicidal vapors have any deleterious effect on the plague bacilli, I conducted the following experiment :—I spread the plague bacilli from Agar cultures on a piece of "gauze," and also spread them on a piece of bladder. I placed them in steam from a 5% solution of carbolic acid over the mouth of a jar, 5 to 12 inches from the mouth of the jar. After ten minutes the bacilli did not grow. A similar experiment was conducted with a 2% solution of lysol ; they perished in 15 minutes. But whether or not it has the same effect on the plague bacilli when they adhere to the mucous membranes, is a matter of uncertainty.

PNEUMONIC PLAGUE.

Dr. Kame Yamanaka, age, 47.

	4th January, 1900.		5th January.		6th January.		7th.
Centigrade.	M.	E.	M.	E.	M.	E.	M.



Temperature record of disease 6 p.m.

Died 7.20 a.m.

CHAPTER XIX.

Intestinal Plague.

It has been proved by experiments conducted by Drs. Yersin, Ogata, Okada, and others that *Intestinal plague* can be produced by food and drink containing the plague bacilli. The experiments conducted in our Institute of feeding healthy rats on the corpses of plague-infected rats proved the fact that they thus become infected with plague. But as described elsewhere, the plague bacillus is very sensitive to certain chemicals. If the stomach is healthy and its gastric juice normal, it has power to prevent the growth of the bacilli. In olden times, when it was said that infection by the stomach

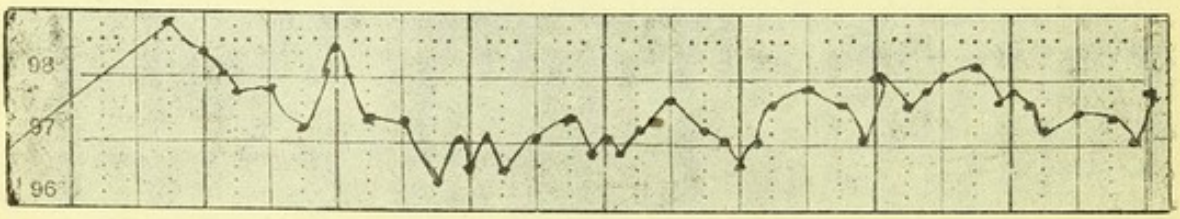
and intestines was rare this may have been the cause. It will be observed from the report of the Austrian Research Commissioners in Bombay that animals otherwise immune can be infected by introducing large quantities of the plague bacilli into the stomach through a catheter. So will it be in the human being : the person can be infected if a large quantity of the bacilli are introduced into the stomach ; but it is a positive fact that a very small quantity of the bacilli in the healthy stomach will not infect. The acid of gastric juice will not permit the growth of bacilli. It may be that the infection takes place either when a large quantity of the bacilli are introduced, or when the gastric juice in the stomach is diminished in quality or quantity through some cause, or when the gastric juice is greatly reduced and altered through indiscretion in diet.

The Post Mortem Appearances.

The mucous membranes present characteristic features. Numerous hæmorrhages are clustered together, varying in size from a hemp seed to a little smaller. The hæmorrhage is limited to the mucous membrane, and does not penetrate the submucosa. Moreover, there are ulcers in the mucous membrane in various parts. The floor of the ulcers is necrosed and disorganised. The margin and surrounding area is catarrhal. In the small intestine Peyer's patches are greatly enlarged, presenting a peculiar yellow colour, having in various parts red macular hæmorrhages. Large quantities of the bacilli are found in the glandular texture, and in the mucous membranes. The solitary glands are also swollen, being the size of a pea or large bean. They are soft, and have hæmorrhages in various parts.

KENNEDYTOWN HOSPITAL.

Fig. 43 (i.)—K.C.U., age 61, male, opium smoker. Intestinal Plague. Chinese. 15th April, 1896. Plague bacilli were found in the fæces on 23rd April.



Plague bacilli were found in the lungs, liver, spleen, and glands.

FIG. 43 (ii.).—K.C.U. (continued).

23rd April, 1896.

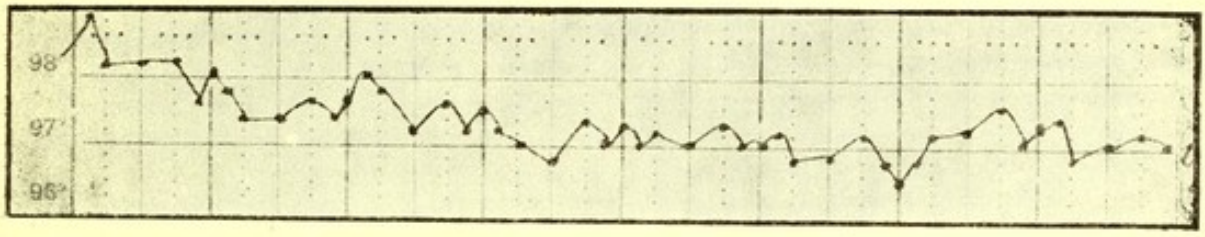
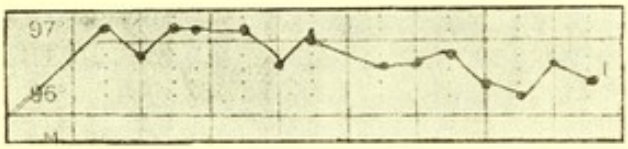


FIG. 43 (iii.).—K.C.U. (continued).

1st May, 1896.



Died on 7th May, 1896, at 3.30 p.m.

THE CLINICAL SYMPTOMS.

The clinical symptoms are somewhat similar to typhoid fever or malignant typhus. The patient feels unwell with muscular pains and pains in the joints. Such will be the prodromal symptoms. Then rigors, febrile temperature, abdominal pains, and liquid mucous evacuations set in. Loss of appetite, purging, gastric, and intestinal flatus are present. Blood becomes mixed with the evacuations. The spleen rapidly enlarges, which is painful on pressure. It is very difficult to diagnose, as there are no superficial glandular enlargements. Septicæmia sets in after three or four days from the onset of the illness. A bacteriological examination of the evacuations with the microscope will not assist in the diagnosis, as large quantities of bacilli morphologically identical are present. According to the report of the Bombay Commissioners, the best *modus operandi* is to rub the shaven portion of an animal's body with the plague bacilli when the plague bacilli will be absorbed into the system, and the bacillus *coli communi* will multiply locally, and not invade. I regret that I could not observe any actual symptoms of intestinal plague during the epidemic, as no cases came under my observation. However, the following is a case which came under my notice, and was regarded as intestinal plague :—

THE CASE OF MASTER HIDEYANE, AGE 2.

This is the child that the nurse girl, Kiwa Yamasaki, nursed in Osaka. He was healthy and sound, and had never been ill before this.

On the 3rd December, 1899, he was restless about 2 a.m.; and a sudden rise of temperature (38·7° C.) had set in. He became convulsive, the whole body being rigid. The teeth were set. In glancing at the symptoms they appeared to be those of meningitis, but no purging or diarrhœa was present. By this time the house and family were isolated and under the inspector's surveillance.

On the morning of the 4th, when the inspector arrived, the convulsive seizure had disappeared. The temperature was 39·8° C. There was hyperæmia of the conjunctival membranes, but the child was still sucking the breast as usual. No glandular enlargement was present, excepting a bean-sized swelling in the right cervical region. This was pierced, and its contents were examined; but no plague bacilli were found. Next day, the 5th, there were several convulsions; but on the 6th the symptoms were relaxing. The temperature was down to 38° C. It became an unmistakeable case of plague; death occurred suddenly on the 8th December at 4 a.m.

According to the inspector's report who presented it that morning, the body is stated to be well nourished; vis mortua considerable. Only a few signs of death were present on the back. In piercing the heart and spleen a large number of plague bacilli were unexpectedly discovered in the liquid contents. Only two or three plague bacilli were discovered in the excised gland in the neck when examined.

As the diagnosis was difficult, we applied for permission to hold a post mortem examination. The principal features are as follows:—

Stomach.—The mucous membrane all over was hypertrophied, and of an ash-white colour, with several small hæmorrhagic areas.

At the pyloric end there was a diffuse hæmorrhage which presented a dark purple colour.

Intestines.—The small as well as the large intestines were considerably hypertrophied, and of an ash-white color. Peyer's patches were swollen, and several small hæmorrhages were present in them. They contained a yellow ash-white liquid. This liquid contained numerous plague bacilli.

The Mesentery and Mesenteric Glands.

The large and small omentum was ash-white in colour and very fatty. The mesenteric glands were enlarged to the size of grapes. Plague bacilli were present in the glands.

Spleen.—The spleen was three times the normal size.

The splenic tissue was friable. A large number of plague bacilli were detected.

LIVER.—The liver was enlarged, congested, and hæmorrhages were present under the capsule.

HEART.—The heart was dilated, especially the right ventricle. The right ventricle was filled with a large blood clot. The left ventricle was empty. The pericardium and pericardial fluid were normal. A large number of bacilli were detected in the blood of the heart.

LUNGS.—Both lungs were congested. There was a small quantity of fluid in the pleural cavity.

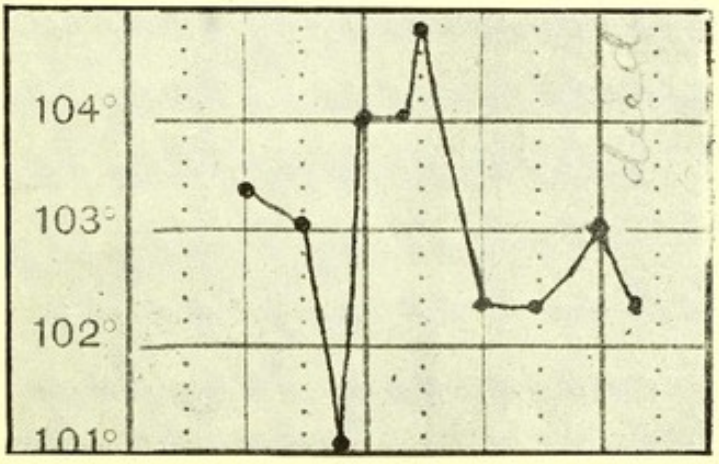
BRAIN MEMBRANES.—The dura mater is thick with fat at the falx cerebri. It could not be easily detached from the bone.

The pia mater was œdematous. There was a large quantity of hæmorrhagic cerebro-spinal fluid. Large numbers of plague bacilli were found in the cerebro-spinal fluid. If the cerebro-spinal fluid had been obtained by the lumbar puncture in this case and examined during life. the plague bacilli could thus have been detected. The Kitasato-Yersin bacilli were discovered in the organs when examined by cultivation. The spleen and blood of the heart contained equally the Kitasato-Yersin bacilli. In the cerebro-spinal fluid there were more of the Kitasato bacilli present than the other pleomorphic variety.

KENNEDYTOWN HOSPITAL.

FIG. 44.—L.F., age 28 ; male. Bubonic. Chinese.

2nd May, 1896.

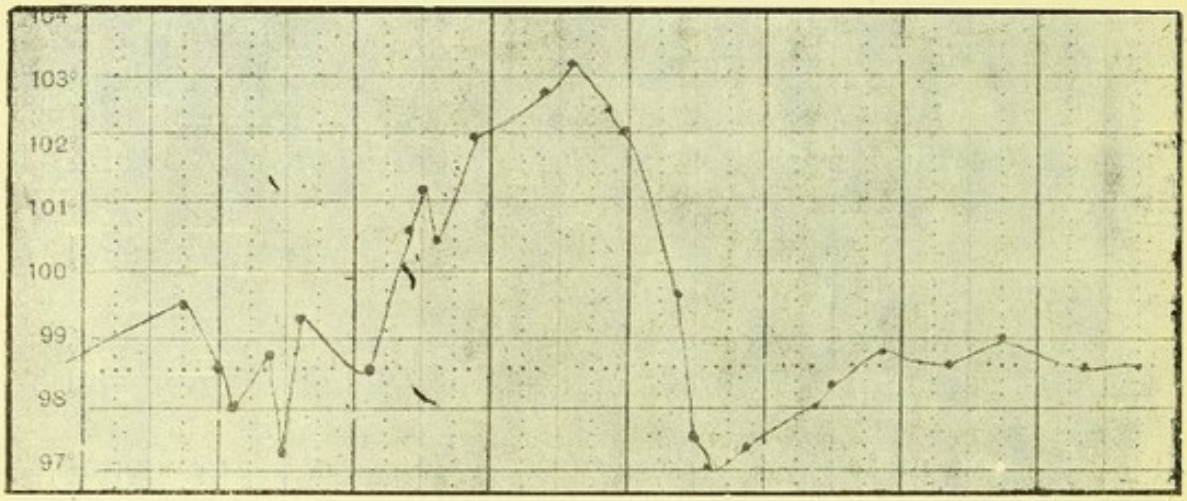


Calomel.
Gr. V.

Died 4th May, 1896.

KENNEDYTOWN HOSPITAL.

FIG. 45.—K.K., age 3 ; male. Bubonic. Chinese.
30th June, 1896.



Discharged 18th July, 1896.

CHAPTER XX.

CLINICAL MEDICINE.

When I was in Hong Kong a few years ago with Profs. Kitasato and Aoyama all the patients whom we examined were bubonic plague cases who had already been removed to the hospital. Most of them were in the final stage of the disease, and we observed none of those which presented difficulty in diagnosis during the first stage. However, during the epidemic in Kobe and Osaka I had a good opportunity of personally attending some of the doubtful cases, and of conducting the bacteriological analysis. I herewith note down some of these observations. The diagnosis of infectious disease whether the case be mild or malignant has an important bearing on the remedial and preventive measures. The disease must be diagnosed in its first stage. Especially with acute and malignant cases, a correct diagnosis is of the utmost importance with reference to the prevention of the disease. A correct diagnosis cannot be made as described until the pathognomonic bacilli are discovered.

Bubonic plague can be easily diagnosed in the early stage by puncturing the gland or excising a piece of the glandular tissue. But the diagnosis of pneumonic. intestinal, and-cerebral plague is very difficult, since the superficial lymphatic glands are not enlarged.

No. 1. Bubonic Plague.

The infection through the lower limbs attacks the internal femoral glands, and the inguinal glands. When infection is through the upper limb the axillary glands are attacked. But the portal through which infection has occurred cannot, except in a few rare cases, be proved.

Bubonic plague is to be distinguished from the following :—

1. Venereal Disease.—Pain, fever, and no trace in the sexual organs of gonorrhœa or venereal sores will suffice.

2. Simple Wounds and Ulcers.—Lymphangeitis and enlarged glands are caused through wounds or sores together with a febrile condition.

3. The Cervical and Submaxillary Glandular Enlargements.—Any inflammatory condition of the throat or gums may be accompanied with rigors, fever, and enlargement of the cervical and submaxillary glands.

4. Lymphadenoma, or Chronic Syphilitic Glandular Swellings.—Without any apparent cause, the temperature may suddenly rise in these diseases.

In such cases if the enlarged glands are sensitive and painful to pressure, if the temperature rises suddenly with a rapid pulse, if the conjunctival membranes are hyperæmic, and if a tired and languid feeling is present, they are doubtful and must be subjected to a systematic examination.

There is no necessity to puncture the glands if the glandular swelling and febrile condition can be attributed to some known cause. But if there is comparatively little proof explaining the causation, and the hyperæmia of the conjunctival membranes, together with languor is present, then the gland requires puncturing. Attention is to be directed to the fact that sometimes in bubonic plague, although the bubo is very painful the rise in temperature may not be considerable, also to the fact that in the early stages the bacilli are found in small quantities in the affected gland.

No. 2. Pneumonic Plague.

Secondary pneumonia occurring during the course of bubonic plague is not difficult to distinguish, as it is a simple pulmonary affection. It is difficult to diagnose primary pneumonic plague in its early stage. However, in most cases there is some history of infection. If an incubation period exists at all, it will consist of a languid feeling of the body. a fulness of the head, and rigors. But in most cases it begins with sudden rigors, headache, restlessness at night, insomnia,

pains in the limbs and loins. The temperature ranges between 102° F. to 104° F., with drowsiness and coughing. The pulse is over 100. No alteration in the percussion note is perceptible at this stage. Still in several areas, or in one lobe impairment of the respiratory murmur will be detected. However, at this stage of the disease it cannot be distinguished from ordinary pneumonia, influenza, or any other acute pulmonary affection. When coughing and expectoration commence then the bacilli can be detected in the sputum. But the sputum usually contains other bacilli resembling the plague bacilli, and which are comparatively smaller in quantity. The microscope alone cannot decide, and culture and animal experiments are necessary. Pneumonic plague is so acute and rapid in its progress that we generally observe blood in the expectoration of the patient before we can obtain results of the cultivations and animal experiments. Scarcely any other bacilli will be found in the hæmorrhagic expectoration at this stage, but almost a pure culture of the plague bacilli will be obtained from the sputum. In most instances Fränkel's pneumo-bacillus will be found. It is characteristic of the sputum that it contains no fibrin as described elsewhere.

Pneumonic plague cannot be diagnosed by the symptoms and microscopic examination in its early stage. Therefore, during the epidemic when some patient, who has been exposed to infection, should present such symptoms in the respiratory system, it is better to treat the case as one of plague, and wait until the expectoration begins.

The infected patient generally expectorates within 24 hours after the rigor. When once one has examined a case of the disease a more or less accurate opinion can be formed regarding others from the prostrate expression of the countenance, the hyperæmia of the conjunctival membrane, the respiration, and the agonising pain to which the patient is subjected.

No. 3. Intestinal Plague.

It begins, as described, first with a rigor, fever, the symptoms of gastric and intestinal catarrh, diarrhœa, pain in the abdomen, mucous and bloody evacuations and urine. The patient succumbs and dies of septicæmia within three or four days. A characteristic of this disease is the sudden enlargement of the spleen, which is tender on pressure. So when a patient complains of being feverish, of diarrhœa during the epidemic, examine the evacuations at once, the

examination of which, as described elsewhere, is very difficult. The case can only be determined by cultivation and inoculation experiments on animals. (Vide Fig. 46.)

Examine the fluid contents obtained by puncturing the spleen. When fever sets in, the case can generally be diagnosed, for the spleen contains the bacilli. Puncturing the spleen must be conducted with very great caution under antiseptic precautions, as regards the position of the organ. It has often been observed that the intestines and other organs have been punctured. Conducted antiseptically, puncturing the spleen is not a dangerous procedure. It is better to obtain permission from the patient, or from his relatives before conducting such an operation.

THE PRINCIPAL MICROBES.

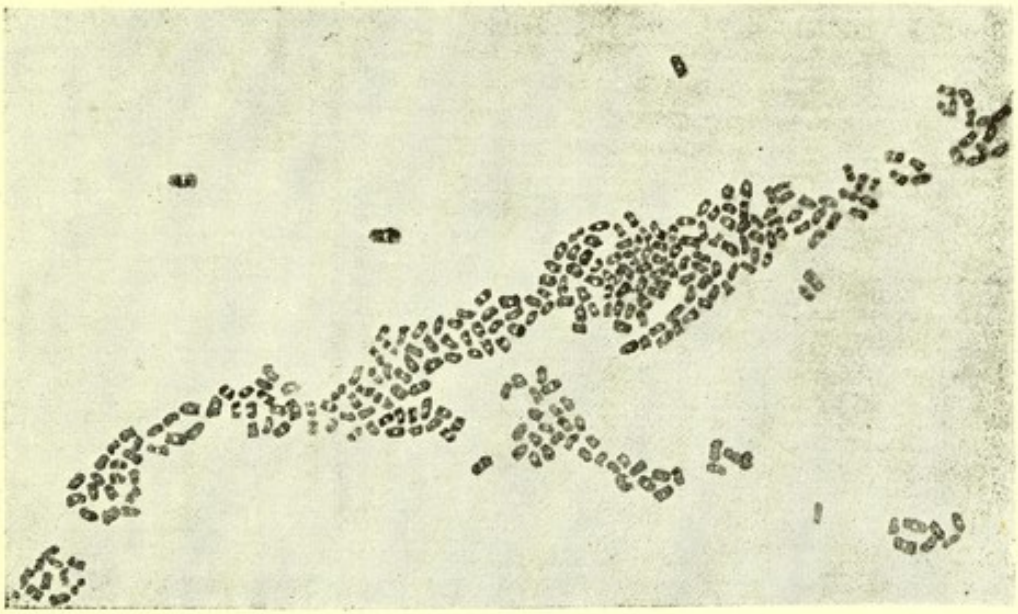


FIG. 46.—B. typhi abdominalis. Agar culture. Fuchsin. X 1000.

No. 4. THE INVESTIGATION OF THE BACILLUS.

The epidemic in Kobe and Osaka has subsided; still in a country like Japan where extensive traffic with India, China, and our new colony, Formosa, where plague prevails almost endemically, is kept up, we must be prepared for future invasions or an epidemic that may be introduced through infected rats. The bacteriological investigation is of the utmost importance in this disease.

1. The Bacteriological Laboratory.

The following ought to be observed :—

1. No admittance.
2. Convenience in fumigation and disinfection.
3. Light and air must be efficient.
4. The ingress of rats and cats must be obstructed.

The laboratory is divided into *the Rat Dissection Room* and the *Examination Room.*

The Rat Dissection Room to consist of the following :—

One table. A dissecting board (about 4 or 5 squares).
The requisite dissecting knives and scalpels.
Soda disinfectant apparatus.
Large quantities of germicidal solutions.
A few sterilised petri plates.

When a rat is sent in, immerse it in a 5% solution of carbolic acid to destroy the vermin. Place it on the board ; wipe the abdomen ; and take care that the germicidal solution does not penetrate the incision. Use a pair of scissors and a knife that has been immersed in the soda disinfectant bath.

When dissecting the following is to be observed :—

1. Is the subcutaneous tissue tumid ?

2. If the femoral and axillary glands are enlarged. The glands generally adhere to the subcutaneous tissue and skin when the skin is pulled off.

3. Observe if there is any fluid in the abdominal or pleural cavities.

4. Observe if the spleen and liver are enlarged, and greatly changed in colour.

If a glandular swelling is present, excise it, taking care not to injure it. Excise the spleen, heart, and liver, and deposit them in petri plates, covering them over at once. Fumigate all the instruments and the dissecting board. Afterwards burn the rat's corpse, preserving it all the time in the germicidal solution. After having done these things, wrap the petri plates containing the organs tightly with lint that has been dipped in 5% solution of carbolic acid, and send it round to the Examination Room.

The Examination Room to consist of the following :—

1. The microscope.
2. Stand for staining re-agents.
3. Test tube stands for the stock culture media.
4. Cages for animal experiments.
5. Inoculation stand.

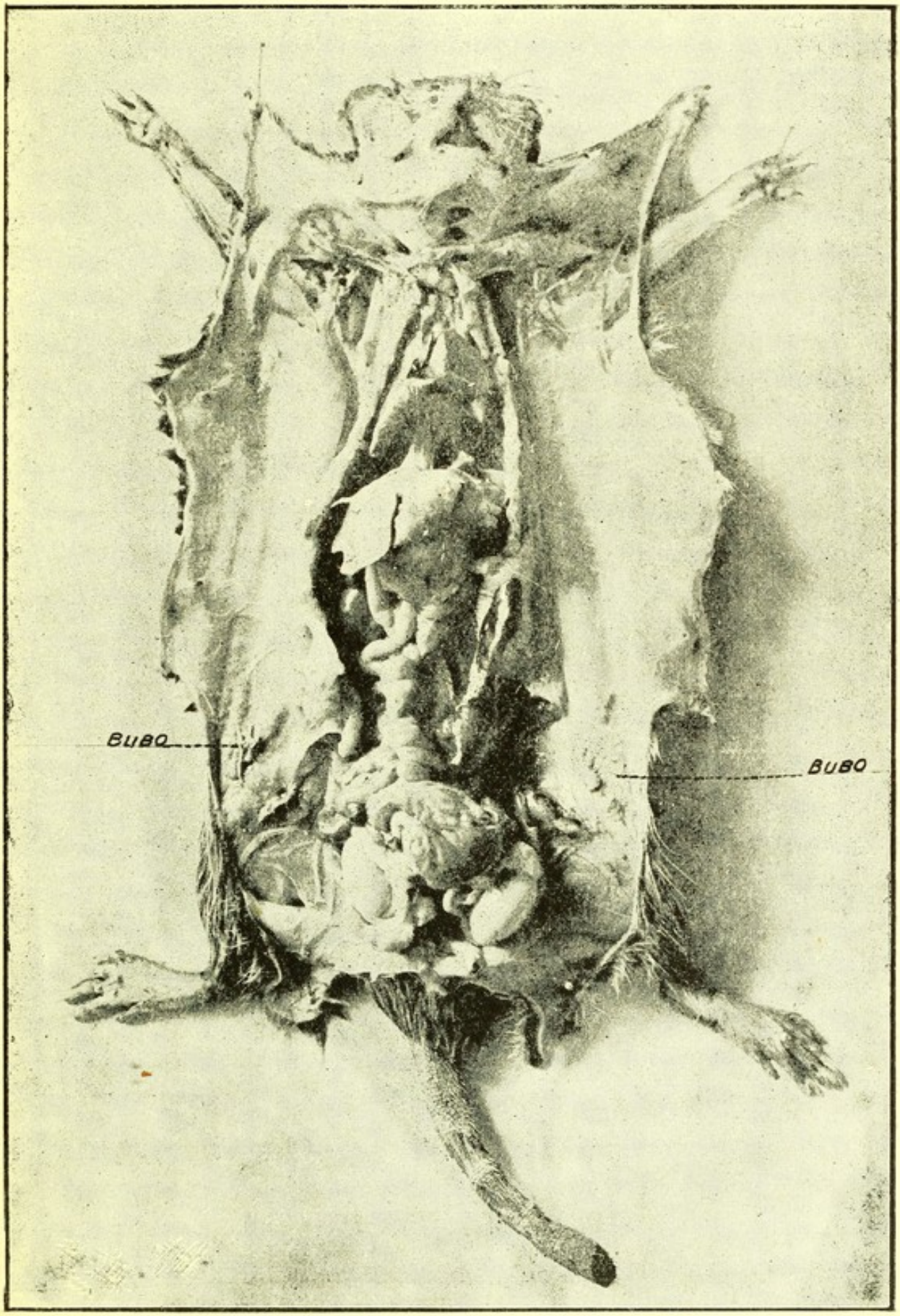


FIG. 47.—Bī she **beng**, plague rat showing buboes. Rat dissection showing enlargement of liver and spleen. Plague inoculated specimen two days old.
Gall.

The various tables on which these things are placed must be arranged so as to avoid confusion. Several other instruments, apparatus, and appliances necessary for the investigatoin must be provided.

2. The Methods of Collecting Materials.

It is best for the medical man to collect the materials for examination himself. If the collection of materials be entrusted to one who possesses no bacteriological knowledge, he is apt to spread infection ; and it may also result in securing incomplete material.

As described in Chapter XIII. on diagnosis, the materials are as follows :—

For bubonic plague :
The liquid contained in the swollen glands.

For pneumonic plague :
The sputum or expectoration.

For intestinal plague :
The evacuations.

For cutaneous plague :
The purulent matter and secretion from the infected part.

For the examination of the corpse :
The contents of the spleen, heart, or the venous blood, and cerebro-spinal fluid.

For collecting these materials, a Pravaz's injection syringe is the instrument used. The following are the requisites for excising a gland :—A knife, a pair of scissors, a needle and thread, collodion, germicidal solution, absolute alcohol, sterilised petri plates, and bandages. All the necessary instruments had better be kept in a portable metal box, the whole of which must be sterilised. If absolute alcohol or boiling soda solution be used as antiseptics, they are apt to injure the injection syringe. It is better to wash the syringe with a 5% solution of carbolic acid, and then afterwards with sterilised distilled water. In the event of conducting cultivations and animal experiments, it is essential to get rid of all the carbolic acid that may remain in the instrument. The smallest trace of it must be avoided. Great care must be exercised when washing with the germicide. Rinse it at least ten times.

Sterilization of the part is to be strictly adhered to when puncturing the gland.

The process as already described in another chapter is to

be adopted. Directly after the syringeful has been obtained, pour out the contents into a sterilised petri plate.

It is not advisable to take the contents home in the instrument, as it may often be found coagulated and diminished in quantity when so carried.

After the operation, collodion must be applied to the puncture mark left by the injection of the instrument.

All the blood that may exude during the operation of excision of the gland must be carefully wiped away with lint that has been saturated with some germicidal fluid, as it contains the infection. The wound must be stitched, collodion applied, and it must be bandaged.

Sputum, fæces and pus had better be collected in petri plates. Wrap up all the articles used in thick cloth which has been saturated with the 5% solution of carbolic acid, and place them in the metallic box.

The petri plates containing the materials are to be wrapped up in oil paper, and again in lint which has been saturated with 5 % solution of carbolic acid, which the medical man must take with him. Under no circumstanese whatever must the parcel be entrusted to any one else when it is taken away.

3. The Mode of Examination.

The examination of these materials is to be conducted with the utmost care and discretion. Never allow anything to happen through carelessness. A pair of Cornet's forceps must be used when handling the cover glass on which the material is spread. Such a common mode as removing the cover glass with the fingers must be avoided, Use a platinum wire when spreading the material on the cover glass. Dry the material spread on the cover glass in the air, and to harden it, pass it three or four times through the flame. Examine it with the microscope, and the bacilli, as in the illustration of the frontispiece, will be detected.

Be vigilant as regards the flies in the room. Always cover the materials.

4. The Diagnosis of Analagous Bacilli.

The detection of the pyogenic bacilli in bubonic plague and the pneumo-bacilli in pneumonic plague have been described in their respective chapters.

As regards recognising the typhoid bacillus and the bacillus coli communis by their staining properties and their morphology, it is not only accompanied with difficulty in

diagnosis, but owing to the peculiar nature of some of these large colon bacilli having no inclination for motility, an attempt at diagnosis with the microscope will be futile. Therefore, an examination of the evacuations must be conducted by cultivation and animal experiments.

The organic materials of dead rats required for bacteriological examination are the swollen glands, spleen, heart, and blood.

Several similar bacilli are found in these which often lead to erroneous results, especially is it the case in rats which have been dead for some time. The bacillus coli communis is often discovered in the organs of dead rats. Although the bacillus (bacillus coli communis), that has invaded the blood of the heart and spleen in most instances is a pure culture, and could be distinguished by observing its movements, yet its detection will become very difficult when a large quantity of putrefactive bacilli are present in a dead rat that has already undergone considerable putrefaction.

Though there are circumstances in which, as Professor Okada has remarked in reference to this matter, the putrefactive bacilli possessing similar features could be distinguished by Gram's method, still cultivations and experiments on animals are essential in most instances.

The bacillus typhi murium which exists in dead rats that have died from feeding on them cannot be distinguished sometimes by staining ; still the bacillus typhi murium, being strongly motile, makes their detection easy.

5. The Trial of Cultures.

The nature of the sputum, fæces, and the mucous exudation on the tonsils is to be determined as soon as possible. To facilitate this the bacteriological process requires that it be placed on an inclined or flat surface of Agar-Agar and placed in the incubator at 37° C. The colonies will develop within twenty-four hours. Make a formal examination of the colonies under the microscope.

As the plague bacilli have the power of growing slowly at a low temperature, a little over or below 10° C., the organs of very decomposed dead rats, containing very large quantities of other bacilli (if there is no urgency), can furnish culture material for the refrigerator. Gelatine stab cultures or plate cultures may be made. At this low cultivation temperature the plague bacilli alone will grow, so it is convenient for diagnosis. This feature is not observed in other bacilli. Also the plague bacillus can be distinguished from others by

its characteristic pleomorphism. In culture media to which 3% of NaCl is added, the bacilli present rapid pleomorphic metamorphosis. In this media the bacilli soon present the features of large yeast cells.

6. Animal Experiments.

In animal experiments house rats are most preferable, but for fear of them escaping, the Nankin rats (tame rats) are preferred, except in special cases when house rats are required.

The rat is to be placed in an ordinary round glass jar. Cotton and bran are to be put in the jar. The food must contain moisture. When covering the jar place layers of cloth that have been dipped in a 5% solution of carbolic acid or in 1 : 100 mercurial solution, over which a wire net is to be placed. Keep the jar away from the attack of rats or cats. After the experiments burn the cotton and the dead rat at once. The glass jar and wire net are to be kept for six hours in the 5% solution of carbolic acid.

The length of time that Nankin rats take to die varies according to the quantity and virulence of the plague bacilli, as well as to the nature of the rats. For purposes of diagnosis an injection into the abdomen is preferable, as rats die in a much shorter time by this method than by hypodermic inoculation. The minimum dose to be injected must be as viscid as possible. Use Bouillon or sterilised distilled water to dissolve the dose.

The dose to be injected should be 0·3 cubic centimetres. Rats sometimes die from shock when large quantities are injected.

During inoculation fasten the rat to Kitasato's apparatus holder. Pince up the skin of the abdomen with a pair of forceps. Cautiously inject into the abdominal wall, hypodermically directing the instrument diagonal to the body. Care must be exercised because, if any of the organs are injured, the rat will die. In drawing out the needle the liquid is apt to come out of the body, so when the injection is completed unfasten the tail to some extent. Remember to wipe the part carefully with lint dipped in a 5% solution of carbolic acid in order to destroy any infection that may happen to remain behind on the fur.

In conducting experiments on animals more than two are to be used for a given dose, varying the dose thus : rat I=0·1 c.c. ; rat II=0·2 c.c. ; rat III=0·3 c.c., when three rats are used.

If plague bacilli are present in the juice of enlarged glands, sputum, and evacuations that have been injected into the abdomen of rats, they generally die within 24 hours.

In examining these rats there is fluid found in the abdominal cavity. This exudation contains the plague bacilli which have also invaded the spleen and the blood of the heart. In most instances the glands are found not enlarged in such rats as they die so soon. Though they usually die within twenty-four hours, some survive for three or four days ; hence a rash conclusion must not be formed.

Attention is to be drawn to wearing garments specially made for covering one during the process of collecting the materials, the dissection of rats, the cultivations, and the experiments. Exercise also proper discretion in the selection of an attendant whose services may be required in connection with the work of examination.

KENNEDYTOWN HOSPITAL.

Fig. 48 (i.).—C.M., age 26 ; male. Bubonic. Chinese.

Admitted at 8.15 p.m.

25th March, 1896.

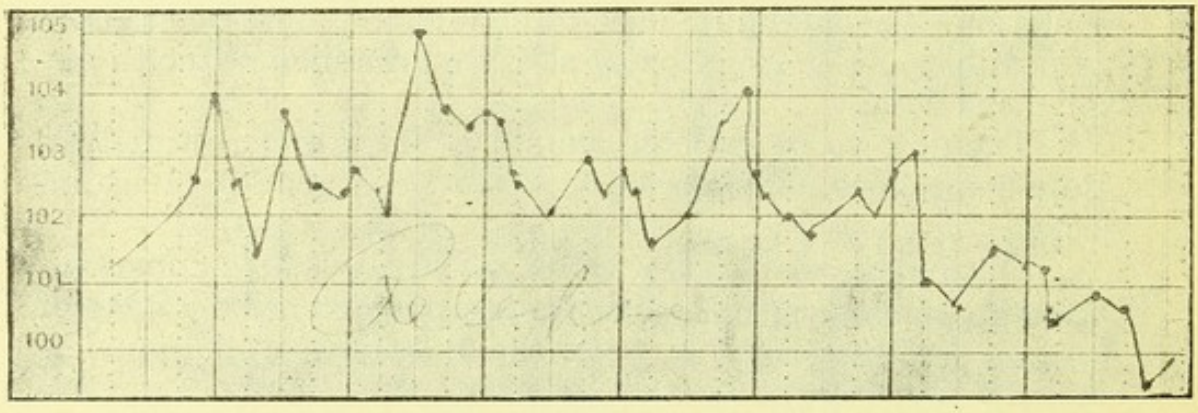
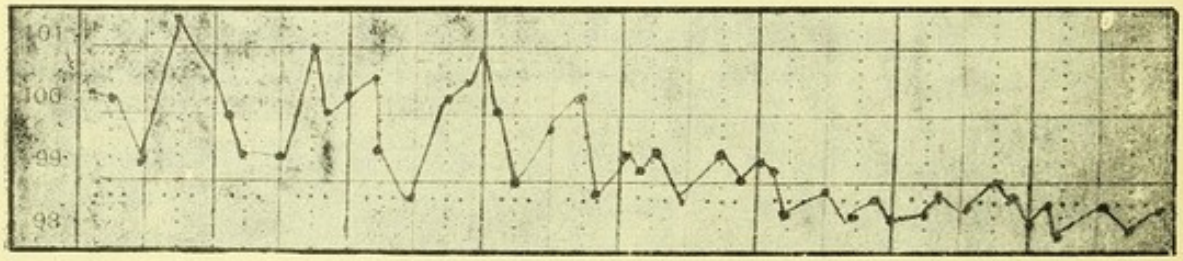


Fig. 48 (ii.).—C.M. (continued).

2nd April.



Discharged 24th June, 1896.

CHAPTER XXI.

Plague Prophylaxis.

A. Preventive Measures.

Judging from traces of plague infection which invaded Kobe and Osaka, it is evident that infection was imported inland by the medium of vessels from India, China, and Formosa.

In order to prevent its introduction, the regulations of epidemic inspection in ports and harbours where such vessels enter must be strictly carried out.

Complete quarantine stations must also be established in all ports where the fumigation of passengers and cargo is to be conducted. But, since such strict regulations might retard commerce, some medical men should be appointed who have a thorough knowledge of the infectious diseases at the various ports ; relinquish the management of the ports to their discretion regarding health matters.

My opinion as regards plague prophylaxis is to observe the following measures :—

1. Epidemic inspectors are to be established in ports.
2. Offices for the examination of infectious disease are to be attached, the supervision of which is to be conducted by medical men specially trained for the post.
3. Let all vessels which trade between Japan and the infected ports carry a medical man who is to keep surveillance over passengers, and be responsible for any doubtful cases, also for dead rats.
4. Concerning the vessels which do not carry or object to carry a responsible medical man on board, they must be subjected to a thorough inspection of the entire vessel.
5. All goods imported are to be fumigated on landing.
6. Fumigation stations are to be established in all ports for the above purpose.
7. As cotton is very dangerous, encourage the manufacturers and others interested to establish co-operative fumigation stations.
8. All so-called " ship's refuse," valuable or otherwise, must be burnt. Mr. Hawaka, engineer to Hiogoken, has designed a fumigator and destructor to be used for such " refuse " collected by the company in Kobe from vessels. At this moment they are under probation.
9. Strict regulations regarding the health matters of all ports are to be framed.
10. Conduct rat destruction from time to time.

1. Train Inspection.

There is not much necessity for imposing rigid measures regarding train traffic, if only the vigorous inspection of vessels is enforced. But as described in a former chapter, some measures must be adopted in this direction. Rat extermination in every station is essential.

2. Isolation.

As described elsewhere, it is best to limit the area of such isolation to a small space, and adopt strict surveillance, as some are apt to shield patients from publicity.

Search for rats in the street where a patient has been discovered. Institute daily visits of inspection from house to house.

Display a notice of some sort in the front of an infected house.

The infected houses are to be fumigated.

The steps of such fumigation, as already described, are to be carried out under the supervision of a medical man.

Spray carbolic acid on the roof and walls of the house. Sprinkle chloride of lime under the floor and in the garden.

3. The Conveyance of Patients.

The jolting of the body is injurious in this disease, so it is better not to convey plague patients too far from the infected house.

So it is not desirable to erect hospitals too far out of town. Establish a hospital if possible in the corner of a town. If the town is large and has a large population, erect several of them in various parts of the town.

B. Concerning the Resistance of the Plague Bacillus Against Chemicals.

(1) Mr. N. Takami, of our Institute, conducted a few experiments. The results are as follows:—

Mercurial solution	1 :	10,000	killed in over	30	minutes.
Do.	1 :	5,000	killed in over	10	minutes.
Do.	1 :	1,000	killed in over	2	minutes.
Carbolic acid,	1 :	100	killed in over	5	minutes.
Do.	1 :	50	killed in over	1	minute.
Do.	1 :	20	killed instantly.		
Lysol,	1 :	100	killed in over	2	minutes
Do.	1 :	50	killed in over	1	minute.
Do.	1 :	25	killed instantly.		

FIG. 49.

$N\beta$ [with olive oil, filamentous plague culture, Horticultural temperature. [*Gall.*

(2) Its vitality on some edibles was experimented on by the same member. He spread a few drops of Bouillon culture on such daily food stuffs as cakes, buns, bread, dried fruits, jellies, confections, biscuits, edible seaweed, and so forth. He kept them in petri plates, excluding the sunlight, and could not detect any live bacilli after the tenth day. The same results were obtained when sputum from a pneumonic plague patient was applied to the same articles of diet.

The experiments by Dr. Glardin on brown bread almost corresponds to these experiments (1 to 3 weeks)

FIG. 50.

$N\beta$ with cocoa butter and olive oil, filamentous plague culture. H.T. [*Gall.*

If the bacilli adheres to such articles, there is danger of infection during ten days.

*C. Concerning Rat Hunting.**

FIG. 51.

$N\beta$ with layer of cocoanut oil; Stalactites four days old. R.T

This problem exasperates the authorities of almost every country.

The Board of Health for Osaka advertised a pecuniary reward for the discovery of a good method of rat catching; but could not obtain any competent suggestion. The Sanitary Society (a private institution) in Tokyo is offering a substantial reward at this moment for a perfect method.

There is no hope of exterminating the rodent tribe; so as regards this matter, it will be our only policy to diminish their number.

It is an important matter to educate the public, and direct their attention to it. Under the discretion of the police authorities or the Chairman of the Board of Health, sanction the use of poison for

* Various investigators have now found different species of microbes effectual, being fatal to rats, but harmless to man (D. McD.)

this purpose. The use of bacillus typhi murium is recommended. Experiments with this bacillus merit a trial. The investigations with this bacillus have been conducted by Mr. Matsuda, an assistant of our Institute, and the results are as follows :—

1. Bacillus Typhi Murium.

This bacillus was discovered by Dr. Loffler in 1890. It is harmless to human beings, but poisonous to rats. It has been stated that the bacillus possesses good merits for field mice, as experimented with in Thessaly. The results of our investigations are as folows :—

In features these bacilli resemble the bacillus coli communis in every respect in size, length, and motility, and are rod-shape bacilli. They have flagellæ and particularly energetic movements. They stain with several of the aniline dyes.

1. It presents the agglutinative reaction with the blood of Nankin rats (tame rats) into which a small quantity of this bacillus was injected and kept for a while.

2. It presents the same reaction as above with the blood of Nankin and house rats that have died through an injection of this bacillus.

3. It does not present the agglutinations with the blood of healthy Nankin or house rats.

4. It does not present the agglutination reaction with
   (*a*) The blood of human beings which have immunity to typhoid fever (intestinal typhoid).
   (*b*) The serum that has been immunised against dysenteric bacilli by bacteriologist Shiga's process.

5. There is no reaction with the blood of marmots into which large quantities of the bacillus coli communis were injected several times.

The Biological Characters.

1. Gelatine Plate Culture.—There was no growth after four days when cultivated at 14° C., and below this. It was then changed to the outside top of the incubator, and after four more days small round white macroscopic colonies appeared. Under the microscope it was of a yellowish colour with a round spot in its centre, and the entire surface was granular in appearance. The margin was irregular. It is pale yellow towards the margin, and gradually fades in colour from the centre to the margin.

2. Gelatine Stab Culture.—There was a growth along the punctural track at room temperature after a week. It grows better on the upper than the lower track. It does not liquefy Gelatine

3. Agar-Agar.—When cultivated on the inclined surface Agar they are similar almost to typhoid bacilli colonies.

4. Bouillon Culture.—After 24 hours in the incubator the growth is luxuriant. The Bouillon becomes cloudy, and, if kept at 12° C. or 13° C., it gradually clears up again at the top from the third day. After a week the upper half became entirely clear with a sediment at the bottom, and a film on the top. It does not produce Indol.

5. Glucose Agar.—After 24 hours in the incubator a luxuriant growth along the stab occurs with gas production, and no smell.

6. Potato.—It is affirmed by one authority that it stains the surrounding media ; but I did not observe this.

After 24 hours in the incubator moist yellowish white colonies were observed.

7. Milk.—It grows well, but produces no coagulation.

Animal Experimentation.

Bouillon cultures were used as they are convenient for feeding purposes. Firstly, a 24-hours' old Bouillon culture from the incubator was injected into the abdomen of Nankin rats in the following proportions.

Rat I=0·5 c.c.
Rat II=0·4 c.c.
Rat III=0·3 c.c.

The weight of the rats were about 10 grammes.

Rat I=0·5 c.c. was still after six hours, and died after 26 hours.

Rat II=0·4 c.c. became still after seven hours, but it did not die. It recovered.

Rat III=0·3 c.c. only presented at the time symptoms of prostration.

Necropsy of the Dead Rat.—There was a small amount of fluid in the abdominal cavity. The spleen was enlarged, and the liver was of a dark red colour. Nothing abnormal was found in the chest. There were large quantities of the bacilli in the liver and spleen ; but none were present in the blood of the heart.

The liver that contained the bacilli was macerated in Bouillon, and this was injected in the abdomen of Nankin rats. This process was repeated several times, and it was found that 0·1 c.c of a Bouillon culture of this bacillus so obtained was

Fig. 52.

$N\beta$ with layer of cocoanut oil. Stalactites two weeks old. R.T. [*Gall.*

highly virulent, destroying a 10-gramme rat in 24 hours. On the other hand, dead rats were given to house rats to feed upon. They were afterwards fed upon vegetables and grain. Their movements became lethargic after seven days, and they died on the 10th day. Feeding other rats with the last dead ones from one to the other up to thirteen, the virulence became intensified each time. A Bouillon culture of this bacillus that had acquired such virulence, was poured on bread, and administered to house rats. They died in 10 days.* This bacillus was found in the rats again.

D. Plague Prophylactic Inoculation and Serumtherapy.

Prophylactic inoculation was first attempted in India by Dr. Haffkine. Though no definite opinion as to its merits can be formed yet, still as affirmed by several European authorities, there is no danger in its application. Experiments with this inoculation in our Institute proved the fact that it is not dangerous

Fig. 53.

$N\beta$ with layer of cocoanut oil; Plague vaccine (Haffkine) Stalactites a month old. R.T. [*Gall.*

(1) Dr. Haffkine's Process: A Bouillon culture of the plague bacillus is kept in the incubator and cultivated for one month. It is then heated for an hour at 70° C. to kill the bacilli. A 0·5% solution of carbolic acid is added to the Bouillon culture. The dose for use is 5·0 cubic centimetres, and over this for the second.

(2) The German Commissioners utilised solid cultures. Inclined surface Agar culures were cultivated for two days in the incubator, dissolved, and mixed with physiological salt. The

Fig. 54.

$N\beta$ with cocoa butter, filamentous plague culture. H.T.

* Dr. Danysz, Paris, has had effectual results with his species of rat-killing microbes (D. McD.)

germs were then killed by heating for one hour at 70° C.; and 0·5% carbolic acid solution is added. The dose for an individual is from ½ to 1 surface.

It is affirmed that solid cultures when used produce less reaction than Bouillon cultures.

(3) Bacteriologist Shiga's process is to triturate the solid culture of plague bacilli and immunised plague Blood Serum together.

An inclined surface Agar culture is cultivated for two days and two nights in the incubator. It is scraped off with the platinum loop, triturated until the bodies of the bacilli are pulverised so fine that when stained they cannot be traced or recognised under the microscope.

One cubic centimetre of physiological salt solution is added to each platinum loopful of the above heated to 60° C. for 30 minutes; and carbolic acid is added in the proportion of 0·5%. This forms the original formula of the liquid.

The first inoculation consists of the original formula to which an equal part of immunised plague serum is added. The dose is from 0·4 c.c. to 0·6 c.c. The second consists of the original formula of the liquid, and is used alone. The dose is 0·6 c.c. to 0·8 c.c.

Therefore the solution injected in these two inoculations will contain one platinum loopful of the saline culture of the plague bacillus.

The triturated one is more readily absorbed, and with less local reaction. When the immunised serum is mixed there is less local and constitutional reaction than without the serum.

(4) The process adopted by bacteriologist Yokote is his own invention. The Bouillon culture of the plague bacillus is filtered, and the filtrate is used.

A Bouillon culture is cultivated for several days, and heated at 60° C. for thirty minutes.

It is then filtered through a chamberlain filter. To the filtrate add carbolic acid in the proportion of 0·5%.

Begin with a 0·5 c.c. dose, and gradually increase it up to 3·0 c.c.

The local and constitutional reaction is less if the filtrate is adopted than if the bodies of the bacilli are used.

Several opinions differ as to the merits of serumtherapy, and it requires future investigation.

French and Russian investigators appear to highly recommend serumtherapy. The experiments conducted in the Monoyama Hospital in Osaka did not furnish sufficient

evidence to form any opinion on account of the number of tests having been too small.

THE OPORTO EXPERIMENTS.

(Vide Bibliography, Part II.)

The resolutions of the meeting held in Berlin in October, 1899, with reference to the plague (vide Bibliography, Part II.).

FINIS (ISHIGAMI).

SPECIAL BACTERIOLOGICAL REPORT.

Case C.

CULTURES (Negative evidence).—Results :

$C\beta$ (1) Phenol Bouillon (4 c.c.–100 c.c.) : No cultivation. After 10 days medium still transparent.

$M\beta$ (2) Milk : No coagulation after 10 days.

Ps (3) Potato : No visible cultivation until after about a week.

$N\gamma$ (4) Gelatine Media (22° C.) : No liquefaction after ten days.

Bs (5) Blood Serum (37° C.) : No liquefaction after ten days.

POSITIVE RESULTS :

$S\beta s$ (1) Blood Serum (37° C.), Löffler : Faint yellowish growth

Ga (2) Glycerine Agar (37° C.) : Moist greyish film.

$N\gamma$ (3) Liquid Gelatine (37° C.) : Culture on surface only.

$N^l\gamma$ (4) Litmus Gelatine (37° C.) : Neutral.

$S\gamma$ (5) Glucose Gelatine (22° C.) : Culture on surface ; no gas production.

$N\gamma$ (6) Nutrient Gelatine (22° C.) : Beautiful discrete iridescent colonies varying in size from a pin point to 1·5 mm. with little increase in size after 10 days.

$N\beta$ (7) Nutrient Bouillon (37° C.) : Presented a dusty floculent growth on the sides of the vessel which afterwards fell down to the bottom in a few days as a sediment, the medium remaining transparent throughout. The bacilli were found in thread and clumps. The organism was a cocco-bacillus non-motile, and decolourised by Gram's stain.

SERODIAGNOSIS.—During the 5th week of the disease agglutination was obtained (1 in 20), with the bacillus cultivated and blood serum ; 48 hours culture.

INOCULATION.—I inoculated a guinea pig in the abdomen with a pure subculture in Glycerine Agar of the bacillus

which exhibited the above morphological, cultural, and staining characters. The following day the animal looked sick with staring hair. It died in about four days with twitchings. Autopsy was held three hours after it died. In the interval between it was moistened with chloroform.

AUTOPSY.—The following appearances were observed. Fluid was found in the peritoneal cavity. Spleen enlarged. The intestines were congested. The right and left femoral and axillary glands were enlarged to the size of split peas. The kidneys were considerably enlarged. Miliary tubercles the size of pin heads were found on the upper and under surface of the liver. There was marked leucocytosis present, Lymphangeitis, extending from the point of inoculation, was evident. Other glands were enlarged, but to a less extent. Bacilli were found in the blood of the heart, the left lung, the spleen, liver, and kidneys in smear preparations. In the femoral glands they were short thick rods with clear centrum and rounded ends with bipolar staining. Occurring also in threads, their breadth in the left femoral glands were ·8μ and length 1·3μ. The kidneys revealed phagocytic casts engorged with bacilli. The tubercles in the liver consisted of phagocytes *en masse* choked with bacilli, and also of disintegrating leucocytes. The polynuclear variety of leucocytes contained no bacilli. The femoral glands, spleen, and left lung gave positive results by cultivation.

Nβ A. NUTRIENT BOUILLON inoculated from the left femoral gland gave a dusty floculent growth on the sides of the flask.

Gα B. GLYCERINE AGAR, after 24 hours' inoculation from the femoral glands and spleen, presented the following.

COLONIES.—The colonies were discrete pin point and pin head in size. By translucent light they had a bluish tinge, granular appearance resembling pulverised glass. They were moist greyish in appearance. In contour they were circular and irregularly quadrilateral. Under magnification they were granular with irregular margins.

Gα C. Glycerine Agar, pure culture, same as B, exhibited bacilli decolourised by Gram's method, with occasional nobs, but no tendency to long threads. Non-motile bacilli with Brownian movement.

Oβ D. Stalactite growths in Bouillon (300 c.c.) with thick layer of milk fat and cocoanut oil; stalactites $\frac{1}{4}$ in. in diameter, $\frac{3}{4}$ in. in length, with streamers on the side of flask.

3%NaCl a E. Hankin's Salt Agar 3%. Pear, spindle-shaped bodies in 24 hours (chloretic bacilli).

VACCINATION.—Inoculated white mouse with sterilised Agar culture (3 loopfuls); became sick and remained so for three days. A fortnight later I inoculated the white mouse again. I used on this second occasion a pure Glycerine Agar culture which had killed by inoculation the aforesaid guinea pig described. With this virulent culture the animal soon became very ill with lethargy and hurried respiration. About 24 hours afterwards it was lying on its side with hurried respiration and staring hair. The head and limbs appeared quite bluish as if it were about to die. However, it picked up in about 12 hours, and became again lively.

VITALITY.—I subjected cultures of the pest bacillus to a temperature of 55° C. for 15 minutes. Cultures of the typhoid bacillus were subjected to the same as controls. The pest bacillus was killed in 15 minutes, as evinced by their failure to grow again when reinoculated on culture media. The typhoid bacillary cultures survived the 15 minutes at 55° C., as demonstrated by further cultivation (D. McD.).

KENNEDYTOWN HOSPITAL.

FIG. 55 (i.).—L., age 40; male. Bubonic. Chinese.

18th June, 1896.

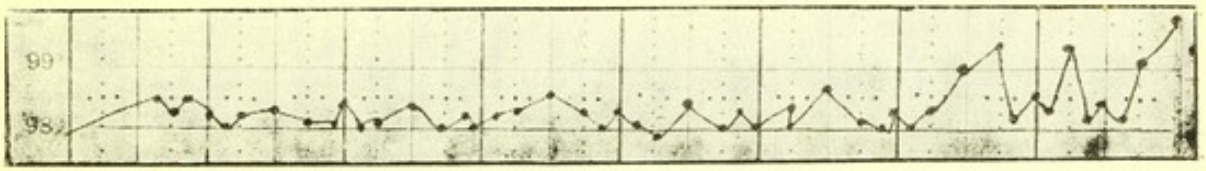
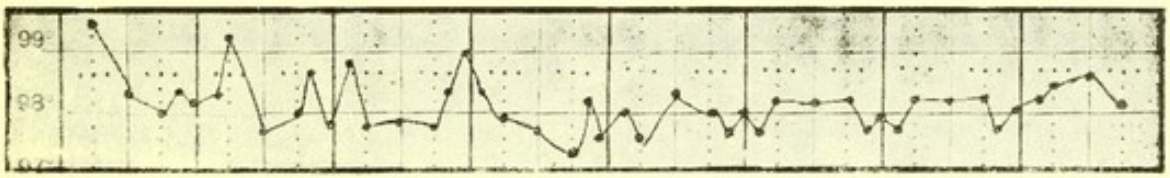


FIG. 55 (ii.).—L. (continued).

26th June. 1st July.

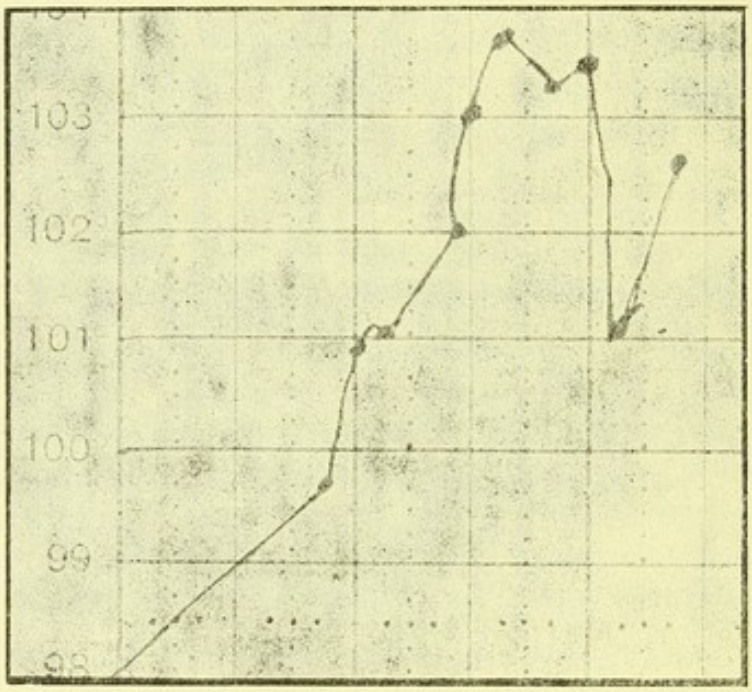


Discharged 23rd July, 1896.

KENNEDYTOWN HOSPITAL.

FIG. 56.—C.S.H., age 34 ; female. Bubonic. Chinese.

9th May, 1896.



Calomel.

Died on 11th May, 1896.

Text of Plate II.

"Here are sweet peas, on tip toe for a flight,
With wings of gentle flush o'er delicate white,
And taper fingers catching at all things
To bind them all about with fairy rings."

—*Keats.*

"The garden's gem,
Heart ease, like a gallant bold,
In a coat of purple and gold."

—*Leigh Hunt.*

"Here is pansies; that's for thoughts."

—*Shakspeare.*

Original oil painting from a specimen of chromogenic self fertilisation M. P. aureus as violaceus (1898).

Bacteriaceæ,
Chromaceæ,
Exanthemata,
Violaceæ.

Formulary XXV.

$$BGY^{v}_{s}\beta NaCO^{3}.$$

100 c.c.

B = Blood serum 50 c.c.
G = Gentian.
Y = Yelk 50 c.c.
$NaCO_3$, 10% m.x.
v = G. violet 1% m.x.
s = Serum.
β = Bouillon.

Mix, filter, sterilise.

Text of Plate II.

" Here are sweet peas, on tip toe for a flight,
With wings of gentle flush o'er delicate white,
And taper fingers catching at all things
To bind them all about with fairy rings."

—*Keats*

" The garden's gem,
Heart ease, like a gallant bold,
In a coat of purple and gold."

—*Leigh Hunt.*

" Here is pansies ; that's for thoughts."

—*Shakspeare.*

Original oil painting from a specimen of chromogenic self fertilisation. M. P. aureus as violaceus (1898).

Bacteriaceæ,
Chromaceæ,
Exanthemata,
Violaceæ.

Kaleidoscopic Chromogenesis.

Bacterioscopy.

Biological Reagent G_s^V

Papilionaceous Leguminosæ.

Dermo-phanerophyte.

MICROBE PLANT.

Vardon & Pritch
Lithographers,
Graeham Street, Ad

PLAGUE

PATHOGENIC HORTICULTURE.

PART II.

PROLEGOMENA.

"At this low temperature (8° C.) the Plague Bacillus will grow alone."—ISHIGAMI.

"Plague Bacilli acquire the appearance of a yeast, or algæ."—HAFFKINE.

"They are often confounded with very fine yeast cells."—ISHIGAMI.

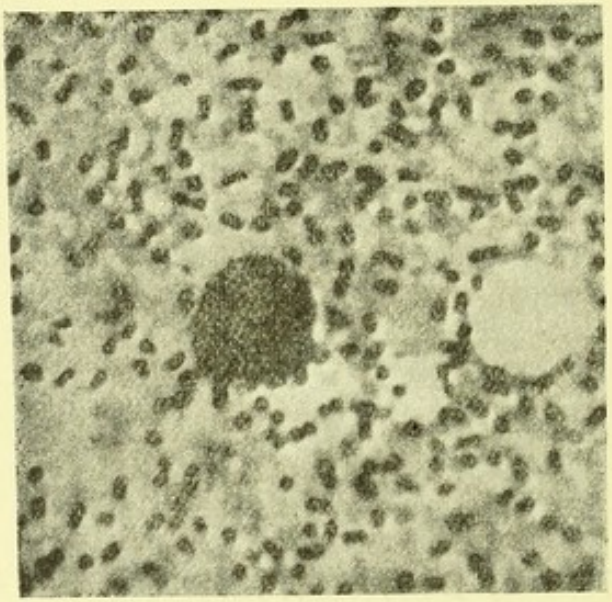


FIG. 57.—Bacillus Pestis Bubonicæ from the pus of a femoral bubo in a Chinese case, Hong Kong. Cover glass preparation. Fuchsin. X 1000.
[*Nelly.*

PART II.

TEXT COMPLEMENT.

CHAPTER I.

Japanese Literature.

Plague Synonyms.

1. Pesuto (Japanese).
2. The Pest (French).
3. La Peste (French).
4. Pestis inguinalia (classical).
5. Pestis glandularia (classical).
6. Bubonen Pest (German).
7. Pestilenz (Italian).
8. Bubonic Pest.
9. Pneumonic Pest.
10. Intestinal Pest.
11. Cutaneous Plague.
12. Black Death (Historical).
13. Bubonic Typhus (Historical).
14. Oriental Pest.
15. Himalaya Pest (endemic).
16. Pali Pest (Indian).
17. Mahamara (Indian).
18. Wăn yik (Cantonese), classical.
19. Wan yih (Chinese).
20. Bew shair jēn (Hong Kong), a long thin snake native to water holes.
21. Bī she bēng, Taiwan, Formosa, rat plague.
    (A similar term is also used in Foochow, the great tea centre of China).
22. Ong ek, Foochow, China.

PLAGUE, WITH BACTERIOLOGICAL RESEARCH, AT THE K.T. INFECTIOUS HOSPITAL, HONGKONG, 1896.

General Summary.

The revival of a disease which had been reckoned almost extinct for two centuries again excites universal attention. The Plague, febris bubonica, occupies some of the darkest pages of human history, and its destructive incursions into the nations of the world at different periods compare more with that of modern warfare than anything else. The "Black Death" of mediæval ages and the Great Plague of London are facts well known to almost any school boy. It has long been prophesied that plague will again revisit Europe in its usual virulence, and the present Oriental epidemic is prevalent on the lines of the ancient plague epidemics. Beginning in Egypt, its home for thousands of years, being nurtured by the variations of the Nile in its production of drought and famines, it disseminated itself by similar methods periodically and geographically, like a despotic invader interfering with human and animal life. During past centuries the causation of this disease was attributed to the religious negligence of the people, to astronomical phenomena, to demonology, or in some cases to religious sages, who were supposed capable of anathematizing a district with the disease. The same casual agencies occupy a portion of the Oriental mind, but now the disease is based on the principles of sound common sense. Certain microbes are known to have retained their vitality for decades, such as anthrax, and it is more than probable that the plague bacillus was originally imported intoEurope fromEgypt in mediæval ages in preserved mummies, which were then utilised for medicinal purposes, and sold very largely. However, its fertilization is fostered by a breach of nature—filth, and filth and the plague bacillus are concomitants. This germ has been unobserved by medical sages for ages in the lower animals, which carry the disease rapidly from one place to another. and thus infect the human race. Thus, domestic animals and mummies, which were held sacred by the Egyptians, cannot now be so in relation to this disease. The lower

animals are very susceptible to plague, especially domestic animals, and thus by divers methods spread the disease to human beings. Thus the health of the lower animals must always be consistent with the health of the human being as

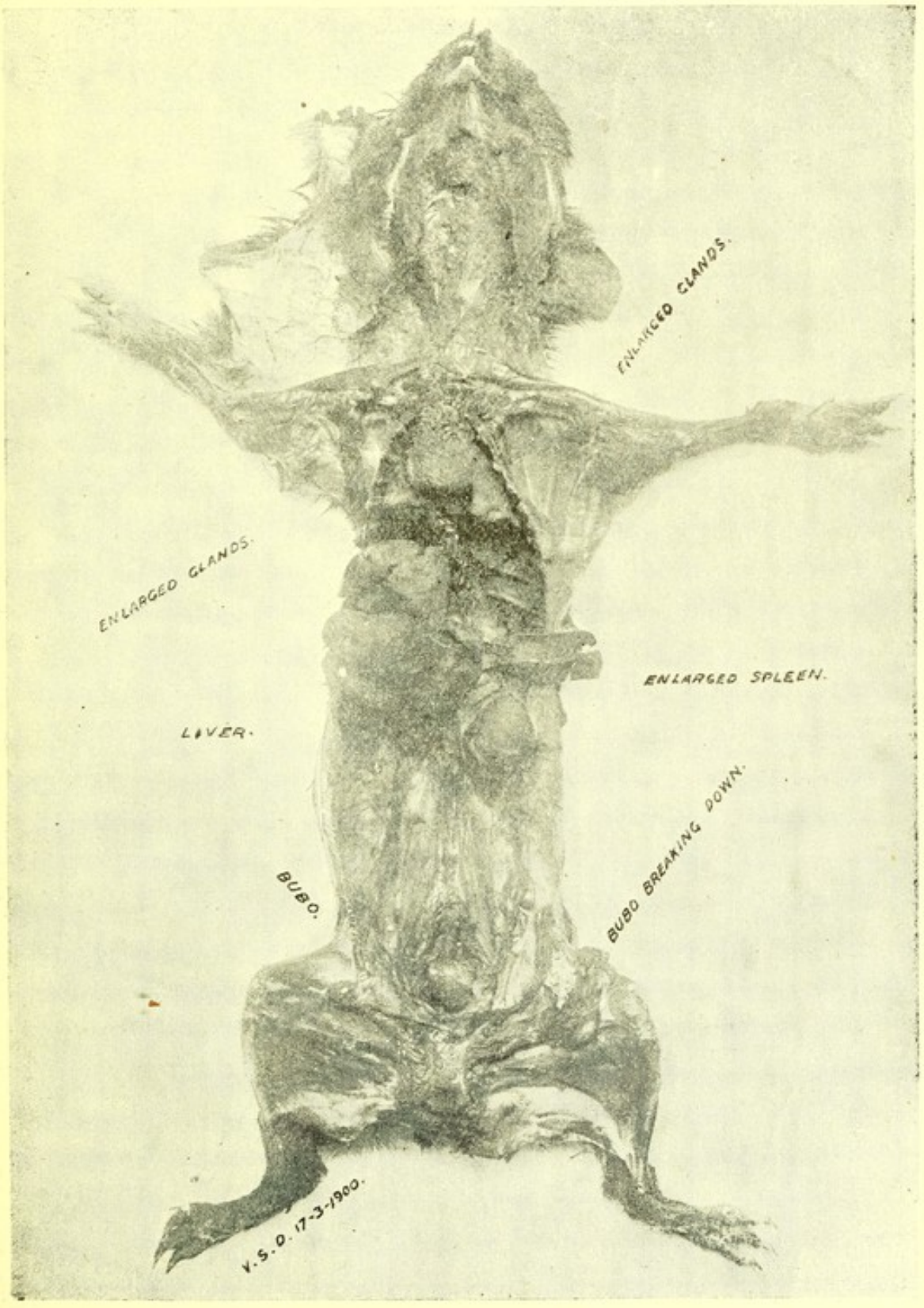


FIG. 58.—Guinea pig inoculated from mesenteric glands of plague case, boy. [*Desmond.*

far as this disease is concerned. I had, directly and indirectly, the opportunity of inoculating forty-six animals with plague, and of making over three hundred post-mortem examinations. Amongst the animals inoculated were Chinese pigs, guinea pigs, rabbits, rats, Japanese mice (white), monkeys, and fowls. The results accruing from these methods of investigation are of notorious value. It is now conclusive that a definite bacillus is responsible for this disease, and whatever contributes to the elucidation of this disease will be generally welcomed. Its apprehension will, moreover, in due time be accompanied with successful preventive measures. The bacillus is found in almost all the fluids of the body, and inoculated animals generally succumb from a few hours to one or two weeks. Experimentation has not yet proved that this disease is contagious in the proper sense of the term. Cultivations from the skin and perspiration gave negative results. One of the most likely impressive features of the bacillus is the fact that it is capable of attacking the ordinary diet of man and animals. Small animals fed by the ingestion of plague-inoculated milk, rice, pork, and chicken, common Oriental articles of diet, died in from six to forty-eight hours. This illustrates probable ancient methods of disseminating plague by the ingestion of mummies for medicinal purposes.

Pathology of Plague.

In this disease, from what I observed, the lymphatic system is seriously involved. The bacillus appears to have a natural selection for the lymphatic structures. Inoculation of animals demonstrates, from the site of infection, lymphangeitis extending in all directions to the nearest lymphatic glands. This is now historical from the fact of post-mortem infection in medical men. In a case that had received severe injuries to the hand, there was manifestly a cordlike swelling in the arm, which was excised post-mortem, and demonstrated the bacillus. Another case with a large ulcer on the left foot had corresponding buboes on the same side, with confirmatory evidence in the ulcer. The inoculation of animals subcutaneously predicates, at least, the possibility of infection by the skin. Infection by the mouth is indicated by cervical and submaxillary buboes. Sixteen acute cases had cervical glandular swellings, three of which were unilateral. Parotid buboes were observed in the convalescent stage. These facts indicate a close relation to the manner of infection like cervical tuberculosis. In fifteen cases the autopsy revealed enlarged bronchial and mediastinal glands, together with œdema and

hypostatic congestion of the lungs. Seven cases had bloody expectoration, in which the bacillus was detected. A simple catarrh, or already existing bronchitis, appeared to lay the foundation for this pneumonic form of plague, a case of

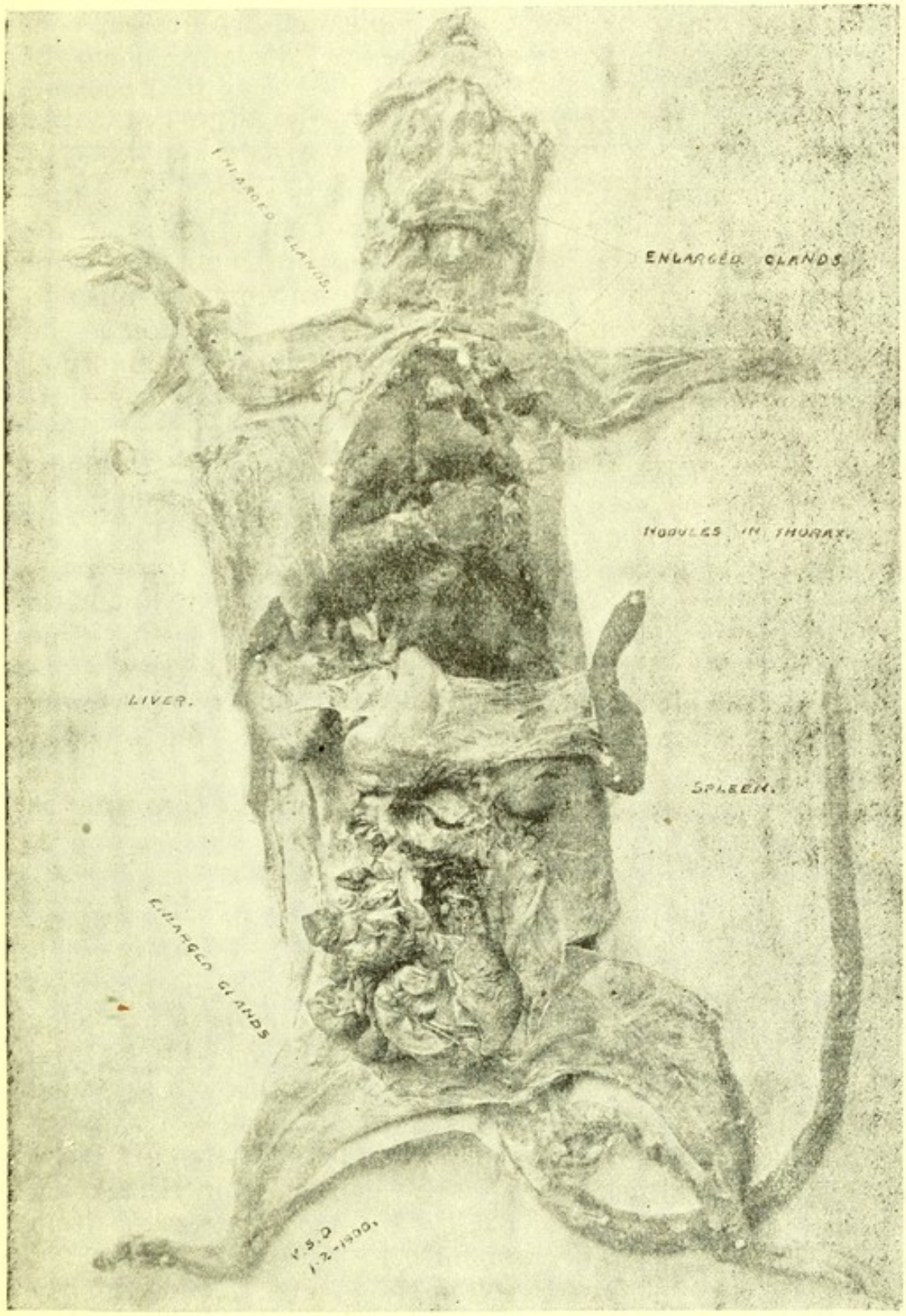


FIG 59.—Guinea pig inoculated with plague bacilli with pathological appearances. [*Desmond.*

phthisis pulmonalis having occurred concomitantly. The retroperitoneal glands were markedly involved in a case which was confined ten days previously, indicating a uterine infection. The closed follicles of the intestines, together with Pevers patches, were usually implicated. The mesenteric glands appeared to be profoundly implicated in all cases, even when no palpable buboes were present. This enteric type of the disease was an evident exaggeration of the initial stage of the general infection, and does not appear to merit the appellation bubonic fever. Twenty per cent. of the cases observed were of this intestinal type. These types were most prevalent at the acme of the epidemic. Febris bubonica intestinalis appears to be a natural division in the nomenclature of the disease, with a separate set of symptoms peculiar to itself. Its severity, its short duration, its localization to the intestinal tract, together with the diarrhœa and subnormal temperature are a train of symptoms reminding one intimately of cholera. It is undoubtedly a very virulent form of the disease, proving fatal in a few hours by its acute toxæmia; the injection of the intestine together with petechiaæ in the stomach, and the congestion and occasional ulceration of Peyers patches were exponents of the Septicæmic type of the disease. The mesenteric glands, like others, were found to be hæmorrhagic, whilst in one case perforation of the intestine had occurred. From a study of thirty-three cases, convalescence was of short duration. The bacillus was not found in the blood after twenty-one days in those examined, whilst its presence was demonstrated in other forms of the disease after three months. Streptococci were fairly constant as concomitants in pus from cervical buboes.

Clinical History.

The clinical histories vary in a large number of cases. Some are conspicuous by their poverty of symptoms, particularly the enteric form. Such patients reveal nothing further than being ill. No buboes or febrile temperature being present. The average incubation period I learned to be from seven to ten days, although in one case it must have extended to fifteen days. The disease is again conspicuous by its absence of prodromal symptoms. The fact that sporadic cases have occurred in Singapore a week's journey from Hongkong, roughly indicates its incubation. A single case came from Macao, where considerable immunity is enjoyed, and contracted the disease when he was a week in Hongkong. This is a fairly accurate instance of its incubation period. The following is a fair history of the incubation period of plague in a family.

The cook contracted the disease and went to Canton ; three days after the cook left, the amma went to the Hospital ; twelve days after the latter the mother became ill. Four days after this the eldest daughter, and in five days the youngest, took ill. The apathetic physiognomy, the characteristic furred and dark-brown tongue, together with the staggering gait, are prominent symptoms of the disease, which sanitary officials soon become acquainted with. The sunken eyes, together with the injection, and in some cases the purulent discharge of the conjunctiva become well marked. Sordes collect on the lips and teeth, and the tongue becomes coated with a purple or chocolate scarf, and exhibits fissures resembling a third week typhoid patient. Occasionally there is a lichenous eruption on the limbs and body, apt to be mistaken for mosquito bites. There is no characteristic eruption, and it is usually absent, as may happen in enteric fever. Dyspnœa, bronchitis, and pulmonary hypostasis develop, and in pulmonic cases prune juice sputum rich in plague bacilli, together with streptococci occurs. Then symptoms of cardiac failure develop. The urine is high coloured, and the presence of albumen is detected. The skin is dry and hot ; in some cases cold perspiration occurs antecedent to death. The perspiration was found to be acid. No plague bacilli were detected in the cold sweats. The acidity would most naturally prove unsuitable for their development. Herpes occurred in a few cases. Vomiting is quite common, together with diarrhœa, and vesical and rectal tenesmus.

Buboes.

The classical bubo varies from a small lentil to a large swelling, which had suppurated before the patient's admission to the hospital. Twenty-two per cent. of the cases had not a proper bubo. Extensive periadenitis occurs. This is one of the most prominent features of the disease. The buboes may be either single or multiple, localised or universal, symmetrical or asymmetrical. The plague gland in itself is indistinguishable from glandular swellings of other diseases. The "pot belly" was exhibited in badly nourished children, and blood and mucous evacuations were observed."

Convalescence.

In reviewing convalescents, I observed that they largely consisted of patients who had accompanying diseases. The simultaneous effect of other microbic disease had a beneficent and curable effect. The following were some of the contem-

porary diseases of convalescents :—Malaria, pyæmia, beri beri, syphilis, eczema, and ichthyosis. Convalescence was prolonged, and in some cases was followed by suppurative fever, which carried some off later on. Parotitis and œdema of the lower limbs, together with boils, were visible remnants of the disease. The prognosis of the cervical variety of bubo appeared to be not so grave as the axillary and femoral. The bacillus was detected very late in the convalescent stage, so that a convalescent must be regarded with suspicion for some time. Bed sores occurred in five cases. The bacillus was detected in the convalescent as late as the third month of the disease. It may be fairly well inferred that one attack confers immunity against a second, as no case in my experience appeared to have had a former attack.

Nervous System.

As many of the nervous symptoms appeared to be conveyed into the convalescent stage, I undertook a systematic clinical examination of the nervous system of a number of cases. The results may not all be confirmed, but in the main they indicate how profoundly the nervous system may be affected. By the alternation of salt and sugar to the tongue of eleven cases, the sense of taste was found to be unimpaired. Similarly, the sense of temperature of the body was found to be intact in all but two cases which were complicated with beri beri, by the alternate application of hot and cold. One of them exhibited hyperæsthesia, whilst the other evinced anæsthesia below the patellæ. Of twelve acute cases, 91 per cent. passed urine and fæces involuntarily. Inco-ordination of the lower limbs, ataxic gait was present in 63 per cent. of thirty cases, and 40 per cent. of them were convalescents. Vertigo was present in 56 per cent. of thirty cases, and 33 per cent. were convalescents. The patellar reflex was absent in 60 per cent. of forty-one cases, and 37 per cent. of them were convalescents. The gluteal reflex was absent in 63 per cent., the plantar in 60 per cent., the abdominal in 30 per cent., the distal in 33 per cent. The cremasteric and scapular reflexes were present in all thirty cases examined. It is thus observed that the co-ordinating reflexes are involved somewhat, and this is sometimes observed in inoculated animals. The toxæmia appears thus to affect the central, and not the peripheral nervous system, and this is evident in the convalescent stage.

Diagnosis.

The diagnosis of this disease will not always be a simple

matter, particularly during non-epidemic periods. As the febrile symptoms simulate other tropical febrile diseases, such as cholera and malaria in their initial clinical manifestations, and as it is possible for no buboes to be present, and even for subnormal temperatures to be contemporary, the clinical history cannot always be relied upon for diagnostic purposes. Again, the buboes are in themselves indistinguishable from the buboes of other diseases. To the general clinical history bacteriology comes as a beneficent aid. I examined bacteriologically the blood drawn from the finger of thirty plague cases. In order to ensure satisfactory results, due attention was paid to the sterilization of parts and apparatus employed, and the following method was mostly adopted :— Having washed clean slides, or in some cases cover glasses in rectified spirits, they were allowed to evaporate, and their opposing surfaces were passed through the flame. The middle finger, after washing with soap and water, was sterilized with perchloride solution. It was finally washed with rectified spirits. Having sterilized the needle by the flame, as many as eight specimens were collected in a single case. The thirty cases examined were mixed, consisting of acute and convalescent cases. The plague bacillus was found microscopically in seventeen cases—more than 50 per cent. By the more delicate test of plate cultivation the irregular square pale blue colonies were recognised in most of the negative cases ; so that it may be fairly well stated that at least 90 per cent. of the cases can be diagnosed thus, by bacteriological diagnosis. Many of the cases which gave negative results from the blood were confirmed from other sources. Very few bacilli, generally speaking, were exhibited in the blood. They were found most numerous in the specimens of blood drawn very shortly before death. Whether this fact would indicate that their number influenced the prognosis gravely is a fair inference. A guinea pig was inoculated with finger blood from an acute case ; it died in five days with the accompanying post-mortem symptoms, and the confirmation of the plague bacillus. Blood drawn from the tail of an inoculated monkey shortly before death was found to be teeming with plague bacilli. The malarial plasmodial elements were found in one case concomitantly, and streptococci in another. Of the thirty cases examined the microscope demonstrated the presence of the plague bacillus in 56 per cent., and 10 per cent. more was obtained by plate cultivations. Thus the examination of the blood gave positive results in 66 per cent. of the cases examined, and henceforth demonstrates the value of blood examination in diagnosis.

Sputum.

Having kept a record of seven cases of prune juice sputum, by the application of Gram's stain, some of the specimens were found to be almost pure cultures microscopically. The plague bacilli were found to be much more numerous in those specimens than in the blood of the same patient. Two guinea pigs were inoculated (23/4/'96) from sputum, and with positive results. Other micrococci, such as staphylococci and streptococci, as in the pus from buboes, were also observed in sputum. The inoculation of guinea pigs with tongue scarf, vomit, gastric juice, scrapings from Peyer's patches, spleen pulp, and fæces, all gave positive results, indicating profoundly the enteric form of the disease.

Skin.

I have examined in over a dozen cases microscopically for the plague bacillus where there was free perspiration, but with purely negative results. Two cases were selected in which the presence of the plague bacillus had already been demonstrated, and plate cultures were made from the cold ante-mortem perspiration, but with negative results. Streptococci were found in the purulent conjunctival pus of two cases. Simultaneously with Dr. Wilm I observed the uniform results of inoculation of animals with such organs as spleen, lungs, liver, doubtful glands, and bubonic pus. Positive results in guinea pigs were obtained mostly in five days. With the discovery of the bacillus the post-mortem appearance observed was mostly similar to those in the human being. Hæmorrhagic, submaxillary, axillary, femoral, and mesenteric glands, together with the spleen were found enlarged. The stomach was found injected. The bacillus was demonstrated in their special organs and fluids.

Experimentation.

As an introduction to the possibility of infection by foodstuffs, I conducted the following experimentations. A chil- was admitted into hospital (2 p.m., 21/4/'96) and died (ad 3.30 p.m.), whereupon an immediate post-mortem was cont ducted. The patient has a febrile temperature, and a small bean-like swelling in the left femoral region. The post-mortem revealed enlarged and hæmorrhagic glands of the mesentery and bronchi; the stomach was inflamed and hæmorrhagic. Peyers patches were swollen and injected, whilst the spleen was enlarged. A cultivation was obtained from the fæces ante mortem; a culture was obtained from the

TABULAR ANALYSIS.—CHINESE PLAGUE.

Fatal Inoculated Animals.	Culture Method.				Cultures.										Culture Feeding.							
	Accidental.	Artificial.	Hypodermic.	Alimentary.	Tongue.	Sputum.	Stomach.	Pyer's patches.	Faeces.	Urine.	Buboes.	Blood.	Pus.	Spleen.	Sweet potato.	Rice.	Chicken.	Pork.	Water.	Milk.	Sugar cane.	Earth.
Guinea pigs ..	4	22	..	..	2	4	1	1	4	2	4	2	2	1	4	..	..	..	1	1	1	1
Chinese pigs ..	..	2	2	..	..	..	..	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..
Chinese white mice	..	3	3	..	..	..	..	..	..	..	3	..	..	..	..	..	..	..	..	..	..	..
Japanese white mice	2	6	2	6	..	..	..	..	..	..	..	..	..	..	..	2	1	2	..	..	..	..
Rats	..	4	4	..	..	..	..	..	..	..	4	..	..	..	..	..	..	..	..	..	..	..
Rabbits	..	4	2	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Monkeys	2	1	..	3	..	..	1	..	1	..	..	..	..	..	2	..	..	..	..	1	1	..
Fowls	..	2	..	2	..	..	..	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..
Cat	..	1	..	1	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..
Pakhoi pigs ..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..

stomach of the child. These cultures were respectively transferred to sterilized milk. Subcultures were again made in sterilised milk tubes. I next inoculated a guinea pig subcutaneously with the subculture from the scraping of the stomach, and simultaneously fed a monkey on the culture from the fæces (ante mortem) 3 p.m., 27/4/'96. The plague culture was inserted into a piece of sugar-cane 3 inches long. A hole was punched out in its interior where the culture was placed, and a cap adjusted to retain it. The sugar-cane was ultimately given to the monkey, which it afterwards devoured. Both animals died on the fifth day after inoculation (10 a.m. and 5 p.m., 2/5/'96). Two days afterwards the monkey became sick, developed a febrile temperature and marked diarrhœa. It picked up on the fourth day and ultimately succumbed on the fifth day. I examined its blood ante mortem, and it was teeming with plague bacilli. The autopsy of the guinea pig revealed prominent submaxillary, femoral axilliary, and mesenteric glands. The lungs and kidneys were inflamed and petechiæ were detected in the stomach. The bacilli were found in the glands and organs. Similar post-mortem appearances were detected in the monkey with positive results. The following deductions can be obtained from these experiments. Both animals died of plague. The infection was by the ingestion of plague-infected food stuffs. The disease can be transmitted by milk, sugar-cane, and fæcal contamination of food stuffs.

Food Supplies.

Three guinea-pigs contracted plague accidentally. They were fed on sweet potato, which must have become inoculated, probably by flies or by carelessness in cleaning the cages. Animals sparsely fed succumbed more readily to plague when inoculated than those which were well fed. I inoculated boiled rice with a pure culture of plague, and fed a Japanese white mouse upon it. The animal died in sixteen hours, and positive evidence of plague was present in the organs post-mortem. Another mouse was fed similarly, and died in two days with positive results. Boiled pork was inoculated with plague bacilli and given to a white mouse. It died in eighteen hours, and with positive results. A piece of boiled fowl was inoculated with a pure culture of plague bacilli from urine. It was given to a white mouse to feed upon. It died in twenty-five hours. The bacillus was found in all the principal organs. A second mouse was allowed to similarly feed upon plague-inoculated boiled fowl, and died in twenty-four hours. The plague bacillus was found to be active

after four days in tap water, roast beef, cheese, soaked bread, cabbage, boiled potato, and tomato by cultivation ; again by cultivation the plague-bacillus was found to be fertile after two days on fish, boiled eggs, carrot, cocoanut, pineapples, and bananas. From a consideration of these facts it is only just to infer that plague may be possibly transmitted by food stuffs, and as a preventive measure, proper cooking of food stuffs is a very weighty matter indeed.

TABULAR ANALYSIS.—CHINESE PLAGUE.

Cultures after Two Days.	No. of specimen	Cultures after Two Days.	No. of specimen
1. Linen	2	13. Pineapple	2
2. Paper	2	14. Banana	2
3. Earth	1	15. Tomato	2
4. Water	4	16. Pork	2
5. Milk	4	17. Ham	1
6. Rice	2	18. Roast beef	1
7. Sweet potato	1	19. Boiled egg	1
8. Boiled potato	1	20. Cheese	1
9. Carrot	1	21. Soaked bread ..	1
10. Cabbage	1	22. Flies	4
11. Lettuce	1	23. Fowl	2
12. Apple	2	24. Chicken	2

Dissemination of the Disease.

Foodstuffs must then be regarded as an important item of the disease. Plague colonies developed after having been frozen respectively for twenty-four and forty-four hours. As factors in spreading this disease sputum, fæces, and urine must be regarded with grave suspicion. In sterliized urine the bacillus was found active after three weeks. The bacillus may obtain access to the human system by the following three tracts, the alimentary, the pulmonary, and the cutaneous. The transmission of this disease by small animals is now classical and historical, and of good repute. The disease can be

transmitted overland by domestic animals and utensils, food stuffs, and clothing, hence it is disseminated along the lines of commerce, and its prevention must always depend on an accurate knowledge of its causation.

Plague Bacillus.

The plague bacillus is a minute vegetable organism with rounded ends, which stain more deeply than the centre. It is decolourised by Gram's stain. It resembles many other bacteria in shape which produce similar septicæmic diseases, and has many points in common with other bacteria, such as the colon and the typhoid. It is killed by sunlight, desiccation, and a number of chemical substances, and is non motile.

ORIENTAL PLAGUE DIET LIST.

Full Breakfast.

Eggs (or fish, 6 oz.)	4 oz.
Rice	12 oz.
Tea	¼ oz.
Sugar	½ oz.

Full Supper.

Chicken	1 lb.
Rice and Curry	12 oz.
Ghee	1 oz.
Sugar	½ oz.

Low Breakfast.

Rice	8 oz.
Eggs (or fish, 3 oz.)	2 oz.
Tea	¼ oz.
Rice	8 oz.

Low Supper.

Chicken (live for soup)	1 lb.

Chinese Full.

Breakfast, 7.30 a.m.		Supper, 4 p.m.	
Rice	12 oz.	Rice	12 oz.
Pork	1 oz.	Salt Eggs	1 oz.
Fish (fresh or 2 salted eggs)	2 oz.	Fish (fresh)	2 oz.
Fresh Vegetables	4 oz.	Fresh Vegetables	4 oz.
Tea	½ oz.	Nut Oil	½ oz.

Low.

Rice	8 oz.	Rice	8 oz.
Fresh Fish	4 oz.	Pork	2 oz.
Tea	½ oz.	Vegitables	4 oz.

CHAPTER II.

The Rat Plague.

(Bī she beng).

Plague will exist wherever the rat is. "The rat plague" or "rat epidemic," as it has been termed for centuries amongst Orientals, is the general precursor of human plague. As it (the rat) has two or three litters in the year, it demands time in its nomadic excursions. Rat migrations on land or sea has always been regarded as a bad omen. They have been known to repeatedly evacuate a plague-stricken district completely. A good object lesson of rat migration was observed during the advent of plague in China in 1894. There was not a single case of plague in Macao,* a Portuguese settlement and pleasure resort in South China, only a few hours sailing from the infected areas, Canton and Hong Kong, where thousands were dying, and whence visitors were daily calling. Now, when there was no plague in South China during 1895, rats began to die in the houses in Macao. Much of the obscure in plague mystery can be explained by "mus." When introduced by rats, plague is an endemic disease like yellow fever, malaria, or beri beri. Its endemic nature is conspicuous, and its double infectivity—man and the lower animals—augments the sphere of an epidemic.

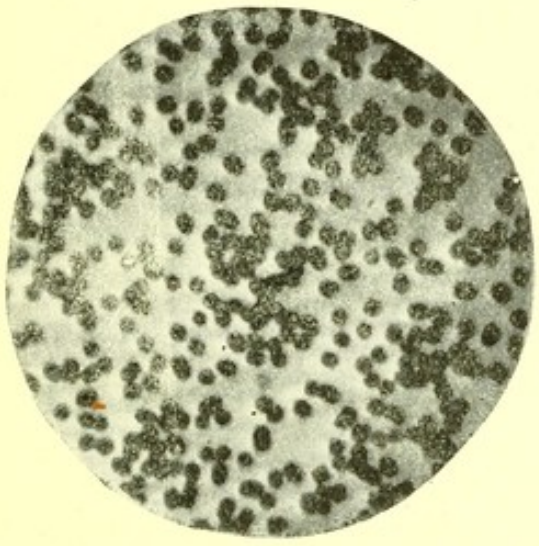


Fig. 60.—Microphoto plague bacilli, culture from spleen. X 1000. [*Nelly.*

Why the disease historically appears and disappears suddenly; why one person is affected in preference; why one floor of a house is only affected; why one district is passed over and not another; why there are more cases after the rains; why one-third of the year is the plague season: can be greatly elucidated by mus.

If the rat plague confines itself to rodents, human plague mortality will be low. If, however, as in India and China, it extends to other domestic animals, the human mortality will ascend. Human mortality is directly pro-

* Shanghai enjoys a similar immunity.

portional to the height in the scale of animal fatality. The flea* plays a dramatic part in plague (Simond). Although fleas and rodents are prevalent in Japan, but not the bed bug, pigs, sheep, goats, or cattle, yet plague has never made much progress there for some reasons. The mortality will be very high indeed as in South and North China if the disease attacks the lower animals in general.

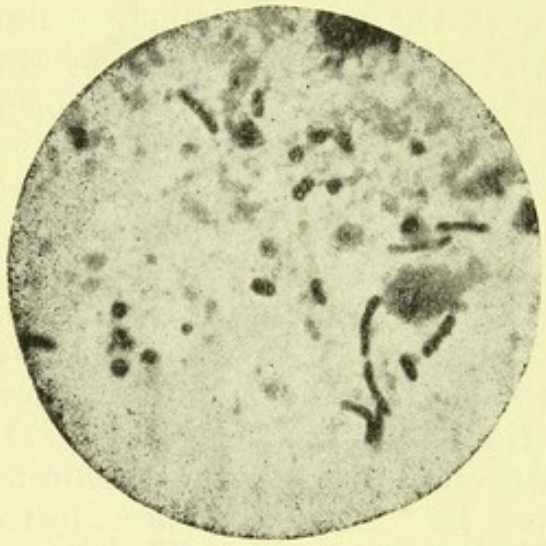


FIG. 61.—Microphoto plague bacilli from bubo of rat. X 1000. [*Nelly.*

1. FORECASTS.—Knocking at the door as plague forecasts will be observed migratory species of insects, animal mortality, a graft of glandular enlargements upon diseases, and a special increase of already existing glandular disease. For about two months an undeveloped species of house fly was observed in one locality which disappeared. Insect pests in the East result in famines.

In its infectivity, plague resembles acute tuberculosis. The glands of the neck may be inoculated from the mouth, the mesenteric from the ailmentary tract, the glands of the axilla and groin from wounds of the limbs. The miliary pseudotuberculosis of inoculated animals is similar to what occurs when animals are inoculated with dead tubercle. The post-mortem appearances of inoculated plague animals do not predicate pest only, but a foreign body. The tubercle bacillus is further pleomorphic like the pest bacillus. Actinomycosis, which is a glandular disease due to a fungus-like tuberculosis, is

FIG. 62.—Microphoto plague pleomorphs, hyphomycetes, alkaline culture media. X 1000. [*Nelly.*

* Pulex philippinensis does not bite human beings (Herzog).

believed to be infective from grain. The house fly is not the plague in the East that it is in other countries, excepting in silk districts such as Canton, where the disease first broke out in 1894. Plague is conspicuously absent in sugar plantation districts. The pest bacillus finds its intermediate host in nomadic rodents and insects, and possibly in birds which feed upon grain. The field rat, which is exposed for sale in Oriental cities as edible, lives mostly upon grain.

Fig. 63.—Microphoto plague pleomorphs, hyphomycetes, alkaline culture media. X 1000.

[*Nelly.*

2. Plague and Famine in ancient times were usually contemporary. Further, it was usually associated with epidemics due to diseased grain such as petechial fever, due to ergot of rye, which appears to be indicated like yeast in the treatment of plague. Insect pest is generally associated with the destruction of grain and animals.

The mortality amongst rodents, particularly in the vicinities where plague patients have been, is now historical, and also a special development of glandular disease on their attendants is notorious. Not a few have been cognizant of an acute epidemic of glandular enlargements, apparently causing little trouble—pestis minor and pestis ambulans. It is beyond dispute that glandular swellings do complicate contemporarily for months other diseases. These are the harbingers preparing the lymphatic system for the pest. Glandular enlargements were found in 30 per cent. of the rodents examined in Canton in 1894 (Rennie).

Were it always as infectious from man to man, as it is from rat to rat, the human race would be somewhat threatened with dissolu-

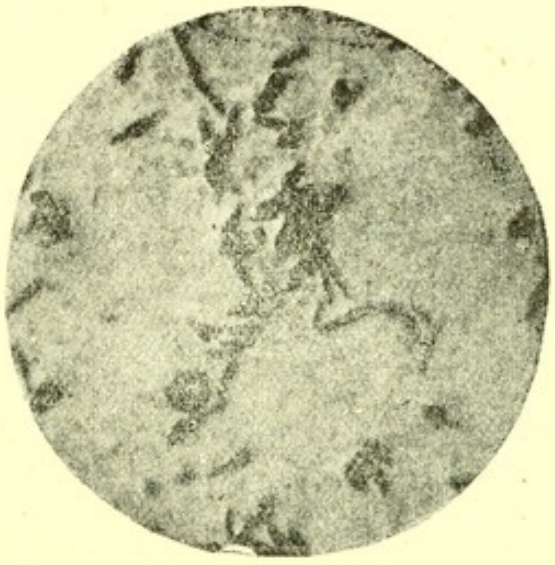


Fig. 64.—Microphoto plague pleomorphs, hyphomycetes, alkaline culture media. X 1000.

[*Nelly.*

tion. There is a total fatality when the unaffected rat is associated with the artificially inoculated.

3. How Does the Rat Become Affected ?—Plague is historically associated with the importation of grain, the staple diet of rodents. In the perpetuation of the disease a plague infected rodent is a greater menace than a plague patient because the granary may furnish suitable media for the cultivation of the pest. The infectivity of rodents is now classical when fed upon plague food stuffs. Small animals, fed upon boiled rice inoculated with pest, very quickly succumb.

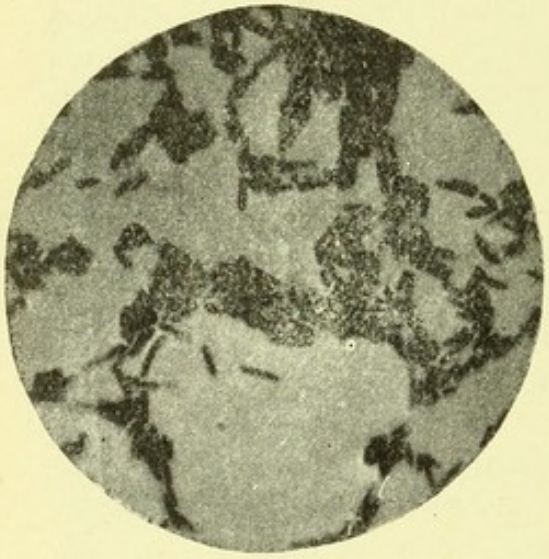


Fig. 65.—Microphoto plague pleomorphs, blastomycetes, salted agar. X 1000. [*Nelly.*

A prisoner that contracted plague in jail was known once to be fed upon nothing else but rice ; and, as the pest bacillus lives upon various food stuffs, including preparations from wheat, it is natural to infer that the staple diet of rodents becomes inoculated with some species of imported insect of a migratory habit, and thus establishes the rat plague. Rats become inebriated with plague. Their staple diet is fermentable. The edible field rat is not unknown in China. The pest bacillus in its fungoid capacity may adapt itself to the staple diet of rodents which in turn will affect higher animals and man. Rodents become affected over a wide area due to some plenary inoculating agents such as insects.

Fig. 66.—Microphoto plague pleomorphs, algal, old alkaline media. X 1000. [*Nelly.*

4. A Pseudo-Tuberculosis.—As plague, however, dies out through serial inoculation of rodents, it is quite possible to over-esti-

mate their share in the procession of the disease. Hydrophobia, like pest, has associated with it the yeast cell in the central nervous system, and is produced by inoculation from the lower animals. Whilst pseudo-tuberculosis of pigeon dealers infected in all probability from grain is related to the aspergillus family.

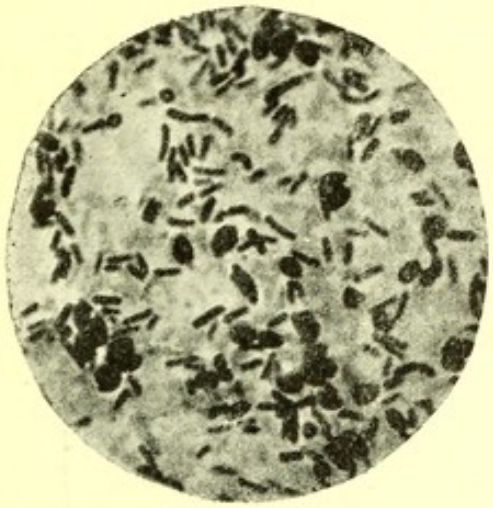


Fig. 67.—Microphoto plague pleomorphs, blastomycetic, old salted agar. X 1000. [*Nelly.*

Scientifically speaking there is no pseudo-tuberculosis, because the tubercle bacillus, like the plague bacillus, is not a bacillus at all, but a higher pleomorphic organism.

Bubonic plague is pathologically an acute pseudo-tuberculosis. Bipolar staining of the bacillus pestis is common to many others; stalactites belong to the yeasts; and the acute miliary tuberculosis of animals inoculated with the plague is a common property of foreign bodies. Small animals, when inoculated, may take as long to succumb as if they had been inoculated with tubercle instead of bubo.

The various types of the disease are produced when the oscillating leucocytosis and hæmorrhages are distributed over the various systems fostered by the bacillus pestis. In the lower animals, then, plague stamps itself as a pseudo-tuberculosis.

5. Experimental, the Cat.—The cat can become a favorite host for the dissemination of plague owing to the high alkalinity of its saliva and that of rat's blood. Having made cultivation experiments with the saliva of a number of cats, a bacillus and micrococcus was found to be present in the proportion of 6 : 1. Three young rats were fed upon cultures of the cat's salivary micrococcus mixed

Fig. 68.—Microphoto plague pleomorphs, blastomycetic, salted agar. X 1000. [*Nelly.*

with bread and two were inoculated hypodermically. Three young rats were also nourished on Bouillon and Blood Serum cultures of the cat's salivary bacillus (bacillus felis septicus) mixed with bread; and two were, moreover, inoculated subcutaneously, one in the nose and one in the hip. All those ten inoculated animals gave negative results with the cat's salivary bacilli; but still the bacillus felis septicus is fatal at times to young rats and mice (Fiocca). Vide bibliography, Faulding's.

*Fig. 69.—Pathological appearance of guinea pig inoculated with plague bacilli demonstrating miliary pseudotuberculosis of spleen, chronic plague (Hunter). Queensland specimen, Brisbane.

* For Figs. 58, 59, 69, I am indebted to J. Desmond, Govt. Vet. Surg., S.A.

6. EXPERIMENTAL, THE RAT.—I inoculated one of three large rats in the nose with a bacillus (N) from dust. It died in a day and a-half after inoculation. A companion rat in the same cage died a day and a-half later. Three days later rat III. also died, and, although well supplied with food, it had devoured portions of the fore limbs, lower jaw, and thorax of its companion. Such serves to illustrate respiratory and alimentary infection amongst rodents.

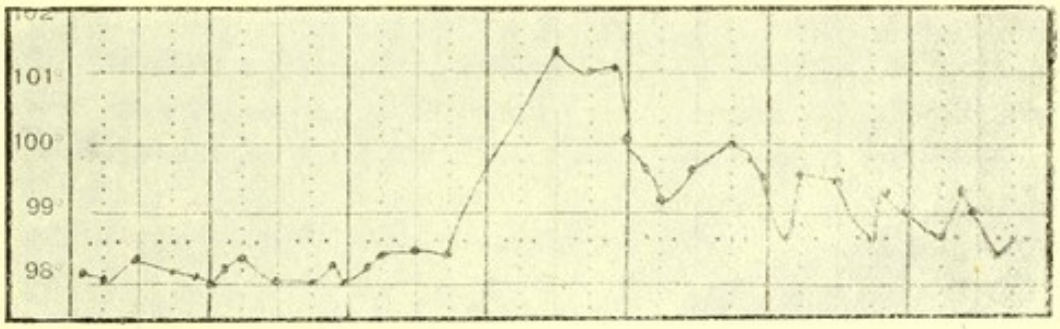
KENNEDYTOWN HOSPITAL.

FIG. 70 (i.).—L.S., age 26; male. Bubonic. Chinese.

22nd April, 1896.

FIG. 70 (ii.).—L.S. (continued).

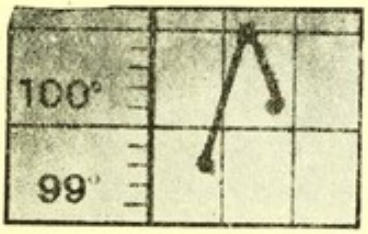
30th April. 1st May. 6th.



Discharged 7th July, 1896.

FIG. 71.—U.K., age 3; male. Inguinal Bubonic. Chinese.

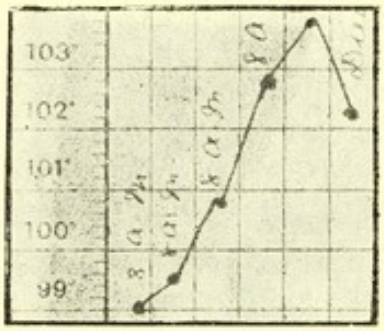
4th May, 1896.



Died 5th May, 1896, midnight.

FIG. 72.—Monkey; inoculated by mouth; feeding. Plague.

27th April, 1896.



Died 2nd May, 1896.

CHAPTER III.

Rapid Method for Plague Histology.

Requisites.—Microtome, iron plane, corks, petri plates, beakers, glass droppers, ethyl chloride, needle holders, cover glass, slides, blotting paper, absolute alcohol.

1. *Initial.*—All specimens preserved in spirit and various preservatives must be soaked in water for some time with repeated renewal of the water before they can be sectioned by the method. It is, therefore, a very rapid method for fresh tissues, crude and raw material which require rapid sectioning and diagnosis. In transit specimens should be immersed in salt, covered with a cloth soaked in carbolic acid or in weak solutions of formalin. The fresh tissue is cut into small square blocks, or in thin slices. The tissue is taken fresh from the animal or patient and placed in the microtome. All the apparatus is portable, and the tissues are sectioned immediately at room temperature. If the animal or patient is under an anæsthetic, an opinion can be given in almost a moment. A counterpart is found in the technique for typhoid fever diagnosis. The sections of the tissues are moreover in their pristine condition unaltered by the process. Structural disorganization of the tissues is thus avoided. Since the plague bacillus is decolourised by Gram's method, and the pyogenic group of bacteria are not, plague histology can be accurately and rapidly demonstrated.

2. *Description.*—A cork suitable to the size of the well of a microtome is split down the centre with a knife. Being portable and convenient I have always used a Cathcart's microtome, but any other having a well is equally convenient. According to the shape of the piece of tissue to be inserted, a small notch at the top to form a pocket is cut out of the central slit in the cork. In actual practice stock corks with various shaped notches are kept. The fresh tissue is inserted in the pocket of the cork, and the latter is now placed in the well of the microtome. At times it is unnecessary to make the pocket, particularly if the tissue to be inserted is a very thin slice. It is then frozen on the one-half of the cork with ethyl chloride, and covered over with the other, and inserted in the well where it is screwed lightly at first. The cork is held on the plane of the well or a little above. The piece of tissue allowed to project above the plane of the cork varies from a sixteenth to a quarter of an inch.

3. *Freezing.*—A tube of ethyl chloride or anæstile is held in the left hand, and an ordinary glass dropper filled with dis-

tilled water s simultaneously held in the right hand. The ethyl chloride spray is allowed to play on the tissue, and drops of distilled water are allowed to fall around the pocket. A layer of ice and snow instantly forms, and is followed by an embankment of ice around the tissue. This is advisable if the tissue is in a thin slice. It should be surrounded by a good embankment of ice. If there is plenty of tissue to spare, it should be pared, before freezing, on the cork like a pyramid. The tissue is now brought level to the plane of the section cutter. If the tissue is frozen too hard, it had better be thawed by passing the finger rapidly over it. The large metallic tubes can be refilled. Trouble often arises with the small glass tubes which refuse to spray. They had better be discarded. One soon learns to use little of the spray by simply freezing the top to obtain sufficent material for diagnosis. A very convenient and useful method is to fill another glass dropper with ethyl chloride. This is instantly attached to a perforated sprayer from a discarded small ethyl chloride tube by india rubber tubing four inches long. A glass dropper is then held in either hand during the freezing process.

4. *The Section Cutting.*—If there is plenty of tissue to spare an initial piece is cut off the top by one sweep of the razor, a knife, or a joiner's iron plane, which is kept for the purpose, and always with a good edge on it. A sharp iron plane cutter is firmly held in the right hand with the bevelled edge looking backwards; whilst simultaneously the index finger of the left hand is kept on the screw of the microtome to regulate the sections. The action of both hands is indiscriminate. This is followed by a very rapid motion of the iron plane at the rate of about sixty to and fro oscillations per second, whilst the index finger of the left hand indiscernibly regulates the sections simultaneously. The iron plane plays horizontally over the tissue. Hundreds of perfect sections in their pristine condition are thus cut from the fresh tissue in the well of the microtome. Such a sectional series is not, however, required for ordinary diagnosis. For immediate diagnosis one or two sections may be all that is required, and one can attain to such dexterity with the method that with a few sweeps of the plane we have all the material desired.

5. *Fishing.*—A section can be instantly obtained by immersing the edge of the iron plane in a glass beaker, a watch glass or petri plate filled with distilled water, and allowing a drop or two of absolute alcohol or rectified spirits to fall amongst the sections on the iron plane to which they adhere.

When they instantly gyrate and fly off at a tangent on the surface of the water they can be easily and immediately caught on a glass slide, or on a cover glass held by an automatic clip. The sections further can be licked off the iron plane with a brush, needle holder, or spatula, and immersed in the petri plate containing water.

6. *Toilette.*—The sections can be bottled, labelled, and laid aside as required in distilled water, with a dash of spirit and carbolic acid, or in weak solutions of formalin. Permanent canada balsam cover glass specimens are made by laying the one cover glass upon the other, a gum label is folded upon itself, and a piece punched out varying in size and shape to that of the section. This is gummed on both sides, and when folded upon itself it is very portable. They are easily made, and cheaply destroyed. Large numbers of them can be easily compressed into small bulk, as they occupy less space than glass slide specimens. The sections can be subjected to any, or all, of the various methods of staining according to one's own taste, or staining, in rapid diagnosis, may be dispensed with. Artificial staining can be instantly instituted by placing mixed colour reflectors between the mirror of the microscope and the specimen. The contrast colours can be according to one's own taste by placing rings of coloured Gelatine, or squares on separate cover glasses between the lens and the mirror of the microscope. The sections are picked up on the glass slide. All the staining, dehydrating, and clarifying is completed with droppers or dropping bottle, without again removing the section. The specimen is rapidly immersed in water when washing it. Everything is absorbed on the slide with small pieces of blotting paper.

7. *Technique.*—All the various tissues can thus be rapidly examined bacterioscopically and histologically. The method is simple and quickly learned. When proficent one will not again return to the barbaric method of imbedding in parafin, and wait for weeks of hardening. Tissues can be frozen, sectioned, and placed under the microscope appearing stained in a minute. Whereas in ordinary routine work permanent microscopic specimens of any tissue can, in two or three minutes thus, be taken from the animal or patient. In this short space of time specimens can be frozen, sectioned, stained, and permanently mounted even while the subject is under an anæsthetic. The specimens thus prepared are perfect in every detail, so that an opinion can be given in a moment at times as in serum diagnosis. The tediousness of the older methods is thus avoided. Bacterioscopically, it

is of paramount importance. Time is an important element in diagnosis, and this led the writer to develop this method which has been easily learned by others. For a number of years the writer has used this method, and has sectioned numerous animal and vegetable tissues by its technique. The simple apparatus, the simple methods, the rapid staining, and the perfect results become a valuable clinical and bacterioscopic aid in the diagnosis of disease. It serves as an instantaneous method also in pathological histology.

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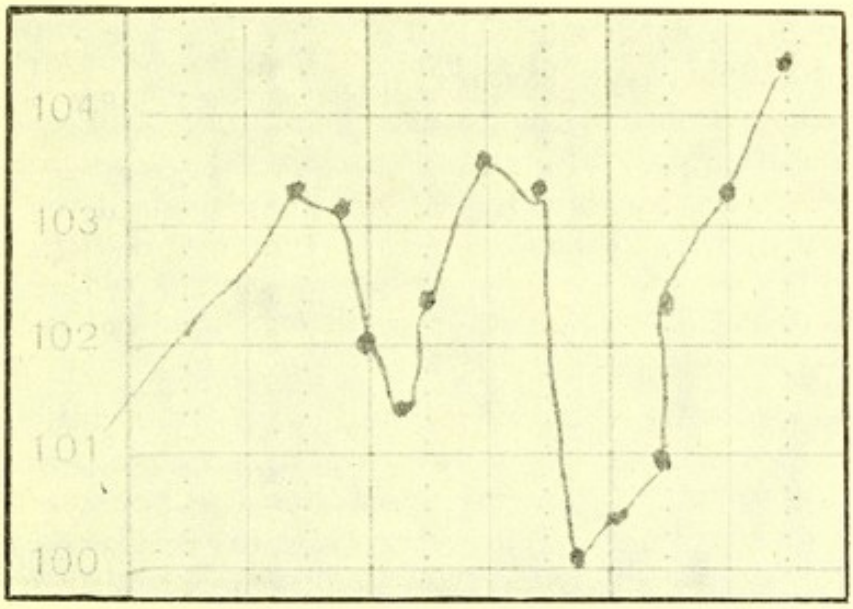
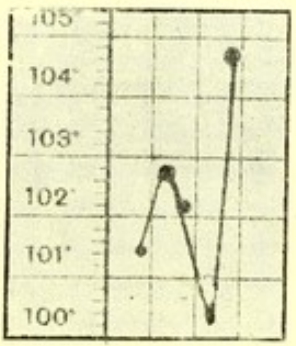
FIG. 73.—N.W., age 32; male. Bubonic. Chinese.
16th May, 1896.



Died 18th May, 1896.

FIG. 74.—W.S., age 25; male. Bubonic. Chinese.
3rd May, 1896.



Died 5th May, 1896.

CHAPTER IV.

A. Experimental Digestive Plague.

1. *Dietaries.*—Like others, Orientals have generally two meals a day, one in the morning and one in the evening. Among the poorer classes, however, they have in addition a midday meal. Disease would be much more prevalent among them were it not for the fact that they cook their food well. Rice is their staple article of diet; fish among the Japanese being next in order, and fowl among the Indians and Chinese. Eggs and vegetables are the next most prevalent foodstuffs. Fruits, which are rarely cooked, are lavishly indulged in. The use of meat as an article of diet amongst Orientals is somewhat restricted particularly on account of the wide prevalence of Buddhism and Mohammedanism, and they are thus a more or less dyspeptic race, owing to their staple rice dietary. The Chinese dietary is a very wholesome one, containing plenty of animal and vegetable foodstuffs. The animal foodstuffs are generally boiled before they are exposed for sale. Fish is usually salted if not sold over night. Fowls are plucked, boiled, and then exposed for sale in Oriental shops; and, although, they may have been thus exposed for days, the buyer does not boil them again; they are simply cut into pieces and heated with warm water. Such is also true of pork. The field rat is skinned and exposed for sale in Chinese cities. Its principal food is similar to the Oriental staple, rice; doubtless the house rat is often sold in its stead. Although the Oriental dietary is spicy and fragrant not only with condiments, but also with decomposition products, the chances of the survival of the pest bacillus in foodstuffs are very remote indeed, owing to indigenous bacterial antagonistic symbiosis. However, fresh and sterilised foodstuff forms the cardinal starting-point of gravity in Oriental as in other dietaries. The soil is then inviting.

2. *Refrigeration.*—Three plate cultures of the plague bacillus were subjected by the writer to a freezing temperature. One of them was frozen for twenty-four hours, and the two others for forty-four hours. They were afterward developed at room temperature for three days. The number of colonies that developed in a given time was inversely proportional to the duration of freezing; that is, a greater number of colonies were visible in the plate which had been treated for one day, than in the plates which had been treated longer.

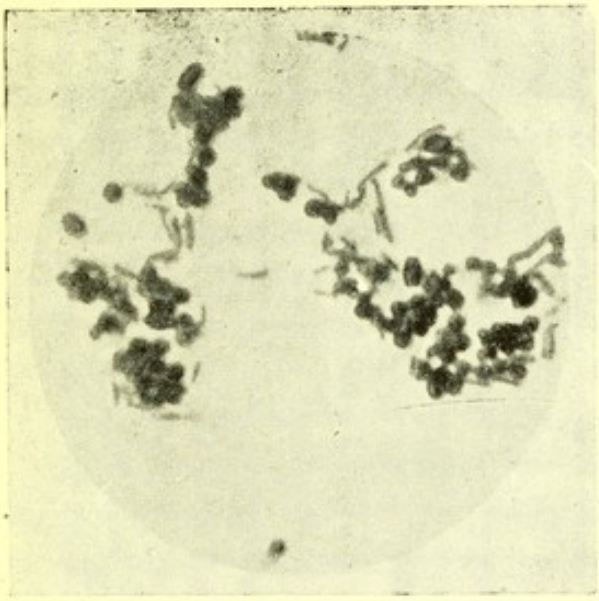


FIG. 75.—Microphoto plague pleomorphs, blastomycetic, salted agar. X 1000. [*Nelly.*

3. *Salting.*—When salted Agar cultures of the pest bacillus have evaporated it is often observed that a perfect salt cake crystallizes out, demonstrating how it is possible for the pest bacillus to exist on salted foodstuff such as fish. It is related to the marine bacilli in its chloretic habits.

4. *Alkalinity and Acidity.*—Although the cat does not always (Hunter) succumb to experimental plague when fed with plague-infected food, it is possible that its saliva may become the domestic host of the pest bacillus; its saliva, being highly alkaline, is an excellent soil for the pleomorphs of the bacillus, which has to contend with only one or two other indigenous species. Owing to its behaviour in acid media, and the fact of its acid-production, it should survive the action of the gastric juice. Plague bacilli lived for two days in a half per cent. solution of hydrochloric acid (Wilm),

5. *Feeding.*—The three principal portals of plague infection — the pulmonary, the alimentary, and the hypodermic — have their comparative importance. The pest bacillus has been identified throughout the whole extent of the alimentary tract, further autoinfection being sustained by the reabsorption of the bile. Positive results by alimentation with plague foodstuffs have been obtained in monkeys, fowls, rabbits, guinea-pigs, and rats, the latter succumbing after devouring their own dead in preference to other food. Positive results were obtained in two days by cultivation with the following

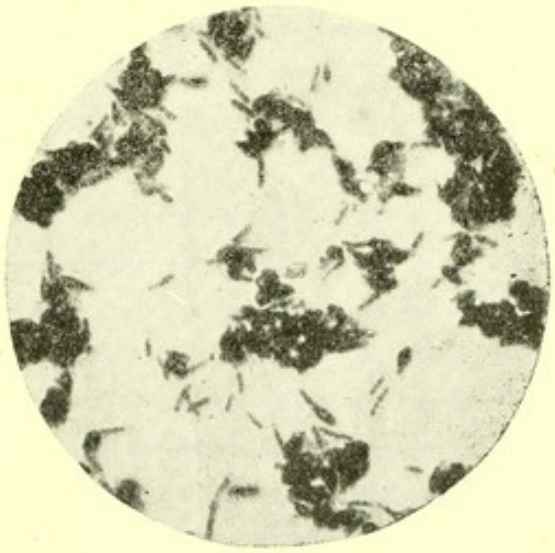


FIG. 76.—Microphoto plague pleomorphs, hyphomycetic, omycetic, and blastomycetic, salted agar. X 1000. [*Nelly.*

boiled foodstuffs which had been inoculated with pure cultures of the pest bacillus, viz., rice, eggs, fowls, pork, fish, beef, and bread: also with the following vegetables and fruits, boiled, viz., cabbage, lettuce, carrot, potato, tomato, apple, banana, pineapple, sugar-cane, and cocoanut. The pest bacillus lives well upon saccharine and fatty matter, on cocoanut milk, butter, and ordinary milk. The following five animals fed upon plague-inoculated, boiled foodstuffs gave positive results, while their controls gave negative:

Japanese white mouse I. was fed upon sterilized rice which was inoculated with a pure culture of the plague bacillus. It died in two days. Positive results were obtained with the lungs, liver, and spleen. The stomach and bladder were observed full at the autopsy.

Japanese white mouse II. was fed upon boiled rice inoculated with a pure culture of the pest bacillus as in the former animal. After having diarrhœa it died in sixteen hours. Positive results were obtained with the stomach, lungs, liver, and kidneys.

Japanese white mouse III. was fed upon boiled fowl inoculated with a pure culture of the pest bacillus. It died in twenty-five hours. It had staggering gait, its hind legs were flexed and wide apart; and when excited it jumped like a frog. Antemortem, its saliva gave positive results. The lungs, liver, blood of heart, and the bone-marrow of the lumbar spine, post mortem, gave positive results.

Japanese white mouse IV., fed upon boiled fowl inoculated with a pure culture of the pest bacillus, like mouse III., died in twenty-four hours. When excited it reeled about from side to side, with its hind legs wide apart. The internal organs gave positive results.

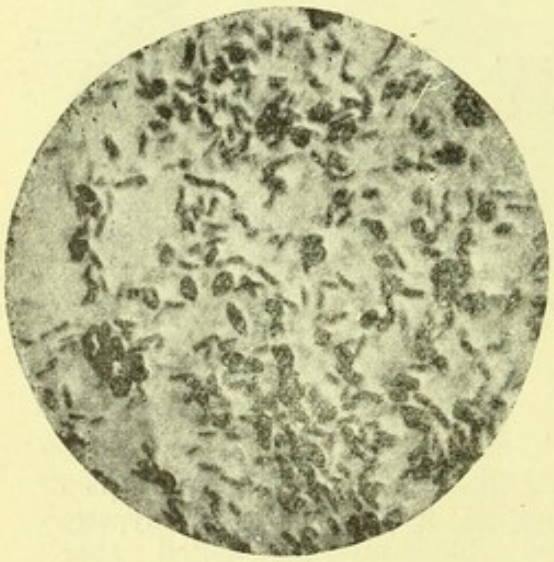


Fig. 77.—Microphoto plague pleomorphs, omycetic, salted agar. X 1000. [*Nelly.*

Japanese white mouse V., fed upon boiled pork inoculated with a pure culture of the pest bacillus, died in eighteen hours. The pork became peptonized. Positive results were obtained with the lungs, liver, kidneys, stomach, and intestine.

The cultures in the above

experiments were furnished me by Staff Surgeon Wilm of the German Asiatic squadron, whose work is referred to in the translation of the Berlin Plague Congress, 1899, by the Queensland Government.

6. *Digestive Vaccines.*—Sterilized plague vaccines are highly nutritious substances, and when animals are fed upon them immunity is conferred. The writer has administered seventy-nine doses to human beings per os. Beginning with ·04 gramme the writer swallowed the vaccine, a detailed account of which is given in the appended chart. A case of phthisis pulmonalis, to which a continued course of plague nucleoproteid was given, was greatly improved and spontaneously left the hospital, stating that he felt nothing wrong with him now; ·04 gramme, taken in some peppermint chloroform water, was subsequently found to be a convenient quantity when divided into three parts, one of which was taken thrice daily. This is similar to feeding with the essential fat of the tubercle bacillus.

*Health, which we enjoy, no matter how it is obtained and maintained, is detrimental to plague. The best antidote is plenty of good food, fresh air, and sunshine, and the nearer we approach to Nature's method the more perfect will be the immunity. There is ordinarily sufficient iron in foodstuffs, yet there are times when anæmia does occur, and when assimilable iron compounds are furnished it is rectified. Plague makes its abode only in the system that has a poverty of "nuclein," its natural defence in the fluids and organs of the body. This poverty can be rectified by its internal administration. Plague is an acute nuclein œdema of the lymphatic system which may be localised or generalized. In civilised countries it is not at all likely that people will turn up annually to be vaccinated and revaccinated, although efficiency of immunity depends upon this; and many will not have plague germs put under their skin, but rather run the risk of getting the disease. Plague is an acute phosphorus poisoning with the plague bacillus, which is very fatal to man and rats. Successive generations of the plague bacillus on culture media, however, ultimately become inert. Plague nucleinate is a highly phosphorized substance, a good nerve tonic, like coca, and a food for young growing animals. In concentrated doses it is as nutritious as bread, milk, and eggs. Thus without the slightest inconvenience we furnish the human system with its own natural defence against this malady. Immunity can be established and maintained in

* Vide Plate III.

several days. Nuclein, the essential element of the plague bacillus, belongs to a tribe of proteid compounds resembling the antiseptic albuminates of silver and mercury. The nucleinic acid of nuclein combines with metallic and non-metallic salts, forming nucleinates insoluble in acids and soluble in alkalies. They resist peptic and tryptic digestion, and are slightly soluble in oils, fats, and mucin. As found in normal blood the alkaline nucleinates are bactericidal and externally are good antiseptics. They are recommended therapeutically for rheumatism, cancer, and infectious diseases. Nucleinate of iodine is used in myxœdema, and nucleinate or iron in anæmias and tuberculosis; milk nuclein, egg nuclein, liver nuclein, thyroid nuclein, and spleen nuclein, all differ from one another. In Haffkine's plague vaccine the nuclein therein contained is not suitable for assimilation unless very large doses be ingested in an alkaline solution, and then at the expense of nausea. Plague gland nuclein, like all other nucleins, is best given by the mouth. Its hypodermic administration is not adapted for a continuous course of the nuclein treatment. Plague nuclein is an albuminate or nucleinate of phosphorus, and intimately associated with mucin.

All nuclein treatment requires therapeutically a continued course, and in the prevention of plague this is clearly indicated. Plague nucleinate is the essence of living animal and vegetable cells—a concentrated natural foodstuff. Plague nuclein, the essential immunising substance of the plague bacillus, is a good physiological nervine, with mild diuretic properties. Externally and internally it has good antiseptic and bactericidal properties. In its transit through the stomach it is not acted upon by the gastric, or later by the pancreatic, juices, but is dissolved and absorbed in its original form in the intestines. Thus it does not interfere with digestion, has no effect on the temperature, pulse, or respiration, but acts as a mild tonic on the lower centres of the spinal cord, and furnishes the system with a defensive substance. Like quinine against malaria, iron in anæmia, or thyroid tablets against myxœdema, it may in endemic plague be taken with advantage periodically. As found in Lustig's prophylactic, it is a very nutritive substance, a chemical food (although yeast nuclein would act equally as well), and a charming ideal method of preventive medication to resist an affection of the lymphatic system with the plague bacillus. By its periodic ingestion it will furnish lymphatic glandular bactericidal foodstuff as is already formed in the human system. It is perfectly harmless and innocuous.

In some respects there is a very striking analogy between the tubercle and the plague bacillus. It differs in being achloretic. Both produce miliary tubercles in inoculated animals, and have a steatogenic basis mostly peculiar to artificial media. In the latter connection the pest forms aquatic

KENNEDYTOWN HOSPITAL.

FIG. 78.—W.C., age 55; female. Bubonic. Chinese.
21st May, 1896.

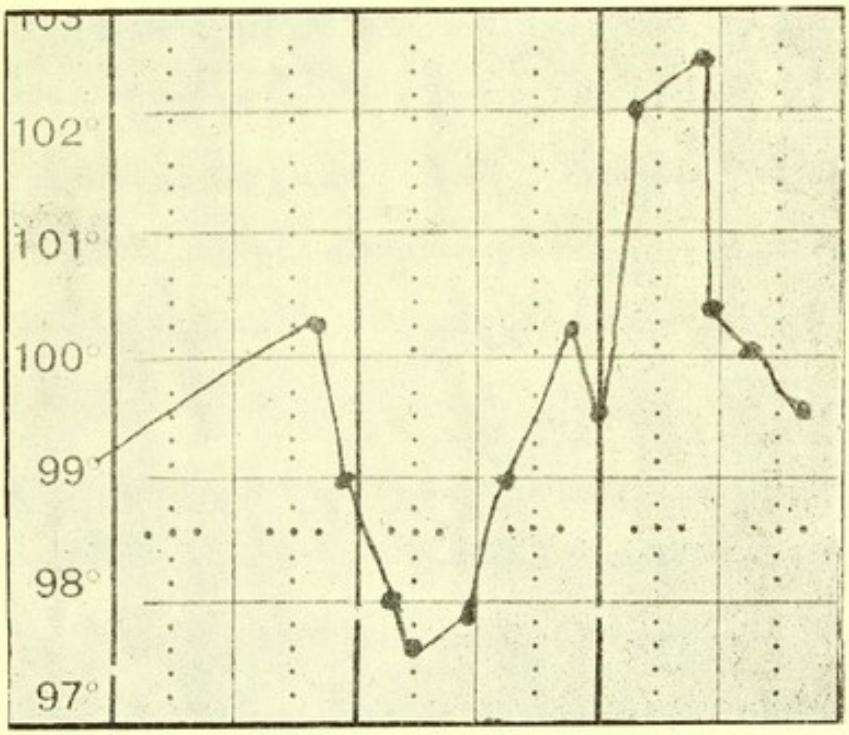
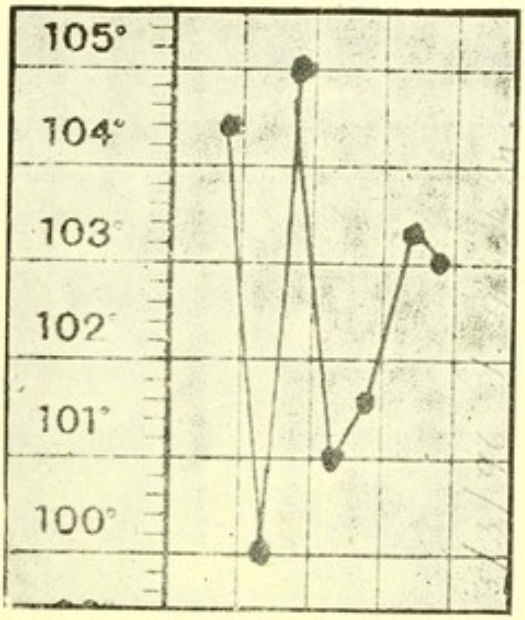


FIG. 79.—C.Y., age 17; male. Bubonic. Chinese.
2nd May, 1896.



Calomel.
Died 5th May, 1896.

SPECIAL REPORT.

Name, D. MacDonald. Date, 4th July, 1900.

Hour.	Condition.	Food and Drink.	Temp.	Pulse.	Ecws.	Urine.	Instructions and Remarks.
10.30 a.m.	Splendid.	.....	.....	..	o	Passed.	
11.30	Active.	One bottle of Lustig's plague nucleo-proteid, .04 gramme, containing sufficient for vaccinating four persons in twelve sittings. Mortal dose for one monkey and two large rats. On empty stomach in a little milk very palatable. [Taken in the presence of Drs. Frost and Gocher.]					
12 noon	Head clear.						
12.0	Active.	.....	98.4°	84	..	℥ xii, normal.	Respiration normal; slight exhilaration; slight rectal and vesical tenesmus; pain in right shoulder.
1 p.m.	Hungry.	.....	.....	..	..	℥ x, normal.	Midday meal; vesical and rectal tenesmus.
1.30	Grand.	.....	.....	..	..	.....	Transient frontal occipital twitches; slight pain in right groin momentarily; vesical and rectal tenesmus.
2.25	Active.	.....	.....	..	..	℥ x, cloudy, neutral, sp. gr. 1010.	Feeling pains in the groins.
3 p.m.	Splendid.	.....	.....	..	..	No albumin, no sugar, phosphates increased.	Abdominal discomfort and frontal occipital discomfort; rectal and vesical tenesmus.
3.15	Active.	Mouth dry, but not thirsty.	.....	..	..	.....	Slight cardiac palpitations.
3.35	Active.	.....	.....	..	..	.....	Nasty frontal occipital discomfort; bleeding from gums (slight); slight incoordination when walking.
3.40	Slight frontal occipital fulness.	.....	.....	.	o	.....	Faint cardiac palpitation.
4 p.m.	Active.	A cup of tea and bread.	.....	..	..	.....	Slight cardiac dilatations; nasty fulness of head as if one had a cold.
5 p.m.	Active, slight nasal catarrh.	.....	.....	..	..	.....	Fulness of head (frontal), as if one has recovered from a narcotic.

5.30	All right; fulness of head less.	Mouth moist.	.....	..	..	℥ x, normal.	Abdominal fulness; slight rectal tenesmus; vesical tenesmus; cardiac dilatation.
6 p.m.	Hungry, active, slight sense of vertigo when walking.	Dinner; had good meal; mouth inclined to be dry, but not thirsty.	.....	..	..	.....	Transient noise in ear; head slightly throbbing; frontal dulness; slight cardiac palpitations and dilatations; vesical tenesmus absent; slight rectal tenesmus.
7.20 p.m.	Active.	.....	.....	..	..	℥ xi, normal.	Rectal tenesmus; faint frontal fulness.
8 p.m.	Active.	One cigar.	98°	80	..	.....	Respiration 25.
9 p.m.	Hungry.	Tea and biscuits.	.....	..	..	.....	Transient abdominal colic; pain in right groin and right iliac region.
10 p.m.	Active; occipital fulness; rectal and vesical tenesmus have disappeared.	.....	.....	..	..	℥ vi, normal.	Slight abdominal cramp.
11.15 p.m.	Excellent.	.....	.....	..	..	.....	Slight cardiac dilatation.
11.30 p.m.	Excellent.	.....	.....	..	..	.....	Symptoms subsided.
12 midnight	Bed.	.....	97.8°	80	..	℥ vi.	Respiration 18. Slight abdominal colic, ¼ hour.
	Slept well.						
July 5, 1900.							
8.30 a.m.	Splendid.	Breakfast.	.....	..	..	℥ ix.	
9.30	.....	.....	.. ...	..	o		
10.15 a.m.	.....	.....	.....	..	..	.....	Return of rectal tenesmus, transient, when walking; bleeding from gums slight; faint cardiac dilatation.
11.30 a.m.	Well.	A cup of tea.	97.6°	..	..	.....	No symptoms present, but slight transitory rectal tenesmus.
						Total, 24 hrs., ℥ 74	
	All symptoms subsided; symptoms compressed into twelve hours.	Hungry for all meals; tonic; good general tonic.	Circulating and nervine irritant tonic; no sickness.		No Diarrhœa.	Diuretic.	

stalactites in nonchromatic material like cocoanut oil or ghee. Whilst the tubercle bacillus on the other hand elaborates its TC (Behring) or vegetable cholesterin fat from glycerined media and also from fatty substances; and it is in this relation, moreover, that we encounter similar steatogenic rhizomes in Nature—the root tubercles of the great flesh and bone-forming family (Medicine).*

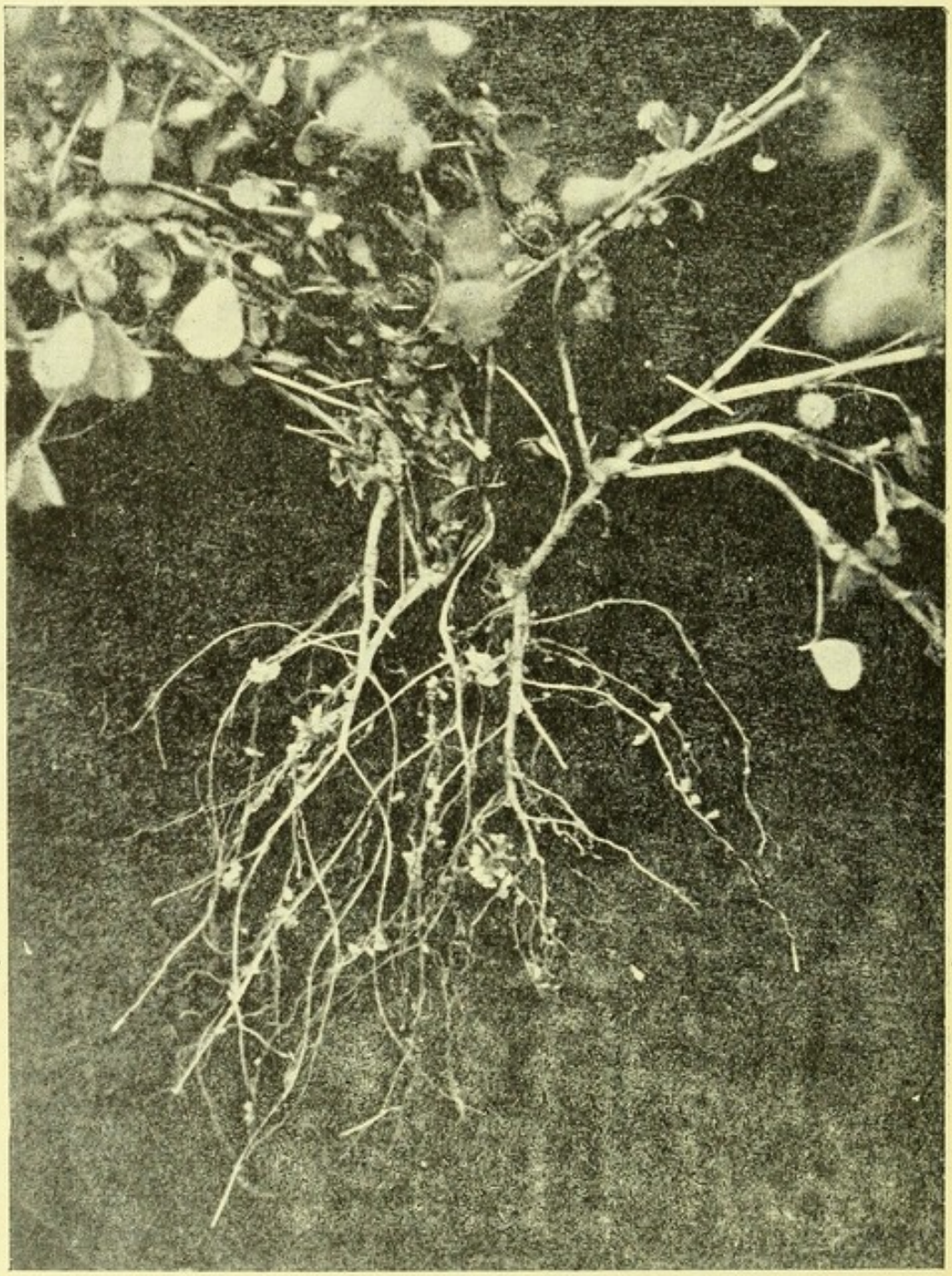


FIG. 80.—Botanical tuberculosis, bacteria tubercles on the rootlets of Medicago denticulata. Analogous parthenogenetic vegetable plant lice (aphides), apterous and pseudo-apterous; the nitrogenous milch cows of the chemical ants, formic acid and formaldehyde.

* Vide Bibliography.

B. Black Death Dietary in the Fourteenth Century (Prophylactic).

Now as soon as the rain shall announce itself by thunder or hail everyone of you shall protect himself from the air; as well before as after the rain kindle a large fire of vinewood, green laurel, or other green wood. Wormwood or chamomile shall also be burnt in great quantities in the market places, in other densely inhabited localities, and in the houses.

Until the earth is completely dry, and for three days afterwards, no one ought to go in the fields. During this time the diet should be simple, and people should be cautious in avoiding exposure in the cool of the evening, at night, and in the morning.

Poultry and water fowl, young pork, old beef, and fat meat in general should not be eaten, but on the contrary, meat of a proper age, of a warm and dry, but on no account of a heating and exciting nature. Broth should be taken seasoned with ground pepper, ginger, and cloves, especially by those who are accustomed to live temperately, and are yet choice in their diet. Sleep in the day time is detrimental; it should be taken at night until sunrise, or somewhat longer. At breakfast one should drink little. Supper should be taken an hour before sunset, when more may be drunk than in the morning. Clear light wine mixed with a fifth or sixth part of water should be used as a beverage. Dried or fresh fruit, with wine, is not injurious, but highly so without it. Beetroot and other vegetable, whether eaten pickled or fresh, are hurtful; on the contrary, spicy pot herbs, as sage or rosemary, are wholesome. Cold moist watery food is in general prejudicial. Going out at night is dangerous on account of the dew. Only small river fish should be used. Too much exercise is hurtful. The body should be kept warmer than usual, and thus protected from moisture and cold. Rain water must not be employed in cooking, and everyone should guard against exposure to wet weather. If it rains a little fine treacle should be taken after dinner. Fat people should not sit in the sunshine. Good clear wine should be selected and drunk often, but in small quantities, by day. Olive oil as an article of food is fatal. Equally injurious are fasting and excessive abstemiousness, anxiety of mind, anger, and immoderate drinking. Young people in autumn especially must abstain from all these things if they do not wish to run a risk of dying of dysentery. Bathing is injurious. Men must preserve chastity as they value their lives. Every man should im-

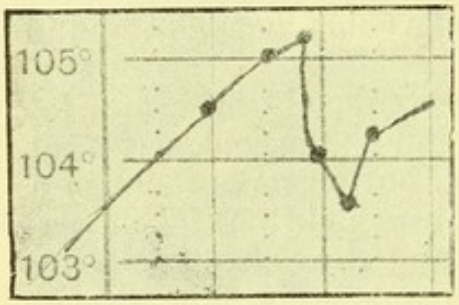
press this upon his recollections, but especially those who reside on the coast, or upon an island into which the noxious wind has penetrated.—" Faculty of Physicians," Paris, 1400.

Tetanus has followed modern prophylactic inoculation (Dupuy).

KENNEDYTOWN HOSPITAL.

FIG. 81.—C.K., age 39; male. Bubonic. Chinese.

16th May, 1896.

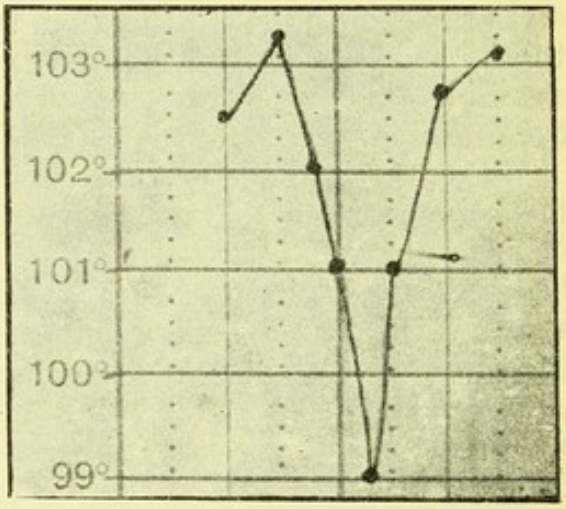


Phenacetin grs. IV.

Died on 17th May, 1896.

FIG. 82.—C.W., age 42; male. Bubonic. Chinese.

2nd May, 1896.

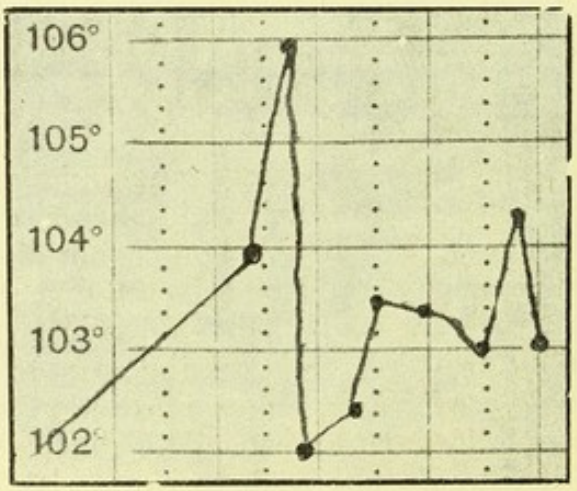


Died 3rd May, 1896.

KENNEDYTOWN HOSPITAL.

FIG. 83.—C.T., age 25; male. Bubonic. Chinese.

17th May, 1896.



Died on 19th May, 1896.

CHAPTER V.

Lecture on the Plague.

Plague was a blow, a stroke, a stripe, anything that irritated or annoyed. It had always been associated with the home, ever since it attacked Pharaoh's household. Rats were described amongst the domestic animals of the earliest nomadic tribes. Plague amongst rodents was the precursor of the disease, so much so that Oriental nations termed it the "rat plague," rat epidemic, or rat disease. In point of fact, it was a rat disease in every sense of the term. They were as ancient as the house fly. Insects and cats were regarded by the ancient Egyptians as sacred. They were defied. In fact, if the cat was to do her duty to-day in killing all the roving rats and mice, a golden image would still be erected to her by various responsible bodies, for her honorable service. When plague-infected animals died, their native vermin, in their transit, disseminated broadcast the germs of the disease, and might affect man. The home of the plague was Egypt. The "Black country," as termed by the natives, not only furnished the "black art," but also the "Black Death." No greater blow could attack a household than the pestilence that walked in the darkness. Death, whether black, white, or whatever colour, was always a sublime subject. Plague was simply a fever accompanied with boils, the "plague of boils." In fact, both the fever and the boils might be absent, and the patients still die of plague. Bubonic plague was the favorable legacy of ancient plague. Many never reached to this favorable type of the disease. Plague was an acute imitation of almost every infectious disease. It was also a mimicry of diseases such as apoplexy, sunstroke, and death by lightning. which were not infectious. It was further a mimicry of death. A person might take his last meal with zest, and die suddenly without any apparent illness. Plague in the lungs was the most infectious variety of plague, and corresponded to the "Black Death" of mediæval ages, when bleedings under the skin and from the lungs were prominent symptoms. It corresponded more with Chinese than with Indian plague, the mortality of the former being almost total, whilst in the latter it was 50 per cent. There were various varieties which simulated measles, cholera, consumption, typhoid, and other fevers. Plague was not a contagious disease, and in some mild cases it was even not an infectious disease, where the germs were locked up and destroyed, never finding an exit. Millions of

plague germs might be cast off from burst boils, and be found in the spit. These were the two most important sources of general infection. There was a limited fatal zone of infection around cases of plague in the lungs. The plague germs might even be destroyed in matter cast off from the boils naturally. The discharges and dejections of patients were not infectious until late in the disease, when they poured themselves into the blood and found an exit. There was a triple channel of infection, as in the inoculation of animals. By the skin, lungs, and digestive tract, it might gain admission into the human system.

We were indebted to the Japanese race for the discovery of the plague bacillus, to the Japanese and Russian for the invention of the plague vaccine, and to the French for the plague serum treatment. These discoveries were amongst the greatest of the century in modern medical science, and compared equally with that of the diphtheria bacillus and its antidotal treatment. The attention of the civilised world had been directed to the patriarchal words of the three illustrious plague discoverers, Kitasato, Yersin, and Haffkine, within recent times.

Natural methods assisted in preventing the dissemination of this fell disease. The plague bacillus was killed by sunlight and by desiccation. Fortunately it had no self-movement like the typhoid bacillus, although it found a host in the rat. It had an enormous potential latitude of adaptability. It ranged in size from the smallest to the largest bacillus. It lived in summer and winter at the ordinary temperature of the air. It recovered after freezing. It lived amongst a long list of various soils, amongst which were the necessaries of life. It had survived, and lived for days in all kinds of waters, including salt water. It had, however, its adversaries to contend with in Nature. The germs of putrefaction invaded the dead body of human beings and animals, and destroyed the plague germs. Plague raged amongst the poor people of ancient Egypt, who could not afford to have their bodies expensively embalmed. The body was salted, which interfered with Nature's method of disposing of the plague germs in embalmed bodies. The plague germs lived well in salted manures. Thus plague was fostered in its ancient home and imported into Europe in mummies for medicinal purposes.

In the manufacture of plague vaccine the plague microbe plant grew visibly to the naked eyes, like ordinary aquatic and marine plants when grown in a broth with a layer of butter

or cocoanut oil. It ranged from the simplest to the great yeast cell of fermentation. When plague germs were spread over salt seaweed jelly, like butter over bread, they developed into yeast fungi. It was not only once or twice that a plague case had been taken up by the police as an inebriate, owing to his staggering gait. Rats became drunk with plague. In fact, plague could be induced artifically in the human being with an injection of yeast under the skin. The plague germ performed the function of a bacillus and a yeast cell. It was a ferment disease. The intense thirst and delirium of plague resembled delirium tremens. Plague was associated with countries that had national fermentable drinks, because the granary was the home of the rat. A combined bacillus and a yeast cell was connected with Russian koumiss, Eurasian kephir, Indian arrack, Chinese samshu, and Japanese saki ; and in the latter it had been traced to a fungus. The plague germ might acquire fungoid properties and live on grain, the staple diet of rats. Inebriates stood plague badly, but total abstinence was no security against the disease. The plague germs were locked up in the plague case as miniature distilleries. If we wished to know assuredly the contents of the bottle of infection, the plague Gordian knot must be untied, and not cut with the sword.

Text of Plate III.

"You cannot have a more complete, a more stainless type of flower —absolute inside and outside, all flower."—*Ruskin.*

"We rise, and rise, and rise—marvels sweet for ever."—*Leigh Hunt.*

After original oil painting from a specimen of the bloom of Health. Ephemeral botanical preservation in good spectroscopic relief (1898).

Bacteriaceæ,
Chromaceæ,
Exanthemata,
Carnaceæ.

Text of Plate III.

"You cannot have a more complete, a more stainless type of flower—absolute inside and outside, al flower."—*Ruskin.*

"We rise, and rise, and rise,—marvels sweet for ever."—*Leigh Hunt.*

Original oil painting from a specimen of the bloom of Health. Ephemeral botanical preservation in good spectroscopic relief (1898.)

Bacteriaceæ,
Chromaceæ,
Exanthemata,
Carnaceæ.

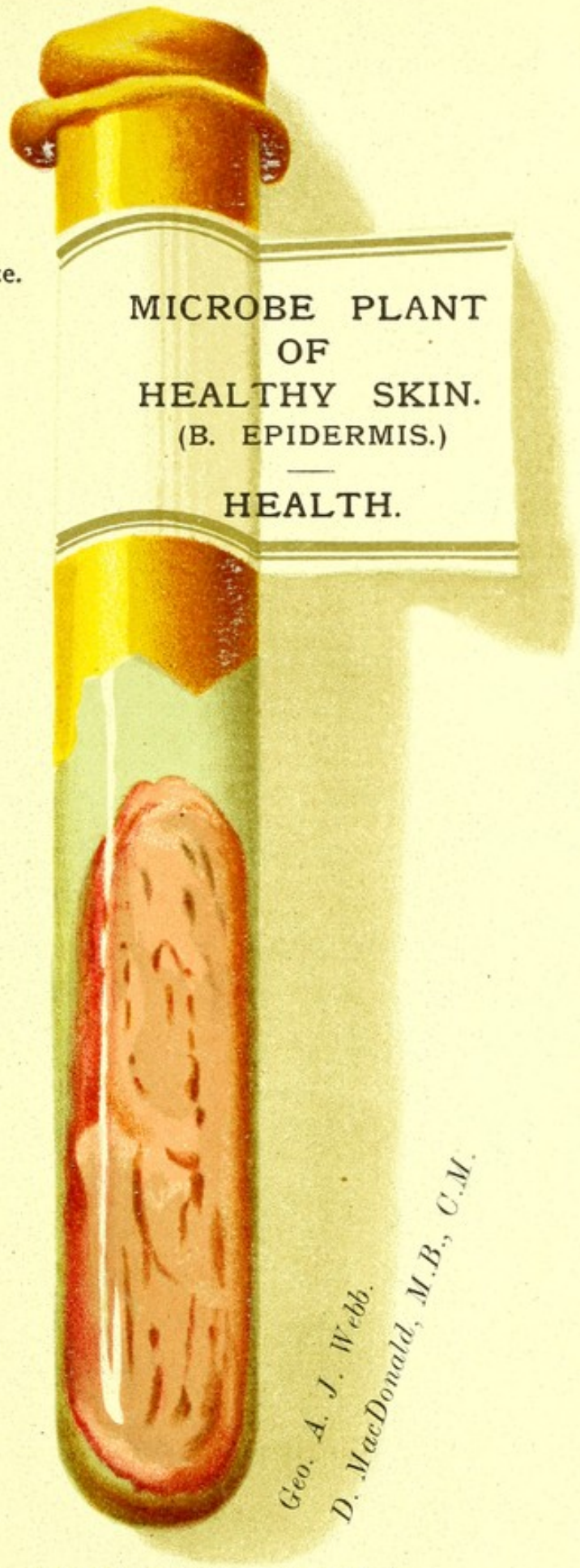
Kaleidoscopic
Chromogenesis.

Bacterioscopy.

Biological
Reagent C.

Papilionaceous
Leguminosæ.

Dermo-phanerophyte.



MICROBE PLANT.

Vardon &

Two Dozen Anti-Plague Golden Rules.

Remember plague is more "Death" than disease.

Never visit suspected or plague-stricken houses.

Never alter a well-regulated diet.

Wash the hands frequently.

Avoid excesses in diet and wines.

Cook food well and preserve from insects.

Heat serving plates to a high temperature.

Cooking utensils wash with boiled or water of undoubted purity.

Rather drink weak tea than suspicious water.

Avoid excess in exercise and bathing.

Never handle dead rats.

Destroy your vermin.

Never neglect a trifling wound, cold, or dyspepsia.

Protect the lower limbs well.

Be vaccinated and revaccinated if you can.

Keep good fires in winter.

Avoid wet feet.

Preserve the head with sunshades in summer.

Use if you can the mosquito net.

Never exchange pipes.

Never kiss the plague suspect.

Avoid plague apparel unless fumigated.

Never fear, rather be cool, calm, and collected.

Remember cleanliness is next to godliness.

CHAPTER VI.

A. Plague Epidemiological Diagnosis.

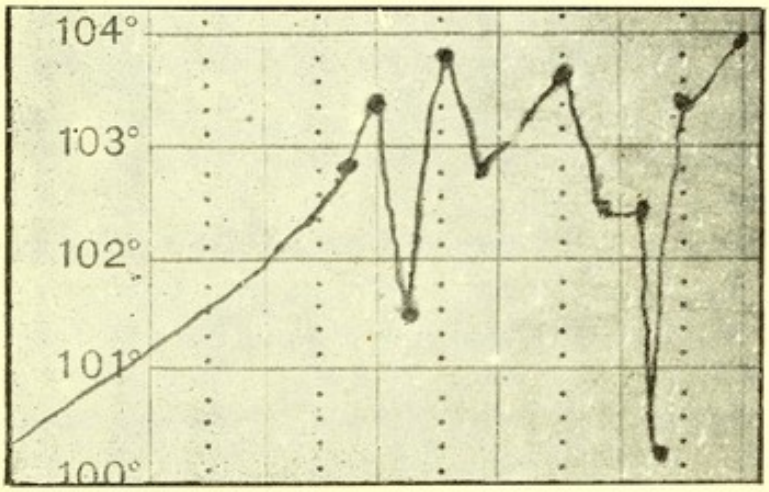
In its subtile evolution the long-looked-for Great Plague of The Centuries has at last unveiled itself in the Austral and Oriental arenas, and still there is little known of the vegetable causation of "Black Death," that form which killed suddenly or in a few hours, that smote, charred, and depopulated, in ancient times by one-third, whole continents of animal and human life, as if by the thunderbolts of Jupiter, and which had no respect for age, sex, race, caste, or climate (*vide* Part I., Chapter II.). In a few months in 1904 it claimed in India more than a million victims (1,040,429).

1. Critical Enquiry of its inception. (Retrospect). Although epidemic glandular enlargements have been known to attack the aborigines of Central Africa and Australia, it was merely conjectural what varieties of plague would first appear. Should it be pestis minor or Oriental plague with a low mortality in its bubonic form, or would it revert to some of the more ancient forms with a high mortality unknown to this generation ? Could it obtain a footing at all and at any time, or would it strike off at a tangent on laws unknown to us, modify and accommodate itself in such a way so as to be afterwards described as Austral Plague or some other so-called variety ? Would it conform to any of the laws of Oriental plague as to climate, season, or moisture, and what would be its relation to enteric fever so indigenous to these colonies ? Would it oust, somewhat replace the latter, and then become annually indigenous ? What should we call plague ? How should we determine its appearance ; by its precursor and contemporary spontaneous destruction of animal life ; by the pest bacillus, or by its high mortality ? Should it arise spontaneously or be imported ? Should it be ancient, modern, true, or spurious plague ? Should it be more or less infectious and none the less contagious than Oriental plague ? Were we to content ourselves with a bacillus of very low mortality for inoculated animals and a high, for human beings, or a high for inoculated animals, and a low for human beings, a high mortality for both, or like the ancients with no bacillus at all ? How was it to be identified ? Such are ponderous questions of the moment. The bacteriological utility, moreover, of stalactites in plague pathogenic horticulture is of immense importance clinically and epidemiologically, being a prominent macroscopic pathognomonic.

KENNEDYTOWN HOSPITAL.

FIG. 84.—L.A.P., age 21; male. Bubonic. Chinese.

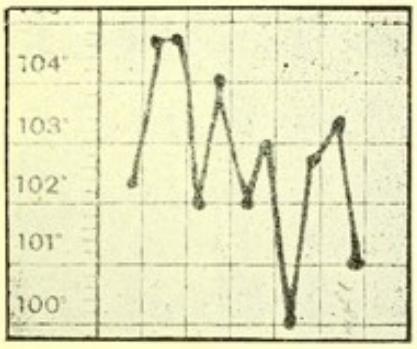
14th May, 1896.



Died on 16th May, 1896, at 4 p.m.

FIG. 85.—N.K., age 38; male. Bubonic. Chinese.

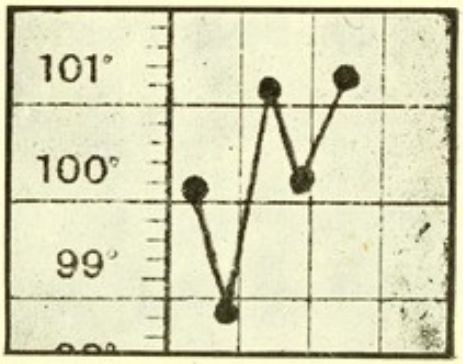
10th May, 1896.



Died 15th May, 1896, midnight.

FIG. 86.—Rabbit (right hip mark) inoculated in the abdomen hypodermically.

25th May, 1896. 26th. 27th.



Died.

2. ITS CLINICAL SUBSTITUTES.—We now have such diseases as pseudo-diphtheria, pseudo-tuberculosis, and pseudo-influenza based on bacteriological nomenclature. With an ancestry of illustrious history the disease designated "Plague" is now standardised and modernised as "Bubonic Plague." This is on account of the acute leucocytosis expending all its energy on the superficial lymphatic system. The ancients, however, classified plague with glandular enlargements on a mortality basis, the bubonic variety being the most favorable. The Kitasato-Yersin bacillus of Oriental

plague, like the Klebs-Loffler of diphtheria, is now standardised as the real cause of modern plague. There is, however, no closer mimicry of clinical plague than a duplicate of enteric and dengue fevers. When tropical influenza or dengue complicates any other infectious fever the product is a hybrid disease, a mimicry of true plague, and merits the appellation pseudo-plague. Dengue and plague are not infrequently contemporarily epidemic, and what is often regarded as pestis minor may yet be found to be a variety of the former. This coalition of dengue, with other infectious diseases, results in an elevated mortality of the principal. The enteric fever pursues a curtailed course, and is generally followed by a tidal relapse. On the other hand, dengue is an extended duplicate of itself in typho-dengue. The duration of the initial attack outstrips the eight days of dengue, but is simultaneously consistent with the curtailed course of enteric.

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FIG. 87 (i.).—C., age 35; male. Bubonic. Chinese.

24th May, 1896.

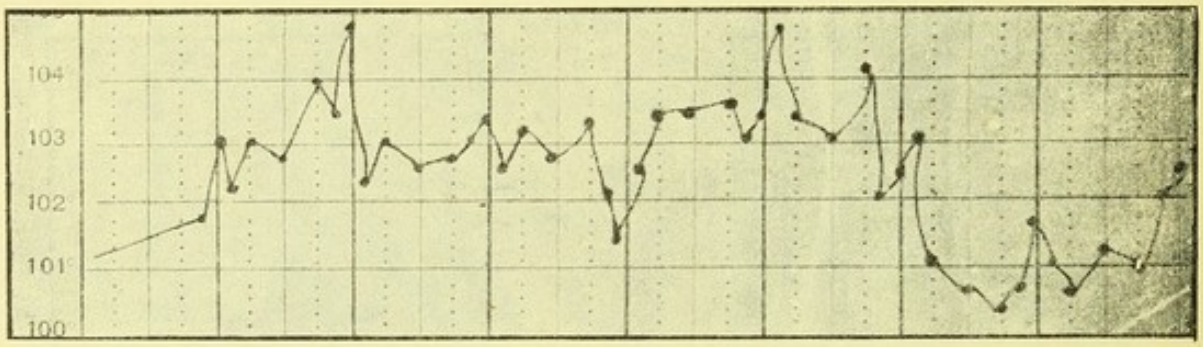


FIG. 87 (ii.)—C. (continued).

1st June.

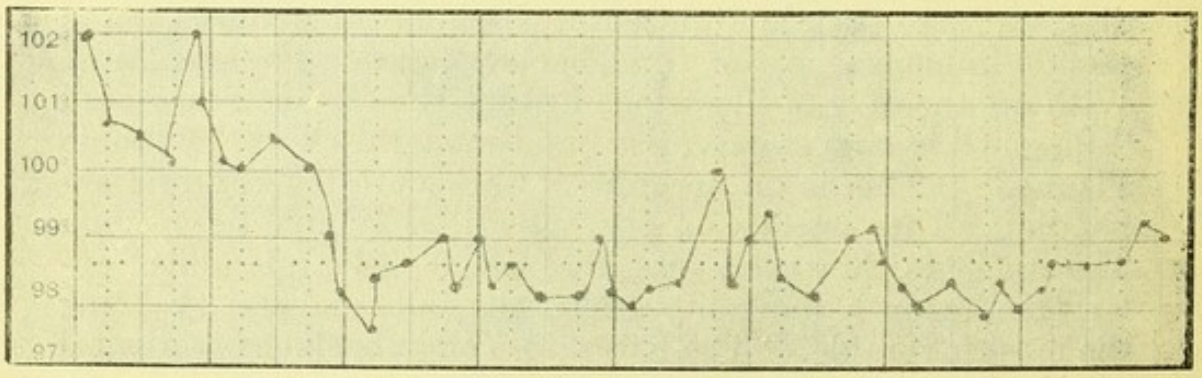
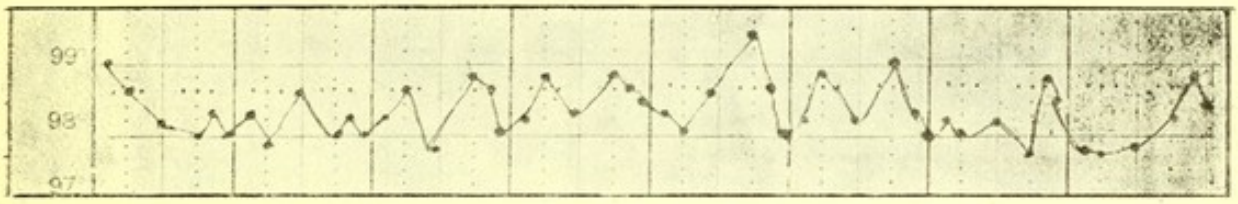


FIG. 87 (iii.).—C. (continued).

9th June, 1896.

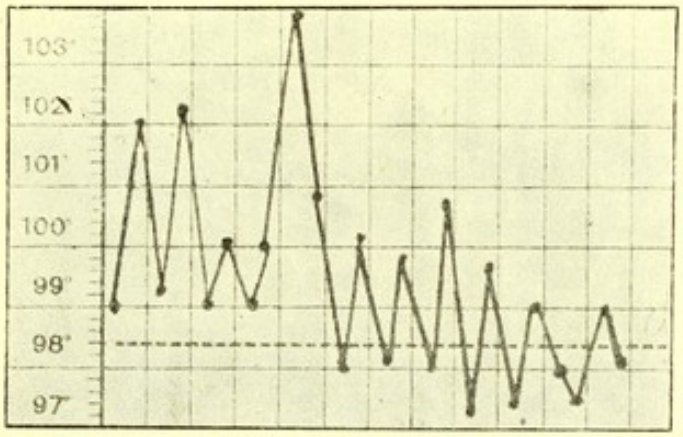


Discharged 4th July, 1896.

KENNEDYTOWN HOSPITAL.

FIG. 88.—I.S., age 13; female. Bubonic. Chinese.

27th April, 1899. 1st May. 8th.



Recovery.

Glandular localization is also well marked in cerebro-spinal meningitis; also a left inguinal bubo, from which two drachms of pus was evacuated, was a prominent symptom throughout a recent case of typhoid under the writer's notice. A tidal relapse is common in such diseases as enteric, plague, and dengue. Rarely does the patient, however, become submerged in this sea of infection except in plague. The only constant pathological property of all varieties of plague is enlargement of the mesenteric glands, and this is the peculiar feature of other diseases. When an acute glandular affection, like dengue, becomes engrafted on enteric fever with a high mortality, we have a natural clinical mimicry of plague. The superficial polyadenitis is then an accidental entity. Suppuration of the glands is not a common feature unless in the upper segment of the body. There is a historical pleomorphism of plague spots. No eruption of a specific character has, however, been described in modern times.

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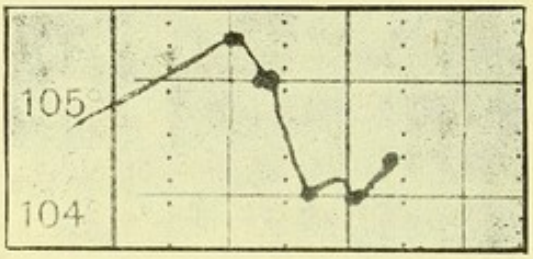
Fig. 89.—L.K., age 22; female. Bubonic. Chinese.

22nd May, 1896.



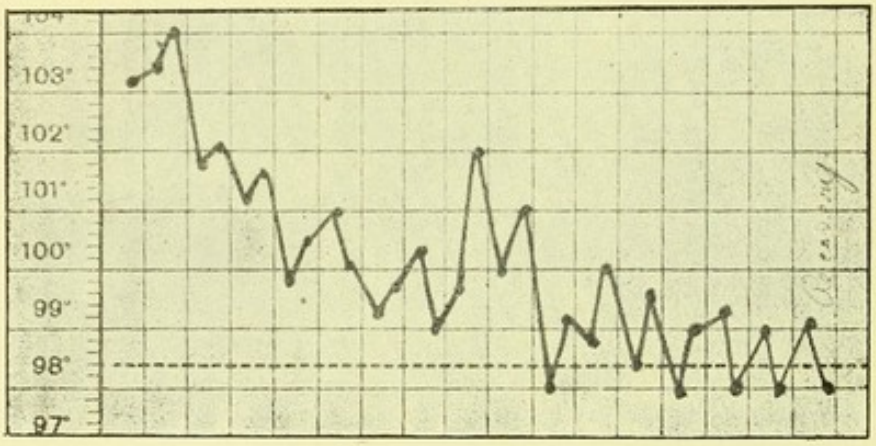
Calomel.

Died on 23rd May, 1896.

KENNEDYTOWN HOSPITAL.

Fig. 90.—T.K., age 43; male. Bubonic. Chinese.

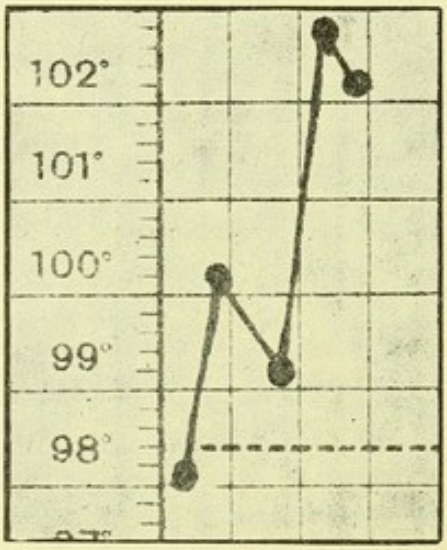
8th April, 1896. 24th.



Recovery.

Fig. 91.—Rabbit (face mark) inoculated by mouth; feeding.

25th May, 1896. 26th. 27th.



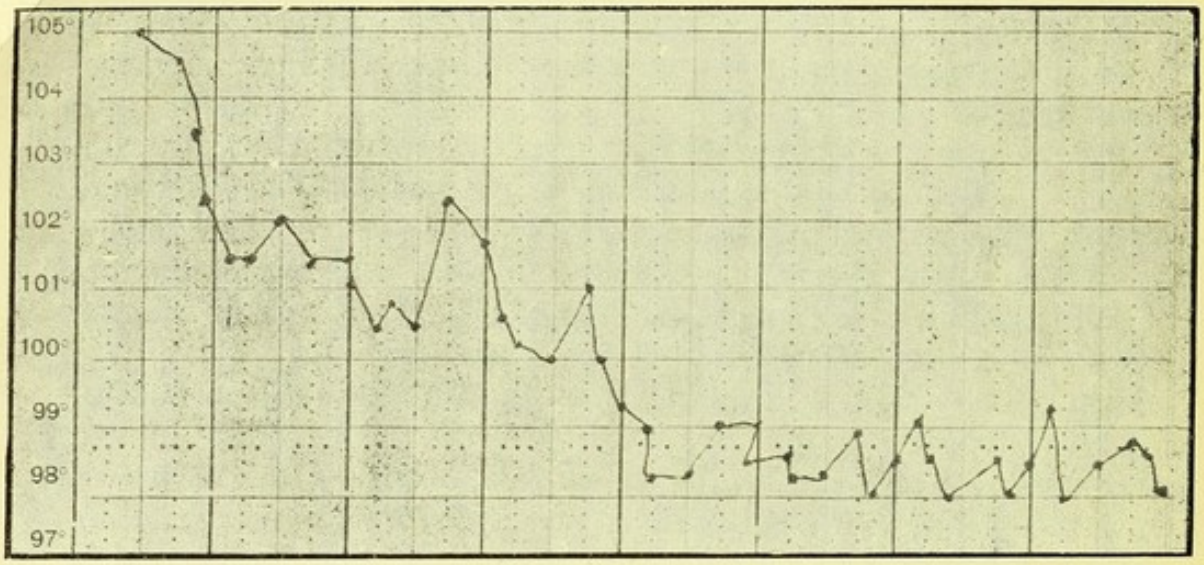
Died.

3. Its Identity.—Pleomorphism is not mere degeneration. Beginning with the standard pest bacillus of Oriental plague, whose pleomorphism ranges wider than that of any other known species of plague, whose botanical morphology varies from the smallest influenzal bacillus to the schizomycetes, algal and yeast cellular bacteria of aquatic habits, and whose biology, parasitism, and saprophytism is inversely proportional to the nutritive media of its inert cultural or vital host, plague has a very wide range of adaptation for environment. This is well illustrated in its serial transit through vital soil—its pathogenesis further increases or decreases—and also in its aberrant staining, cultural, and morphological potentialities. The pest bacillus of Oriental bubonic plague, however, is not a true bacillus but a parasitic and saprophytic form of a variety of mould with aquatic and marine habits. *A true bacillus of plague has not yet been discovered* as a standard for comparison, and to speak of any other as a bacillus of pseudo-plague is not scientific. Long before the pest bacillus was mentioned and rodents were suspected, any disease which suddenly attacked a number of people with glandular enlargements and accompanied with a high mortality, was believed to be plague. However, plague is the only disease which kills in a few hours with an acute glandular affection, and can furnish a yeast or algal bacillus in sparsely nutritive media. The plague bacillus may be either one of hæmorrhagic or leucocyctic septicæmia. Plague, then, most naturally accommodates itself to new environments. To set at rest influenza and enteric fever on a sure foundation paves the way for its identity. With epidemic dengue at the one pole, buboes, no bacillus, and a mortality of almost nil, and plague at the other—glandular enlargements, a pest bacillus, and a mortality of quantum sufficit represents the total area of plague potential elasticity. Around these cardinal pivots centres whether plague has appeared or not. All the acute glandular diseases that might be mistaken for plague find their prototype in dengue fever. Furthermore, *the true plague bacillus* must forthwith embody the ancient idea of the disease and comprise the *B sirius*, *B. luna*, and *B. stellæ* amongst its casual relations.

KENNEDYTOWN HOSPITAL.

FIG. 92.—T.O., age 22; male. Bubonic. Chinese.

31st March, 1896. 3rd April. 7th.



B.

IN MEMORIAM.

*PLAGUE PATHOGENIC HORTICULTURE.

"Did not grudge his life whenever duty called for it."—Wakabayashi.

Japan: Baba, Tei Ichi, Dr., Osaka; Wakabayashi, Dr., Osaka; Yamanaka, Kame, Dr., Osaka.

India: Evans, Prof.

Austria: Müller, Dr., Vienna.

Australia: Wray, Dr., Brisbane, Queensland.

Portugal: Calmette, H. Prof., Oporto.

Germany: Sachs, Dr., Berlin, 1903; Knorr, Dr., Munich.

Formosa: Matsumoto, Dr., Japanese, colonial; Kondo, Dr., Japanese, colonial.

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* Vide Plate III.

KENNEDYTOWN HOSPITAL.

FIG. 93.—Rabbit inoculated in the abdomen (hypodermically)
19th May, 1896. 25th.

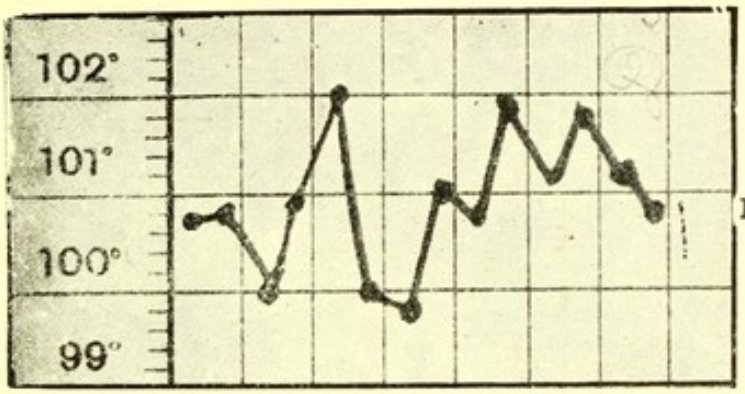


FIG. 94.—Guinea-pig inoculated in the hip (subcutaneous).
17th April, 1896. 24th.

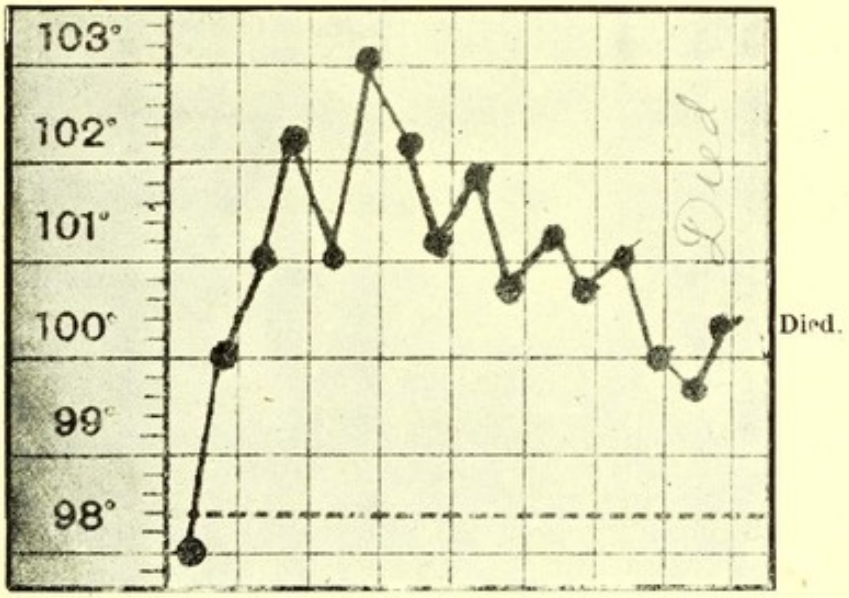
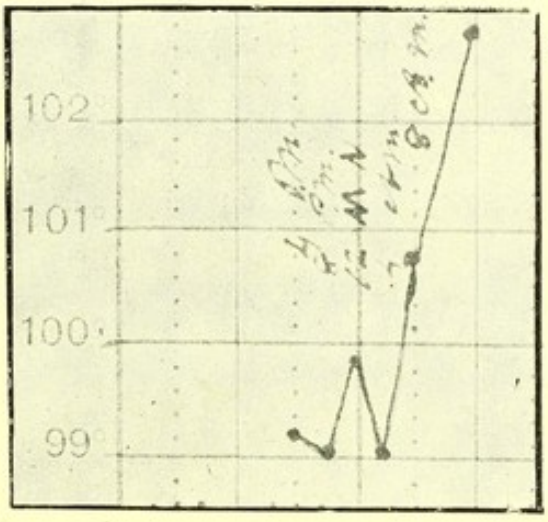


FIG. 95.—M.F., age 42; male. Bubonic. Chinese.
2nd April, 1896.

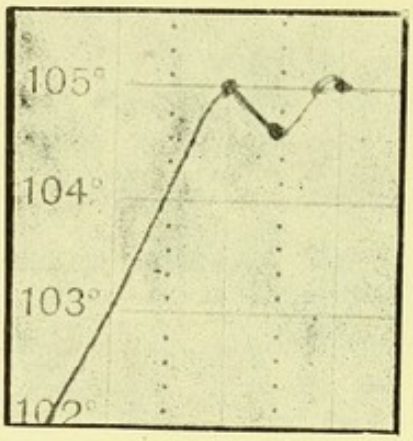


Died 3rd April, 1896.

KENNEDYTOWN HOSPITAL.

FIG. 96.—C.K., age 30; male. Bubonic. Chinese.

18th May, 1896.



Died 19th May, 1896.

FIG. 97 (i.).—C.P., age 36; male. Bubonic. Chinese.

28th May, 1896. 1st June.

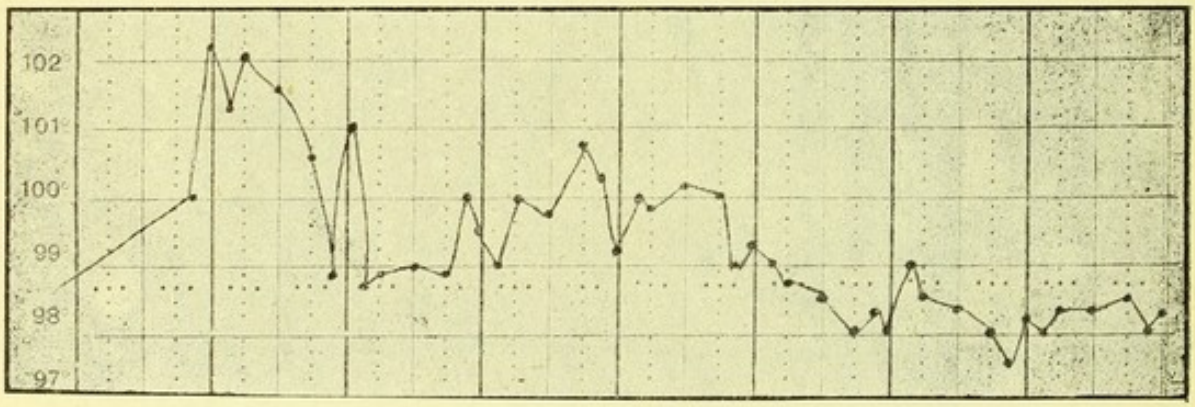


FIG. 97 (ii.).—C.P. (continued).

5th June.

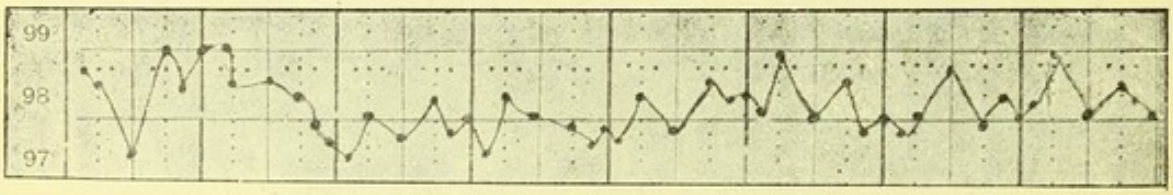
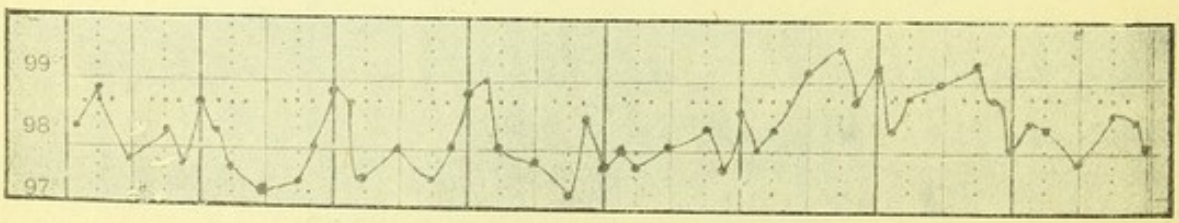


FIG. 97 (iii.).—C.P. (continued).

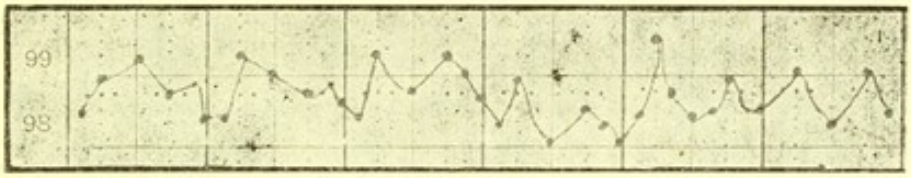
13th June.



KENNEDYTOWN HOSPITAL.

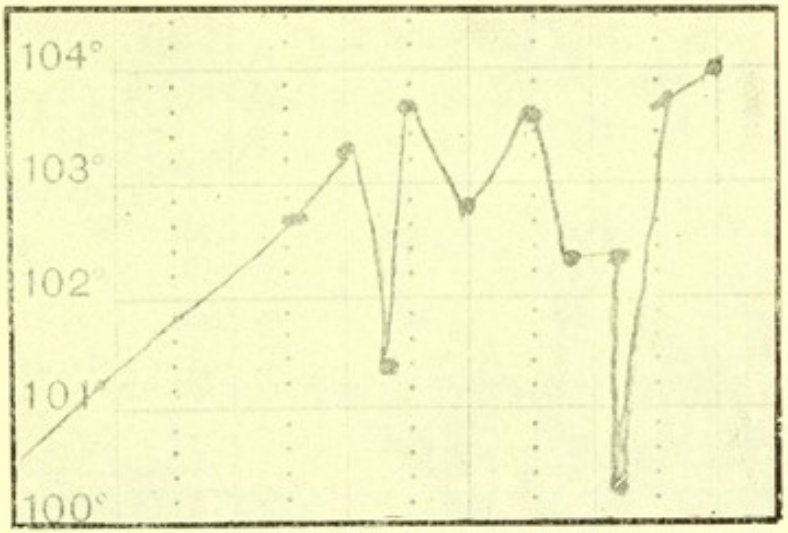
FIG. 97 (iv.).—C.P. (continued).

21st June, 1896.



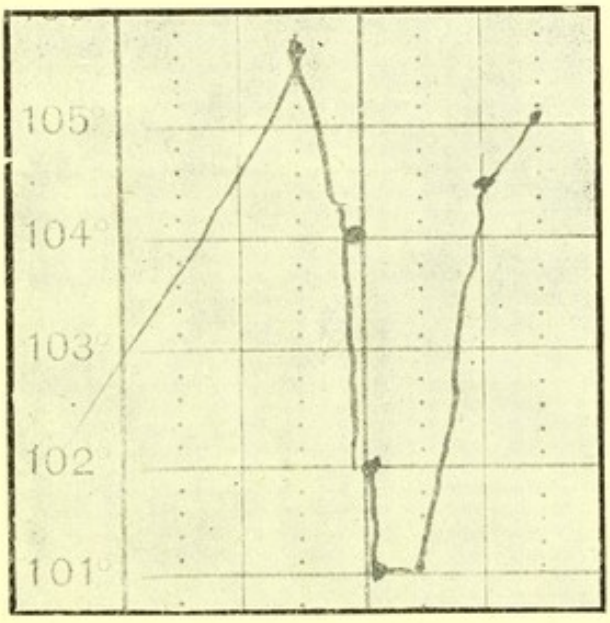
Discharged 4th July, 1896.

FIG. 98.—L.A.P., age 21; male. Bubonic. Chinese.
14th May, 1896.



Died on 16th May, 1896.

FIG. 99.—F.H.Y., age 17; male. Bubonic. Chinese.
16th May, 1896.



Died on 17th May, 1896

KENNEDYTOWN HOSPITAL.

FIG. 100.(i.).—T.K , age 43; male. Bubonic. Chinese.
16th April, 1896.

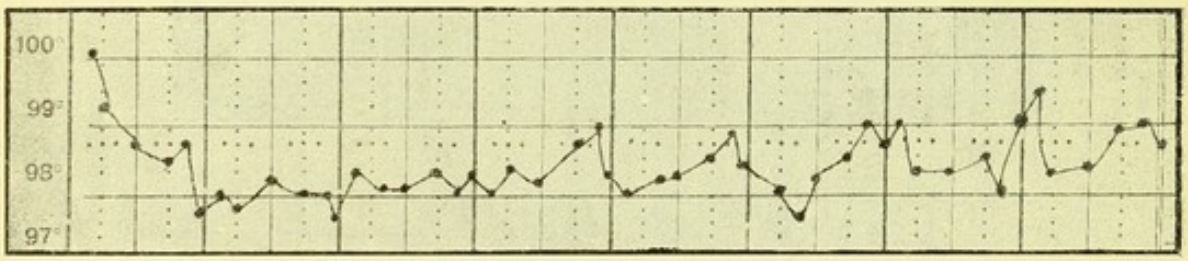
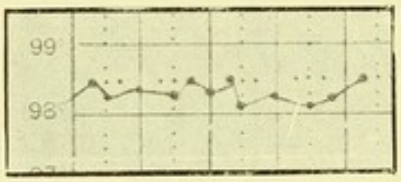
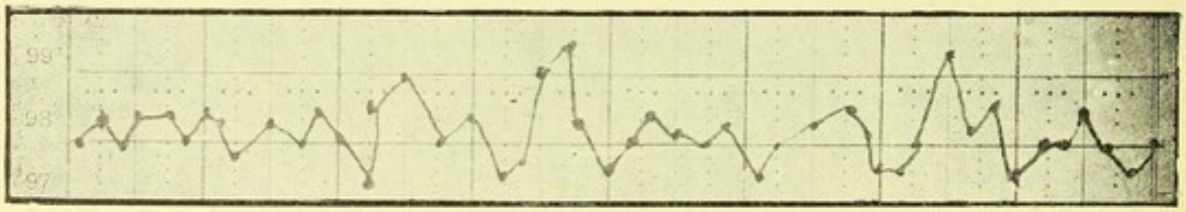


FIG. 100 (ii.).—T.K. (continued).
24th April.



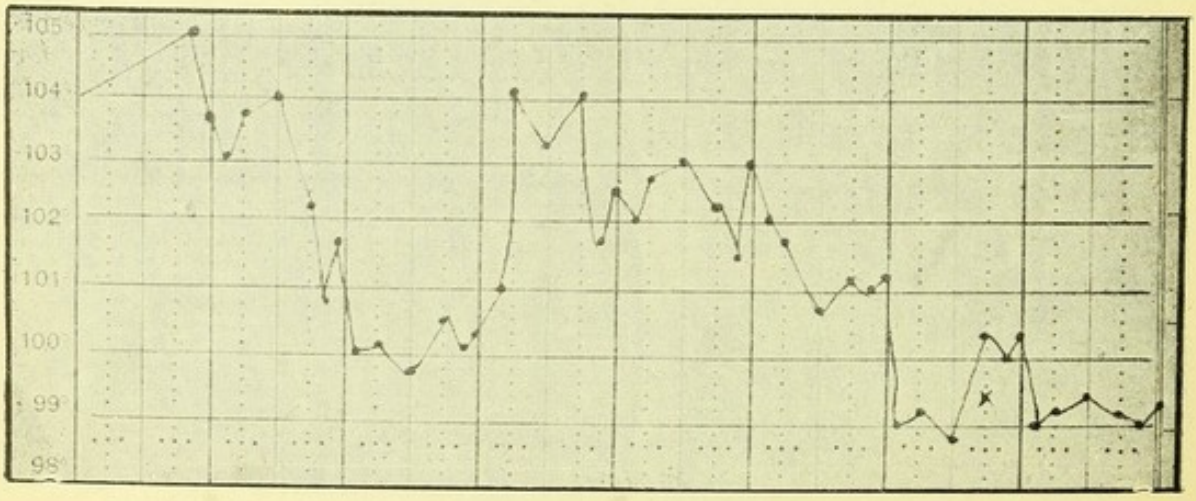
Discharged 4th July, 1896.

FIG. 101.—N.S., age 36; male. La Peste Enterique. Chinese.
9th May, 1896. 16th.



Discharged on 24th June, 1896.

FIG. 102 (i.).—L.S.K., age 17; male. Bubonic. Chinese.
28th May, 1896.



KENNEDYTOWN HOSPITAL.

FIG. 102 (ii.).—L.S.K. (continued).

5th June, 1896.

FIG. 102 (iii.).—L.S.K. (continued).

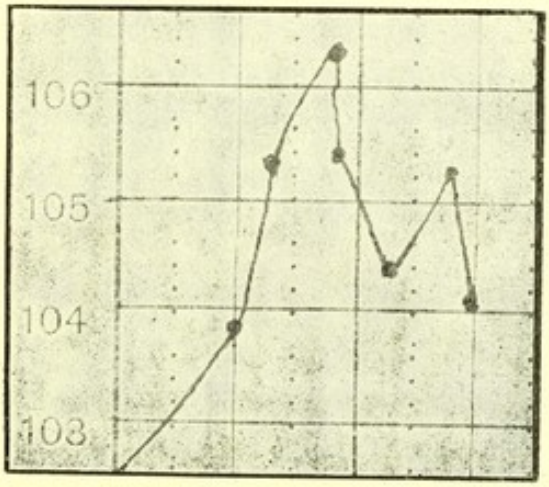
13th June. 20th.

Discharged 4th July, 1896.

KENNEDYTOWN HOSPITAL.

FIG. 103.—C.S., age 16; female. Bubonic. Chinese.

24th May, 1896.

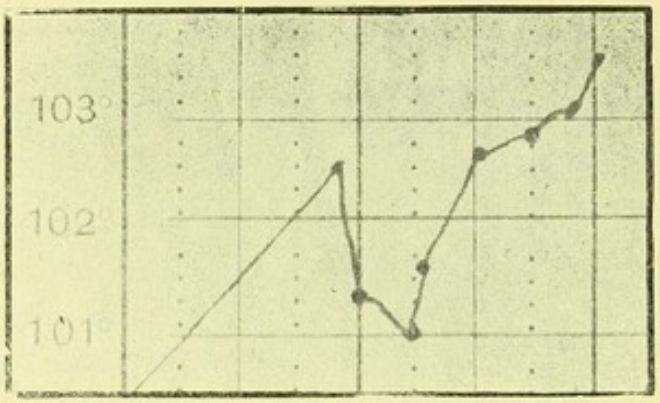


Died on 24th May, 1896.

KENNEDYTOWN HOSPITAL.

FIG. 104.—W.I.C., age 22; male. Bubonic. Chinese.

24th May, 1896.



Calomel. Died 21st May, 1896.

FIG. 105 (i.).—C.T.S., age 23; male. Bubonic. Chinese.

5th May, 1896.

FIG. 105 (ii.).—C.T.S. (continued).

13th May.

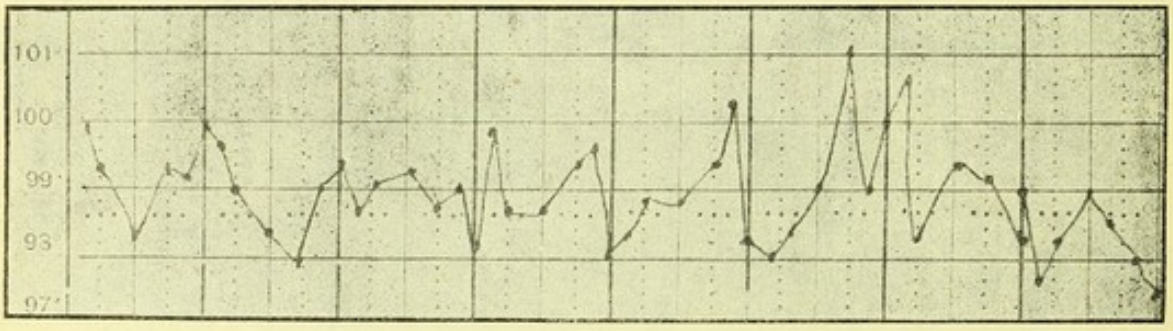
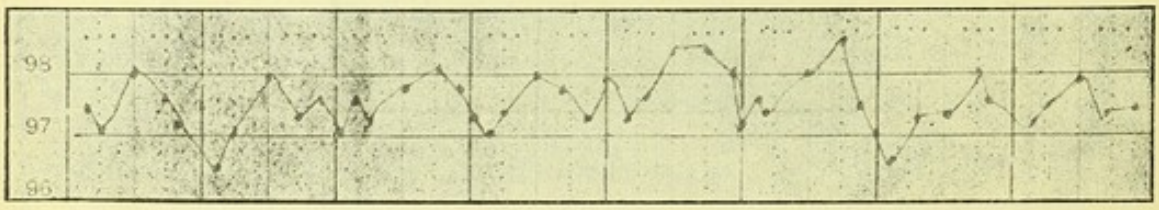


FIG. 105 (iii.).—C.T.S. (continued).

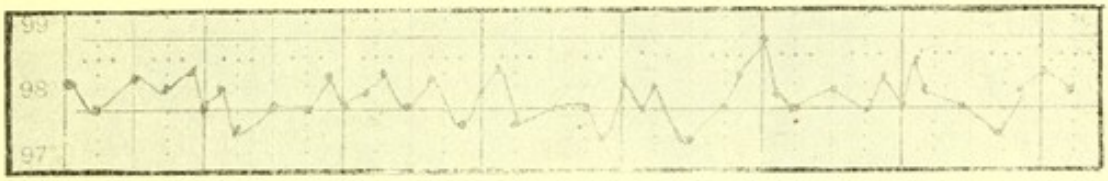
21st May.



KENNEDYTOWN HOSPITAL.

FIG. 105 (iv.).—C.T.S. (continued.)

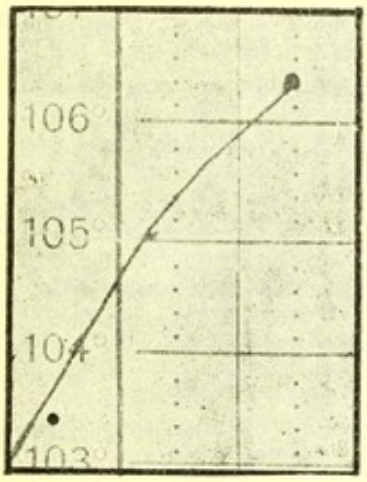
29th May, 1896. 1st June. 5th.



Discharged on 24th June, 1896.

FIG. 106.—K.S., age 26; male. Bubonic. Chinese.

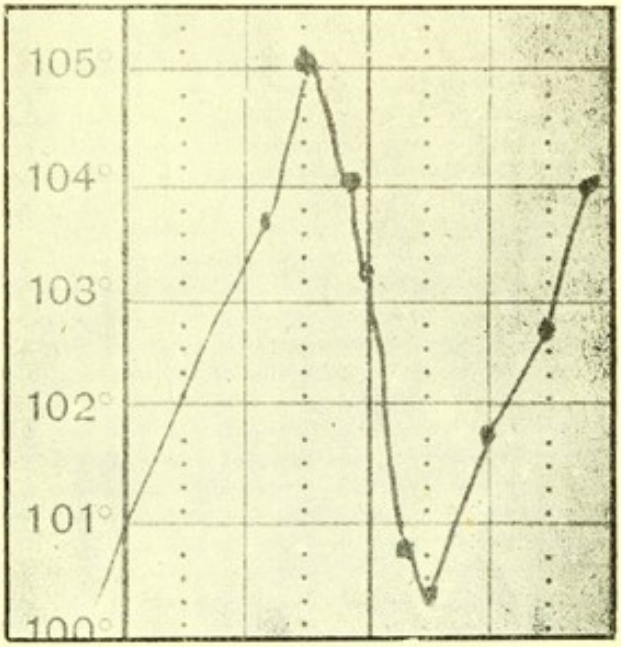
17th May, 1896.



Died 17th May, 1896.

FIG. 107.—L.Y., age 19; male. Bubonic. Chinese.

23rd May, 1896.

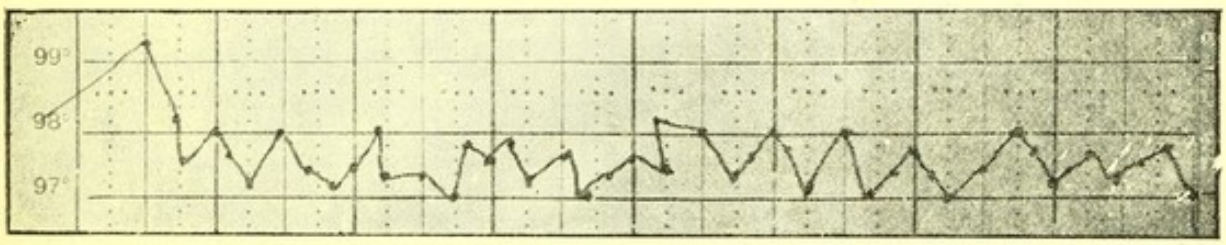


Calomel.

Died on 24th May, 1896.

FIG. 108.—N.S., age 36; male. Bubonic. Chinese.

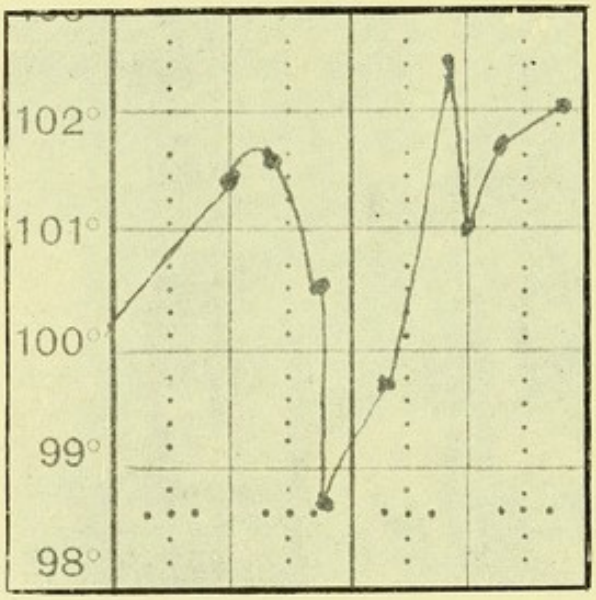
1st May, 1896.



Calomel.

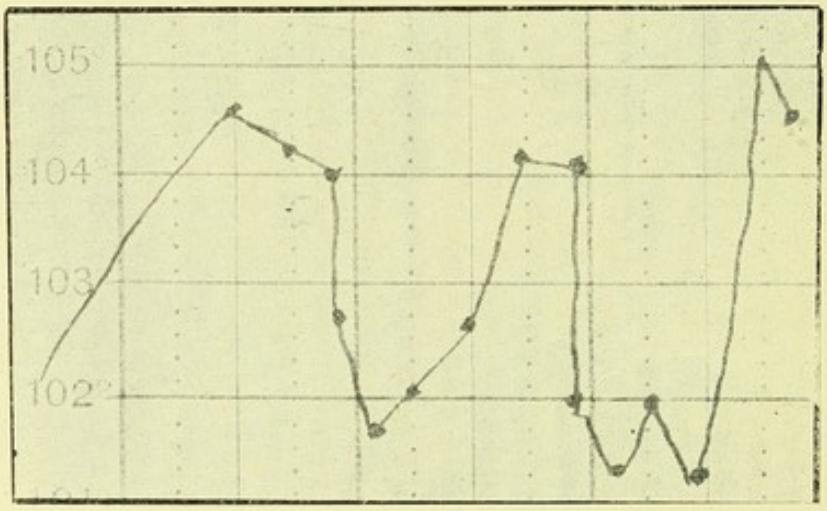
KENNEDYTOWN HOSPITAL.

FIG. 109.—A.T., age 30; female. Bubonic. Chinese.
22nd May, 1896.



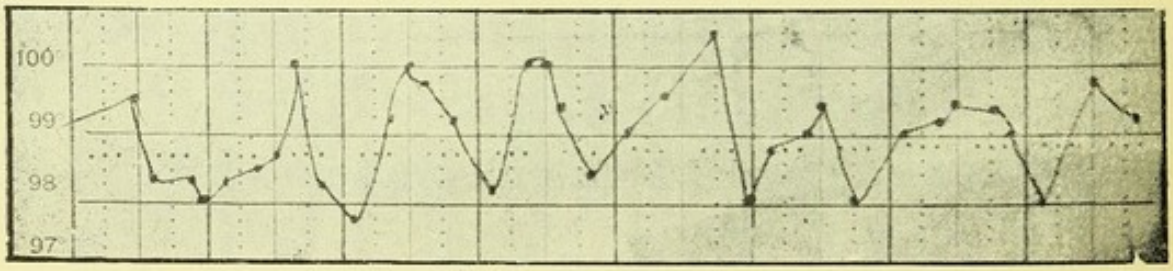
Died 23rd May, 1896.

FIG. 110.—L.S., age 24½; male. Bubonic. Chinese.
16th May, 1896.



Died 18th May, 1896.

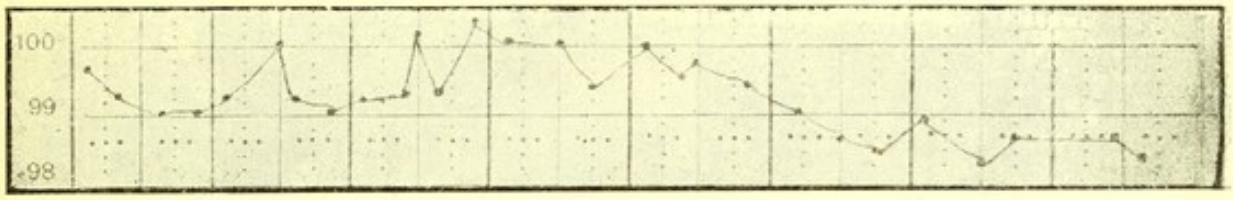
FIG. 111 (i.).—Y.U.U., age 26; male. Bubonic. Chinese.
26th June, 1896.



KENNEDYTOWN HOSPITAL.

FIG. 111 (ii.).—Y.U.U. (continued).

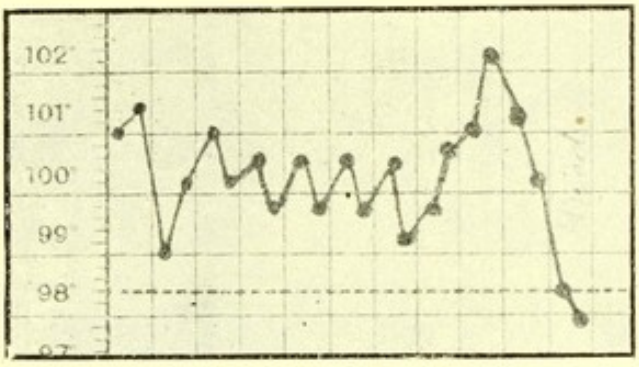
4th July, 1896. 11th.



Discharged 19th July, 1896.

FIG. 112.—Guinea-pig inoculated by mouth, feeding.

6th May, 1896. 16th.



Died.

C.

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End of Part II.

INDEX.

ABBREVIATIONS.

H—Horticulture.
M—Micrococcus.
P—Pyogenes.
R—Room.
T—Tropical, Temperature.
TC—Chromophile Tuberculin.