

The spread of Plague

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A Lecture
ON
THE SPREAD OF

delivered before
Hampstead?

March 22nd

by
James Barr



ACCESSION NUMBER

89186

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Gentlemen: With
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My remarks will
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the latter half of this century, and more particularly to a
description of the information gathered during the outbreak
in China in 1894-6, and of the most recent evidence of its
activity in the Bombay Presidency. The reasons for select-
ing the Hong-Kong epidemic as the basis of my dissertation
are that plague was first studied there bacteriologically, and
that many medical men, myself amongst the number, had
ample opportunity of observing and recording the disease.

Nomenclature

During the latter half of the century the names bestowed
on plague have been as follows:—1. In 1856, in Tripoli, it

was known as "typhus with glandular swelling." When
the disease reached the island of Chios in the same year it
was called "petechial typhus analogous to that of Tripoli."
2. In Mesopotamia, where the disease was prevalent from
1856-85, and possibly up to the present day, the titles,
"adynamic typhoid fever" and "intermittent fever with
glandular swellings" were usual. 3. In Persia, where
plague seems to be endemic since 1863, the term "hemor-
rhagic fever" is used to designate a pestilence which differs
from the diseases with which the Persians are familiar.
4. During the years 1877-79 the Russian province of
Astrakhan was visited by a disease which was, and is,
regarded as plague, but which was variously described as:
(a) intermittent fever with buboes; (b) croupous pneumonia
with buboes; (c) typhus with glandular swellings proving
fatal by pneumonia; and (d) a peculiar form of mumps. That
all the above-mentioned outbreaks were actually plague, a
specific disease caused by a specific bacillus, will never be
known; but the presumptive and clinical evidence is
decidedly in favour of their being one and the same disease.
5. In Hong-Kong, where a moiety of languages obtain, the
epidemic was known as:—(a) the "plague" or "the bubonic
plague" by the British; (b) "la peste" by the Spaniards;
(c) "de peste bubonica" by the Portuguese; (d) "la peste"
by the French; (e) "la pestilenza" by the Italians;
(f) "die peste" by the Germans; (g) "wan-yik" by the
Cantonese, the term signifying an epidemic disease of high
mortality; (h) "yang-tsu-chwang" in Yunnan, which, being
interpreted, means bubonic disease; and (i) "li-tsu-cheng"
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years. The name "black death" was never used, nor were any of the names by which it was known in Tripoli, Mesopotamia, or Persia, or Russia applied to it, although hæmorrhages, pneumonia, petechia, and typhoid signs and symptoms were not wanting. Perhaps the most interesting name is that of the "rat plague," by which it is known in various parts of Asia, thereby affording additional proof, if such were needed, of the widespread infection of the rat during plague epidemics.

The name "plague," however, is not one which helps us to place the complaint in its proper scientific niche in the category of disease. The word conveys the idea of an unusual disease, attacking many persons and attended by a high mortality, but that gives it no scientific value any more than the "wan-yik" of the Chinese. Were I to

Classification

attempt to classify plague I would place it amongst the specific fevers and particularise it under the name of "polyadenitis" and individualise it still further as "malignant polyadenitis." It may be objected that adenitis is not always present, that many plague-stricken patients die before the glands are even swollen, and that therefore the name is defective; but it is needless to point out that in many diseases the most typical of symptoms are at times absent and yet the cases can be classed with their kind. To justify the prefix "poly" to the word "adenitis" it is necessary to remember that although one gland alone may be clinically apparent, most, if not all, the lymphatic glands are found to be enlarged at the post-mortem examinations. The most recent report in regard to this subject is interesting. "Thirty-three cases (22 per cent.) were received without a proper bubo. In most of these cases multiple adenitis was found, the general size of the glands being that of a lentil. In the latter stages of the disease other buboes formed in different localities and complicated the case; 90 per cent. of the cases showed swelling of the intestinal, bronchial, and mediastinal glands in different stages."

The term "bubonic" so frequently applied to the disease is not so appropriate as would at first sight appear. A bubo is essentially the swelling of a gland caused by the entrance of septic materials in the tract of lymphatics over which the gland presides. This cannot be said to obtain in plague, for the so-called bubo of plague can in no sense be regarded as a sympathetic swelling, but merely the outcome of a blood poison. If it is to be classed a bubo at all it must be distinctly understood to be an "idiopathic" bubo, a true "bubon d'emblée." One never speaks of the buboes of tertiary syphilis or of scrofulous buboes. The terms may be clinically correct, but they are not in common use; they have, however, as much right to be called bubonic as the glandular enlargements in plague. With these considerations before us I would venture to name the disease "Malignant Polyadenitis" in the hopes that attention may be drawn to the naming of the disease by more competent students than myself.

Plague or malignant polyadenitis we may define as an acute febrile disease of an intensely fatal nature, characterised by inflammation of the lymphatic glands, marked cerebral and vascular disturbances, and by the presence of a

¹ Report of Dr. M. Wilm, dated Hong-Kong, May 20th, 1896.

Definition

Distribution

specific bacillus. It is only since 1894 that we have been able to set forth a definition with precision; it is only since then that we have acquired the knowledge of a definite bacillus by which the disease can be recognised scientifically when ordinary symptoms are in abeyance. In this definition no mention is made of hæmorrhage, pneumonia, petechia, or carbuncles.

At the present day plague is confined to Asia, but since 1850 it has made its appearance in Europe, Asia, and Africa. The western limit of plague during the latter half of the century is the Canary Islands, where it raged in 1852; while its eastern limit is the Island of Formosa, off the coast of China, where it is now prevalent. The southern limit is practically the Tropic of Cancer, but the northern range is indefinite. Since 1850 plague has never travelled further north than Astrakan, about 45° N.; but we have it on excellent and sustained authority that Moscow, Norway, and Sweden, and latitudes as far as 60° N., have within the present century been visited by plague. All limits are, therefore, fairly definite with the exception of the northerly limit. In other words, plague has been met with from 19° W. longitude to 121° E. longitude and between 40° N. latitude to 19° S. latitude. Plague has never been known in the Western Hemisphere or anywhere south of 19° N. of the Equator.² The Basin of the Mediterranean and the strip of country which runs parallel to that sea across the Asian continent from Turkey to China may be roughly taken as its present belt during the nineteenth century, but the Mediterranean part of the belt has disappeared almost wholly within the present generation. A map of the plague-stricken districts within the last fifty years shows plague astride the Himalayas with a giant limb reaching on one side the Red Sea and on the other the shores of the Pacific.

What determines the distribution it is impossible to say. Racial distinctions do not account for it, as the Mongoloid and Indo-European races are equally attacked. Neither ocean nor air currents have aught to do with its transit. It seems to thrive in the densely packed population of Canton and in the sparsely populated semi-desert regions of Rajputana and Arabia; the elevated plateau of Thibet and the low-lying valleys of the Euphrates are equally affected.

It occurs during the summer's heat and the winter's snows. In fact, no known meteorological condition has any effect upon either the genesis or the exodus of plague.

As animals are affected, it is interesting to inquire what animals are affected, what is their geographical distribution, and what share, if any, they take in its spread. The animal which above all others is known to be affected by plague is the rat; but the wide geographical area throughout which this animal is met with would seem to frustrate any attempt to determine a collateral distribution. On looking a little more closely, however, we find rats (the Murine) divided into two great sections—the mures, inhabiting the Old World (except Madagascar), and the sigmodontes, inhabiting the New World and Madagascar. Of the mures, one sub-family, that of the Nesokia, is met with, reaching from Palestine to Formosa across the northern part of India. The southern limit in India seems to be where the great bandicoot, or pig rat, exists, and here plague is unknown. This is the only animal which presents a habitation well-nigh corresponding to the present distribution of plague, and it is the animal above all others which is looked upon as being liable to be attacked by plague. The geographical distribution of this family of rats well-nigh coincides with the plague belt as delineated.

Continuity.

With regard to the continuity of plague outbreaks it would appear that in the plague belt the disease is ever active, now in one place and now in another. At times we find it in Arabia, then in Persia and the Himalayas, and the wave travels eastwards over Yunnan and Southern China to the sea. The disease oscillates, now east and now west, but with a fatal precision, making the slow swing of the pendulum of plague within a fairly definite area. A glance at the table giving the dates at which plague appeared in epidemic form since 1850 will confirm this. Taking the chief seats passed *en route* between the Red Sea and the Pacific we find that plague appeared as follows:—In 1850,

India; 1850, China; 1853, Arabia; 1853, India; 1856, Tripoli; 1858, Mesopotamia; 1859, Tripoli; 1863, Persia; 1867, Mesopotamia; 1869, India; 1871, Yunnan; 1872, Persia; 1873, Mesopotamia; 1874, Arabia; 1876, India; 1876, Mesopotamia; 1877, Tripoli; 1877, Russia (Caspian Sea); 1879, Arabia; 1880, Mesopotamia; 1881, Persia; 1881, China (Pakhoi); 1883, Afghanistan; 1884, Mesopotamia; 1886, Persia; 1887-89, Arabia; 1890, China (Yunnan); 1893, Tripoli; 1894, China (Canton, Hong-Kong); 1895, China (Macao); 1896, China (Hong-Kong); 1896, India (Bombay); and 1896, Merv (?). A study of this table shows the continuity of the course of plague and how the eruption in one district is followed by its appearance in another. A slow traveller, it, however, stays when it comes, and unless the scenes of its visitation are well garnished it becomes endemic.

Incubation. The conclusions arrived at by all observers during the Hong Kong Epidemic are that Plague has an incubation period of from 3 to 6 days. The apparent exceptions to this rule are so insignificant that they may be totally neglected. The period of incubation is an important point to settle as upon it depends the ^{practical} ~~classical~~ details to be observed in Quarantine ~~measures~~ ^{generally} and in medical inspection in particular. In the British Medical Journal of Dec 12th 1856 ~~was~~ a notification

hearing upon this very point, was,
in still, of more than passing interest

The

following is a statement of the facts: "About the middle of September a man, a Portuguese-Indian steward, was taken into the Branch Seamen's Hospital and died there very suddenly. He was only in about forty-eight hours and no suspicion of plague arose. On the last day in October there was reason to consider that another patient at that time in the hospital was suffering from plague; the previous case was thought of, and it was agreed that if one was plague the other in all probability was of the same nature. The man died; but the medical authorities were not in entire agreement. As a precautionary measure, however, the body was buried in a leaden coffin. The matter was referred for bacteriological examination, the glands of the body were examined, and a bacillus which presented the known characters of the plague bacillus was found." In connexion with this subject there are one or two points calling for comment. 1. The dates given (if correct) would show the vessel, in which the infected persons were, left Bombay about the end of August. Now, we have no direct medical evidence that plague existed in Bombay during August. The earliest official intimation of plague in Bombay was when Dr. Vignas reported, at the meeting of the Sanitary Committee on Sept. 23rd, 1896, that the disease had prevailed in the locality for a fortnight, thereby assigning Sept. 9th as the date of commencement. The first case, therefore, at the Greenwich Hospital could apparently not have been infected from that source, as the vessel could not have been less than seventeen days on the way. Either it is not plague we are dealing with at Greenwich (I firmly believe it is) or the infection must have come from some other source than Bombay; or yet a third condition may obtain, which heretofore we recked not of—namely, an

indefinite period of incubation, during which period "benign" plague may become malignant. This subject will be discussed later.

In elucidation of these cases and the source of their infection I think I can throw some light. I stated above that there was no official evidence of the presence of plague in Bombay before Sept. 9th, 1896—that is, ten days after the first Greenwich Hospital patient sailed from Bombay. This, however, may not be quite correct, for I find in the *Times of India*, Sept. 30th, 1896, the following statement:—

"In Bombay for two months certainly, in all probability longer, the native inhabitants of the neighbourhood of Maudvie, when plague broke out in Bombay, seemed to have been alive to the fact of the existence of a malady resembling plague before the malignant type developed. They had organised processions in August to propitiate the goddess of Plague. This either proves that true plague was present long before the authorities knew of its existence or that a disease resembling plague was mistaken for the malignant type." According to this evidence plague may have existed in Bombay in July, 1896. This evidence will, I hope, help those who are dealing with the Greenwich Hospital cases to disentangle the incubation period difficulty. The second case reported from the Greenwich Hospital had been on shore fourteen days before falling sick. Therefore, allowing seventeen days for the voyage, thirty-one days must have intervened between exposure in Bombay and the illness.

primarily affected is explained, and as mentioned above, the Chinese consider the scale of infection so exact that they affirm, that animals are attacked seriatim according to the height of their nostrils above the ground. The argument is ^{feeble} ~~quite~~ indeed, as all animals lie down and their nostrils whilst they are asleep ~~and many of them whilst grassing &c.~~ are in contact with the soil.

Dr. Rennie found sand and indigestible materials in the stomach of rats dead of plague in Canton and it may be the consumption of the earthy matters that infected the rat.. But if soil bred it ought to be conveyed by the consumption of herbs and grain, and herbi-vor^{ous} ~~ous~~ animals ought to suffer, unless they enjoy an immunity.

So far as this evidence goes it is possible the poison is soil bred, but it is not conveyed to animals which consume worms &c. as do ducks and fowl, nor to animals which so frequently pluck up their nourishment by the roots.

A great deal of scientific and practical interest centres round the fact whether the bacillus is found in the soil or not. This became a burning question ^{in Hong Kong} as to how the worst of the infected localities should be dealt with.

Should the houses be destroyed by fire against this there are two potent arguments

1st Will the burning kill the bacillus in the soil? It had to be proved the bacillus was there, and if there, how deep it went down? The effect of burning the houses might not be sufficient to kill a bacillus deeply seated in the soil.

2nd Plague terminates spontaneously in even the localities where the worst hygienic arrangements are possible. Why therefore is it necessary to destroy houses or even disinfect when the disease works its own cure. The only justification for so doing is the fact that plague is endemic in many places and once it is rooted

in a locality it is expected it will recur. It is an historical error that the plague in London in 1665 was slain by the great fire. It recurred for 3 or 4 years after the great fire. A fire, even a great fire has but a partial or local effect even in a city how much less a bearing has it towards a district. Plague flourishes in sparsely populated parts and in semi-desert districts what effect then can the destruction of an entire village have on its arrest. The perpetuation of the idea does good inasmuch as the demolition of foul quarters is ensured, but that it can stay the advance or even prevent a local outbreak of the disease is a childish credence. Escape from the dilemma is not helped much by the bacteriological examination.

Kitasato once found plague bacilli in the dust of the house but never in the soil.

Yetsin found a bacillus identical in microscopic appearance and bacteriological properties as regards culture but the microbe seemed to have been innocuous when injected into the mouse or guinea pig.

Ta Ka Ki found a microbe which looked like the plague bacillus but which on close examination was really different.

This earth bacillus was shown me by another observer but I could see but little resemblance, even microscopically and disavowed its identity.

Dr. Lowson found a similar bacillus in a plot of garden soil in the Civil Hospital.

So far then the infection of the soil by a bacillus identical with the plague bacillus as found in man is not proven. This does not prove that the infection is not soil bred; it only contributes a scientific fact that so far as present day enquiry goes that the bacillus is not found in the soil. Is it then a soil bred disease? The answer is, popular belief has it so,

Deputy

but scientific enquiry has not proved the belief to be founded on fact.

Water. The spread of the disease by water is not to be entertained. The public water supply of Hong Kong is as regards quality perfect, as regards quantity something is to be derived and more especially during the year 1894. There happened to be a very ^{dry} spell and but a sprinkling of rain fell between the months of October and May a period of well nigh seven months, many ascribe the plague to the prolonged drought. This is not to be entertained. This slowly advancing disease travelling across the southern provinces of China now stayed owing to varying climatic occurrence but steadily and stealthily advancing reached Canton and necessarily Hong Kong, which, so far as immensity of communication of traffic is concerned is but a suburb of Canton. Had the interior of China been in touch with the civilized world the progress of its advance would have been watched and told from mouth to mouth as town after town was infected; but the foreigners got to know of its ravages, only when the great metropolis of Canton was attacked a city in communication with the outer world and known to foreigners.

In Canton and Macao with their shallow wells and in Hong Kong with its magnificent public water works plague ran its course until it spontaneously ceased, killing its thousands and not stayed by the quality of the water supply.

Food. ^{Animal} No food known is a carrier of plague. The Chinese ascribed the disease to the eating of infected pigs from Pakhoi. But Canton has but few if any pigs from Pakhoi and further the pigs were not ill of any disease nor was there a scarcity of pig supplied to market as there would have been had plague swept off the pigs in Pakhoi. No other food is suggested as being a carrier of plague.

Dr. Thomson in the N. W. Province of India puts down rat *infection* to the consumption of musty grain
 other is much in the suggestion to
 cause us to have & consider the *circulation*
of mites infections

however probable there is substantiation
infection to musty grain but, ^{of the} of course, that is a mere surmise.

The only animals infected are held to be carnivorous or omnivorous animals so that the consumption of flesh would seem indicated on a means of infection but neither oxen, sheep, ~~pigs~~, fowl or fish, the usual flesh providing animals have ever been known to suffer from the disease.

No vegetable is looked upon as a source of infection.

Bad Sanitation by which is meant, bad drainage, overcrowding and a general filthy condition of the habitation is no doubt a potent factor.

Bad Drainage Where no drains are present as in Yunnan, in the upland plateaux of Thibet and in the semi-district regions of Rajapootana plague is epidemic and often endemic. In Canton the semblance of a drain consists of a trench dug in the middle of the 7 foot wide street, and lay slabs of ill-fitting stone laid house vessels across the trench. This channel has no face, leads to an outlet and is flooded when the river rises on flood, or the spring tides force the water up through the slabs of stone spreading the accumulated filth broadcast at the house doors.

Macao is innocent of drains also whereas Hong Kong has been drained and re-drained by the most approved methods. The separate system being on vogue through. Yet plague came and went, appeared and disappeared spontaneously ^(seemingly) ~~is~~ respective of good or bad or no drains.

If good drains could have stayed plague then Hong Kong ought not to have had an epidemic in 1894.

Overcrowding Where the Chinese are there is an over-crowded town or village or house, ~~usually~~. Over-crowding means poverty and poverty means filth. But the over-crowding need not be a town or village it may be met with and almost invariably is encountered in the labourer's ^{hut} far removed from other habitations. In Thibet poverty, but still more the extreme

cold of the winter, causes people to herd together; and whether it is a Thibetan hut or an Irish cabin, plague on the hand, and typhus fever on the other will develop as the result of over-crowding in regions where the virus of either is attainable. In semi-tropical regions the over-crowding is increased not in winter but during the rains of summer when the populace who practically live and sleep out of doors in fine weather are driven in, ^{the} ~~to over-crowd their habitations~~ to escape the rain deluges. In Hong Kong the plague commenced during fine weather and towards the end of a long period of drought. When the rains came however the plague increased in intensity. This may have been merely the time at which the epidemic reached its climax and the high rate of mortality may have been independent of any climatic influence but the rain fall and the attainment of the increase in the virulence of the disease is a coincidence to be noted. *one thing is certain that rain falls concomitantly with the summer months in both Canton & Bombay*

In Southern Yunnan the summer months, the months of the rain fall are those during which plague appears. In Northern Yunnan on the other hand plague appears during the winter months. In Russia it ^{was wont} ~~is~~ to appear both on the banks of the Volga and in the city of Moscow during the winter, that is at the time people seek the smallest rooms so that they may be kept warm by the stove.

Poverty As long ago as 1665 the disease was baptised in London "the Poor's plague", ~~and from many sources~~. Since then poverty has been proved to be a prevailing precedent. From 1854-1858 the province of Bengazi in Northern Africa was ruined by drought and consequent failure of crops and loss of sheep and cattle in 1858 plague brought out it seemed spontaneously and claimed many victims. From Persia, Kumaon and Turkey, the same tale of extreme poverty either by failure of crops, from drought, from loss of cattle by disease, or from inundation is again and again repeated. That poverty was specially noticeable in the Southern Provinces of China is not recorded; but when one knows

the frugal diet of the Chinese coolie in what is an monetary E. C. Dorrallo to Chinaman, Hong Kong, we can safely say poverty is always with them in the interior.

Poverty and over-crowding the commonest form of sanitary guilt are responsible for many diseases and are the hand maidens of plague. So nothing else than to the better sanitary state can be ascribed. The immunity of European dwelling in the midst of natives during a plague epidemic in Asia. They breath the same air, eat similar food, drink the same water and still whether in Yunnan, Canton or the Kumaon hills it is the rarest thing possible to find Europeans attacked. In Yunnan 2 missionaries have been known to have succumbed to plague, but when one knows that missionaries very often affect the food, dress and dwelling of the Chinese it can only be wondered more have not had plague. Of course the assumption of Chinese habiliments does not mean diet and poverty, although the latter is frequently assumed as an imitation of the early Christians, but if ones house is on a dung pit it cannot afford much sanitary good if closet happens to be clean. So we are driven to the same conclusion VI 3 that dirt over-crowding are to sole foster parents of plague. It is not racial distinction.

Climatic effects The strip of country where plague has flourished during the last 20 years have climates as variable an their physical geography is apart.

From Western Arabia to the lofty Plateau of Thibet is ^a far cry; *from the Kumaon* district in the Himalayas to the flat plains around Pakhoi is surely from altitudes and climates as apart as possible. It is believed plague never ~~came~~ ^{comes} South of the Tropic of cancer, the northern limit of the sun's course in summer; but in Arabia and to a lesser degree in China we find the plague belt which striding like a crescent across the continent of Asia has its horns within the tropic ^{of} cancer.

This belt sits like a rider astride the Himalayas with its limits reaching the Red Sea on the West and the China Sea on the East. So pronounced is the observation as to the Southern limit of plague that in Egypt it is recorded how plague travelled South as far as the town of Assonan on the Nile; but dares not cross the Tropic of cancer to reach further South. These may be a very simple reason for this delimitation, namely that communication beyond the first cataract of the Nile is much less frequent than below it and that the record of disease like the record of all other matters political and religious were but little known until a few years ago. In 1883 cholera could be traced up the Nile as far as Assonan but beyond that it was lost sight of. This seems an analogous instance, namely, *but on the other hand it may merely mean that beyond Assonan* ~~that~~ the epidemic was lost sight of merely from want of recorders. Still plague has its limits and we must accept the facts as they stand that the regions within the tropics are climatically preventive.

There seems no limit to the Northern limit of the development of plague. The city of Moscow having been visited frequently, and Norway and Sweden in the old days. In these northerly regions plague flourishes in winter, but so does cholera in what seem totally opposite conditions in more southerly *climes* ~~climes~~. The explanation given of the prevalence of cholera in winter is that round a Russian house the excreta &c. are thrown out haphazard on the snow, and that the water supply is derived from melting snow gathered round the house. The explanation of the prevalence of plague during winter, I hold to be what I attempted to expound previously, viz: the over-crowding of the house in cold weather, and the bad ventilation rendered almost a necessity to defy the intensity of the cold. These considerations may serve to explain the perpetuation of both cholera and plague, *but* ~~lest~~ they do not help one towards gaining a knowledge of the Genesis of either. Whatever the explanation plague has a southerly but no northerly limit.

Varieties of Plague

1 Abortive ~~of~~ larval plague is a name given to a painless form of glandular swelling which at times suppurate and at times disappear spontaneously. In my private practice I had many glandular swellings in children some accompanied by fever. In adult Europeans 2 or 3 cases occurred early in 1894 in which they developed a general enlargement of lymphatic glands during the course of what seemed to be ordinary remittent fever. These buboes puzzled the medical men who saw the cases but even with the light of a plague epidemic following hard upon these anomalous swellings one hesitates to associate them *therewith*

During the past three or four years a form of bubo very prevalent amongst Europeans in Penang, Singapore, throughout the Straits Settlements in Hong Kong and in South China generally, has prevailed.

The bubo is either in the oblique or vertical inguinal glands and may be single or multiple. The bubo is not venereal, that is to say it occurs in men who have neither had a gonorrhoea or chancre in any form. It occurs of course in men who have had these diseases but mostly at a remote period; and the usual syphilitic treatment has no effect upon it. The anatomical characters are:- the formation of a groin bubo painless at first, slow in development, single in half the cases, double in many, multiple in a few. The gland becomes in about three weeks a large globular swelling, slightly tender to pressure, of the size of a hen's eggs. The patient is meantime feverish and out of sorts. The only cure is excision; and the operation reveals a gland or glands with multiple abscesses, lying loose in a surrounding of sero-sanguineous or sero-purulent material. The ~~wound~~ *wound* is usually slow in healing and it takes a month or so before the patient regains his strength.

These glands are not syphilitic, they are not tubercular; and I have not proved them to contain the plague bacillus. They are typical cases of Bubonic plague.

~~In conclusion it seems absurd to discuss such matters in connection with plague; but they crop up in text books and they are mentioned here merely to disclaim their connection with plague in any way. There being noticed at all is merely that during a bubonic plague epidemic one's attention is arrested by buboes in every form.~~

2 The Fulminant form of plague is a merely name signifying virulency. The same term might be used to describe intensity of infection or susceptibility in any illness.

3 There is one recognized variety which stamps plague as a distinct disease, it is that form which develops in a few days its maximum, and kills 80 to 90% of its victims in about one week.

Invasion.—The onset of the disease in malignant polyadenitis is known to be usually sudden and severe. The patient is seized with headache, fever, and prostration and compelled straightway to seek his bed. In a few hours or a few days a swollen gland develops and the disease runs its course. But we have on record many cases where high fever existed and the patient did not even feel ill, or where a gland swells up with no general feverish state. Dr. Lawson in his valuable report states: "In one case I took the temperature of an Indian who looked ill, but who had merely come to see about the burial of a friend and who complained of no unpleasant symptom. He was rather amused at my using the thermometer, but I found he had a temperature of 103° F and at the same time discovered a small cervical bubo." Be it observed, as bearing upon the intensity of the virus, that this was a non-fatal attack of plague. In many cases a bubo may precede the fever, the patient suffering no inconvenience even. Such a case is that recorded from Calcutta, where "the patient, a boy from Bombay, had had for fifteen or twenty days before developing fever a swelling in either groin. Subsequently he developed the ordinary symptoms of plague and the diplococcus was found in his blood." These two cases are sufficient to show that fever or plague may exist and the patient not be aware of it, and that glands may be affected for a fortnight before the disease declares itself in the usual way by headache, fever, and prostration. The onset of the disease, therefore, even during a plague epi-

demio, may not be so definite as we should like to believe, and as yet we have no charts recording the condition of the patient between the time of exposure and the invasion.

Signs and Symptoms.

On approaching a patient suffering from plague as he lies in bed one is inclined to disbelieve the gravity of the affection, not only in the early stages, but frequently when the disease is advanced. The spectator may be deluded as to the near approach of death. The writer's first view of plague was in a ward with 20 patients; on walking round to gather some of the more salient symptoms of the disease, it seemed as though the cases were all doing well. What was one's surprise to learn that during the half hour so spent no fewer than three of the twenty had died. So little is to be gathered from either the signs or symptoms of how near the end is. The terribly fatal nature of the malady is soon brought home to one. This peculiarity of the disease is owing to the placid appearance of the face; the wrinkles and lines of which become obliterated, in harmony with the muscular relaxation common to all muscles of the body. Moreover the mind is in abeyance, so that the patient, as he hears or sees one approach, or is spoken to,

turns towards one with a dazed look as if but semi-conscious. His attention seems to be fixed for a moment and then, either by his speech or gesture, it is brought home to the on-looker, that the patient's thoughts have wandered, and some shadowy creation of his grey matter has diverted his senses. He at times understands the request to protrude his tongue, when spoken to sharply, but the next moment he seems to disregard the order and the effect of one's words seems to have come a stimulation of some outlying centre, as the patient hesitatingly points to some fanciful object, or turns his head and eyes upwards as if looking for something on the wall above his bed. All movements are performed with a tremor and purposelessness

and it is evident his cerebral functions or their expression are stammering. The patient lies in bed for the most part on his back; on either side however he can turn and remain there for a time. Whilst on the back he invariably flexes the limb on the side on which the bubo exists, so that one knee is generally drawn up. Whilst on the side both lower extremities are drawn up. The position of the limbs is no doubt determined by the condition of the inguinal glands, that of flexion being assumed to relax the fascia in the thigh and relieve the tension over the painful glands.

Frequently the patient sits up in bed with his hands clasped over the ankles, looking quite comfortable and as if convalescent after an illness. It is only on speaking to him it becomes evident that he is but semi-conscious that he is deeply affected by plague, and that this position was assumed involuntarily in an attempt to relieve a failing heart. It is a common position in which to die; the heart refusing to respond to the extra strain thrown upon it by the assumption of the sitting position.

The face is either flushed or tinged yellow and the skin generally sympathises. So much so is the case that at first it seemed

as though one might attempt a clasification based on the variety but it soon became apparent the condition was dependent more on the stage of the development of the disease than on any real variation. The conjunctiv^{va}~~ae~~ harmonised with the skin in appearance; the mucous membrane around the cornea being red or yellow, and adding to the jaundiced appearance of the patient or to that intoxicated appearance which the staggering gait seemed to indicate.

The question of a eruption is one fraught with some difficulty. Scarcely a patient was seen without a rash, but it was on the exposed parts of the body mostly and the conclusion came to by all the observers was that the bites of insects were responsible for the spots on the skin. Petecchiae seemed to the result of these bites, and a subcutaneous homorrhage occurred readily wherever the skin was knocked or bruised accidentally. The chest and neck were at times the seat of wide spread purple patches; these caused by the Chinese custom of dry cupping which consists in seizing the skin between the fore and middle fingers or between copper cash and pinching hard and pulling at the same time. The result is a homorrhage into and below the skin in area about one inch square; but in plague owing to the tendency to homorrhage the effusion of blood was wide spread and caused an area of homorrhagic discolouration of many inches in area.

~~Dr. J. A. Lawson holds that 95% of the spots met with in plague were caused by mosquitoes or flies. This statement is in accordance with the community of medical opinion in Hong Kong during the summer of 1894.~~

~~The 5% of cases unaccounted for, showed varieties of eruptions, some quite locally around the bubo, some of true petecchiae indicating a speedy death.~~

Digestive System. The rapid coating of the tongue and the condition of the gums and mouth generally indicated a cerebral affection rather than one arising from digestive derangement;

it resembles what is met with in apoplexy or functional paralysis. At first a thin white fur covers the whole dorsum of the tongue, which speedily changes to a thicker coating with a gingery yellow tinge in the centre. By the second day frequently, but invariably by the third, the tongue has shrunk in size *become* and pointed at the tip and the gingery dorsal fur has assumed a mahogany tinge; and the whole organ becomes dry, hard, rough and fissured longitudinally, with, in the worst cases a black crust occupying the whole dorsum. The edges are red in contrast but not of the pink hue noticed in many febrile states.

The lips dry and crack, and the gums and buccal cavity generally are thickly coated, with occasional ulcers, as if form broken down petecchiæ, the teeth are covered by sordes and the breath has a heavy and foul odour.

The ~~fa~~^uces appear somewhat pink in colour; and if the cervical glands are affected one or both tonsils but certainly the one on the affected side is swollen and looks bare with some follicular activity. This is an important clinical fact in the discussion of infection or how the poison enters the body.

Those who hold that it is after the nature of an inoculation, argue that the poison, in the condition of the tonsil just mentioned, enters by the tonsil, and subsequently gives rise to the enlarged gland below the angle of the jaw; and that it is not the infection of the gland which alters the tonsil but vice versa.

That the gland in the neck is a true sympathetic bubo; as in the case of the femoral glands from a wound in the foot.

This cannot be determined by the evidence before us, although all medical witnesses in Hong Kong in 1894 would testify as to their inclination to believe the inoculation theory and the sympathetic nature of the bubo.

Vomiting is not uncommon in the early stage and frequently ushers in the disease. The vomit is of a bilious character

and at times of a chocolate colour, almost coffee ground in appearance. True Hematemesis was not met with.

The vomiting is associated with epigastric pain sometimes of severe character. It is difficult to say definitely whether the vomiting was cerebral or abdominal in its character; at times it seemed cerebral in its unprovokedness, but the epigastric pain and discomfort, the ^{attending} bilious vomiting following on injection of food point to its gastro-hepatic nature. Like many other symptoms of plague both the pain and the vomiting yielded readily to treatment, a blister on the epigastrium and a calomel purge seemed to allay the distressing symptoms.

The appetite for both food and drink is remarkable; patients seldom refused food that could be swallowed easily ^{such as} milk, soup, beef-tea, congee (rice water) or even rice itself.

The ~~appetite~~ appetite remaining good, at times up to the very end of life, adds another perplexity in the diagnosis of how gravely ill the patient is; a basin of beef-tea may be taken with relish, a sudden vomiting follows within 3 minutes and within 3 minutes more the patient is moribund.

Associated with vomiting and injection of food, but towards the end of the disease independently of either, was hiccup; at times it was painfully persistent but it yielded most miraculously to a hypodermic of $\frac{1}{4}$ grain of morphia combined with $\frac{1}{200}$ grain of atropine.

A puffy abdomen was a rarity and unless lumbar, iliac or mesenteric glands were affected there was neither tenderness nor distension. Spleen and hepatic tenderness was never extreme; nor could palpation or percussion ever reveal more than a shade of increased bulk in an occasional case. Flatness even hollowness of the abdomen was common.

Obstinate initial constipation was frequently followed by looseness of the bowels or diarrhea of a slight or severe nature. The constipation seemed to correspond to the jaundiced aspect

and the bilious vomiting but when the constipation was relieved either spontaneously or a dose of calomel all these were dispelled together. It was the fashion during the late epidemic, and a very excellent one to follow, to give a purgative Calomel.

The faces are hard, dark yellow or brown in colour. When diarrhoea is met with as it occasionally is at the end of the first week, the dejections are of a light mustard colour and frequently involuntary. Some motions are considered ^{rather} ~~a good~~ favourable sign.

The Temperature rises rapidly; in two or three hours may reach 105° or 106° . After 36 hours the temperature subsides, even in rapidly fatal cases may not rise again. Frequently, however without evident reason, the temperature after a gradual fall for three or four days a larger may suddenly rise to 105° or 107° (in children) & that too without fatal ~~to~~ indication

In cases which do well the temperature gradually but irregularly falls, & becomes subnormal after 14 days or so.

Yet again it may rise at the end of the fourth week even, a recurrence of fever is not infrequently observed.

Circulatory system.

The pulse varies in force, frequency, or character. In the early stages it is considerably ~~accelerated~~ accelerated indicating a range from 85 to 140 per minute. Movement has ~~little~~ no perceptible effect on its frequency & it remains at the same rate whether the patient is lying, sitting or standing.

The force of the pulse varies more than any other condition, at times so it is compressible, & again ~~hard & full throughout~~ seldom

~~wrist indicating a weakened ventricle and an easy blood flow through the peripheral vessels. The radial pulse is however frequently~~
 ample to a degree, might even be termed bounding ~~but~~ is never incompressible. This condition is fairly constant in the early stage of a case with gradual onset; but curiously enough it may remain throughout and the patient may have a pulse which is full ample or even bounding immediately ^{before} sudden death overtakes him, evidently from sudden nervous failure of the heart. The pulse tracings published in the British Medical Journal August 25th 1894 are here reproduced. ~~Dr. Lowson's report issued June 1895 shows pulse tracings with similar issues.~~

It will be readily noted on perusing these tracings that the line of descent as far as the aortic notch is deep and low indicating a marked fall in arterial pressure. Dicrotism is not a marked feature, and appears at times more evident in the sphygmographic tracing than palp^{able} ~~itate~~ to the finger; an almost anacrotic line is apparent in several of the pulse beats to the right of the No. 1 tracing.

The condition of the blood and smaller blood vessels shows blood deterioration and a *vaso-motor* paralysis. It is owing to these altered states that hæmorrhages occur without seemingly any pathological change in the organ affected. Hæmoptysis, although not common, occurs without any discoverable lung affection; Hæmatemesis or Melæna may be present and the abdominal organs seemingly sound. Bleeding from the nose, sub-conjunctival hæmorrhage, hæmaturia and uterine hæmorrhage are all affections which occur with certainty ~~in~~ every epidemic, but the number of cases are few and any hæmorrhage is the exception. Native reports have it that hæmoptysis is common; but this is not confirmed by accurate observers. To the native mind coughing or vomiting blood is a phenomenon to be remembered and spoken about, and the magnitude of the occurrence grows with each tale-bearer, until it is unfolded ~~to the foreigner~~ as a matter of

consequence in the enumeration of the symptoms. This is, I believe, the explanation of hæmorrhages being so frequently recounted, ~~as I observe they are chiefly magnified~~ by writers who are reliant upon natives for the information.

Associated with this state of blood and blood vessels are the subcutaneous hæmorrhages met with. Petechiæ, no doubt, occur in some cases scattered over the body discretely, & mosquito bite, or bite of other insect, a bump, or knock will almost certainly cause a subcutaneous hæmorrhage. Around the buboes petechiæ are common, assuming the appearance of a blotchy rash, or a general subcutaneous purplish discolouration, as if from extensive hæmorrhage, appears. This appearance around the apparent glands, gives rise to the notion, that hæmorrhage from the lung or intestine &c. may be accounted for by a deep seated diseased gland in the thoracic mediastina or in the abdomen. That this gland infiltration may be the lesion which causes on the one hand hæmoptysis without evident disease of the digestive part. Nothing is more common post mortem than to find mesenteric or retro-peritoneal glands extensively involved without the condition being noticeable during life. The phenomena presented by the blood itself acquired during the epidemic is confined chiefly to the important fact that a bacillus is present. This fact obscures all other observations and raises the record above that of a mere re-iteration of what is met with in almost any prolonged febrile state. The blood showed ^{on 2nd day} ~~on 2nd day~~ ^{temperature 104°} ~~temperature 104°~~ 11 a.m. *August 2nd*

- 1 Red corpuscles 15% normal (that is rounded and smooth.)
- 2 Red corpuscles 85% crenate *Some* even roseate *in* appearance like the so called plasmodium malaria.
- 3 White corpuscle 1 to 180 Red.
- 4 ^{an} Melanin masses or frequent lumps and pigment laden white corpuscles as if the red corpuscles had lost their pigment and found refuge in the *protoplasmic mass* of the white.
- 5 A few free minute pigment bodies.

The Heart sounds are not
conspicuously altered, neither
beats a regular rhythm with
but irregularity, in the cardiac
impulse & the intensity of sound
is well nigh constant.

The Respiratory tract during the
Hong Kong Epidemic maintained
a remarkable immunity,
an occasional Pneumonia or
pleural effusion occurred but
much less frequently than
during other febrile diseases &
even during other recorded
epidemics of Plague.

It will be remembered that
one of the names given to
the Epidemic in Astrakhan
in 1877-79, was "Gonorrhoic
pneumonia with buboes" & during
the present epidemic in the
Bombay Presidency pneumonia
is a very frequent cause of
death; and that deaths from
pneumonia are put down
to Plague in fact the reverse
is the case for the fact of pneu-
monia being present serves as
a good note whereby the diagnosis
representing the disease as Plague
is avoided. We can be no
doubt however that pneumonia
is a very frequent accompaniment
of Plague ~~also~~ ^{and} ~~although~~ the latter
met with in South East China
was perhaps peculiar in that
~~the~~ cases affecting were exceptional.

The Governor's Palace

Butzenborg

Serabaya

The abdominal organs

neither clinically nor pathologically
at the Post mortem Examinations
were the abdominal organs found
characteristically affected.

The liver boundaries ~~are~~ are definitely pronounced both by palpation & percussion indicating a fulness perhaps but no great enlargement of area.

of area
No spleen ~~with~~^{is} in many, plague
cases, found enlarged; but we
have to remember that - in malarial
countries, this sign has to be
discounted. ~~as~~

The Kidney. Throughout the 1834 epidemic in Hong Kong the urine was unaltered in specific gravity, colour & quantity & chemical composition; but during the recurrence in 1836 ~~the~~ albumen was frequently met with. Admitting proofs such as required of the unstable nature of the evidence of this disease.

In fact the 94. outbreak was
characteristic by cerebral ~~vascular~~
~~the~~ circulatory disturbance
the 96 by abdominal symptoms.

~~The academy itself~~

Inspection of the abdomen
itself reveals that in the

magnity, & even it is flat
& concave even as if the intestines
were empty, but it is not in-
frequent to find it full & tense.

The condition depends on the
state of the retroperitoneal
glands; as it is not uncommon
to find the lumbar & iliac glands,
the seat of disease & even the
mesenteric glands enlarged
& characteristically 'leukemic'.

The Haemoglobin is diminished after the 2nd day of the disease.
(Lowson)

The cerebral state. From first to last plague patients are affected mentally. The disease is ushered in by an intense headache, chiefly temporal, followed ^{or} speedily by one ~~another~~ of the following states:-

- a. A listless ~~apathetic~~ condition, associated with a placid expression, a dazed look, as if semi-conscious; in fact, the patient seems as if intoxicated by alcohol.
- b. A very common occurrence is sudden ~~delirium~~; the pupils dilate, the skin flushes, ^{the patient} he tosses about in bed and attempts to get up; and may dash at the open window or attempt to reach the verandah, ^{what seems} with suicidal intent ~~him~~.
- c. A comatose state, may and generally does succeed either or both of the states ^{described} ~~described~~ as a pathetic and furious.
- d. Convulsions ~~recorded by Lowson~~ were very rare. One patient I saw ~~and~~ ^{had} had a series of convulsive seizures easily set up by the slightest skin touch. The convulsions ^{was} evidently of a functional character and under ^{Bromide} ~~of Potassium~~ they speedily disappeared and the patient permanently recovered.

Subsultus tendinum is regarded as a constant sign; picking at the bed clothes, muttering, suspicious, eyeing of attendants are all in evidence in plague.

In many cases consciousness is maintained up to almost the moment of death; on the other hand ^{intermittent periods} ~~attacks~~ of consciousness, and ^{attacks of} ~~delirium~~ are the rule.

this ^{fact} ~~is~~ is obscured by the extensive peri-glandular oedema. Pain of an acute nature ushers ^S in the adenitis, and pressure causes exquisite ^{site} suffering. Pressure over some one of the seats of ^{deep} glands groups may be the sole means of eliciting the bubonic ^{tender} nature of the disease; as evidence from swelling, or *tumefaction* even, is frequently wanting. Glands around the deep *femoral artery*, especially, are apt to be missed when ocular proof is alone depended upon.

The multiple nature of the lymphatic infection was so extensive at times that *a chain* of glands from the thigh along the iliac region to the lumbar region showed a mass of inflamed glands bound together by the semi ^{gelatinous} ~~viscid~~ material effused around *the* plague infected glands.

On examining the *adenitis* ^{white} ~~will~~ developed or, developing, it will be found that the skin is frequently discoloured over and around the gland. The discolouration may assume the nature of petechiæ or of a livid purplish *injection*, wide spread, and with petechiæ dotted around the margin may cover the whole of, say; *Scarp's* triangle. The actual gland (by the 4th or 5th day) cannot be ^{individualised} ~~felt~~ owing to the soft, and in places boggy, nature of the ^{ad} ~~edematous~~ and puffy surroundings. A doughy feel in parts gives place to that of fluctuation and it may be that purulent effusion has taken place. More often however this is not the case; sero-sanguineous fluid only, ~~not~~ ^{not} ~~pus~~, following an incision into the mass.

As the swelling increases in size, the pain becomes less; and although tender up to the period of spontaneously sloughing, the acute agonising pain accompanying the examinations of the recently inflamed gland is not present.

When an incision is made into a well-developed mass of plague-inflamed glands; the moment the subcutaneous region is reached serious fluid oozes away. The tissues superimposed ^{ed} will be found rather tough and leathery to the knife, but swollen ~~as~~

~~it was~~ into a fibro-gelatinous mass by the fluid. The peri-glandular tissues is injected by a sero-sanguineous fluid, which becomes more and more ^{viscous} ~~sandy~~ as one approaches the gland, betraying the intense hyperaemia which is present. The extent of this infiltrated tissue is at times extensive both in depth and breadth. Starting over the femoral region it may spread backwards, reaching even the gluteal region; and downwards and inwards over lying the inner side of the thigh and Hunter's Canal.

Deep down at the bottom ^{of the wound} the gland will be found looking white & glistening in a dense capsule.

The gland when cut into shows a marked capsule, & a reddish, pulpy, shagreened looking tissue within. According to the stage of the disease in the gland uniformly red, mottled, or peppered with white or yellow points where suppuration is actively threatened. If the patient ~~has~~ died late in the disease you may be met with as in other suppurating glands.

Varieties.—It is not my intention to mention merely the varieties of symptoms which predominate at any one time or throughout any one epidemic, but to draw attention to varieties which are so pronounced in their character as to justify the belief that whilst dealing with this subject we are actually attempting to bring in line under one heading diseases which may be even separate species. The commonly accepted types are these: (1) abortive (or larval) plague; (2) typical plague; and (3) fulminant plague. The two latter are so well understood and have been so largely and fully dealt with elsewhere that there is no advantage in recapitulating what can be read in any text-book; but I wish to draw attention to the first-named. The abortive form is sometimes styled larval, ambulatory, or pestis minor. The names partly declare the character of the disease and create a condition so apart that it has at all times been separately described. It is at the same time so indefinite in its duration, so unstable in its relation to malignant plague, and so uncertain in its onset and departure that up to the present no scientific precision has been assigned to the relationships. As long ago as the year 1666, when plague ravaged London, we have it on the authority of Sydenham that "at times tumours may break out without either fever or any important symptoms. . . . Those to whom this happens may walk about in public as usual and attend to the common duties of life. . . ." Foderè, writing of plague in 1820, observes: "In the Levant and in the Marseilles epidemics of 1820 cases were to be seen which were not ushered in by any alarming symptoms and where the natural functions were undisturbed and where buboes and carbuncles appeared without fever or only with slight fever, or the buboes went on to a healthy suppuration more or less prompt, or even disappeared and went on to resolution without the help of art, without any inconvenience, and with a perfect integrity of all the functions. This state is comparable to benign small-pox, during which children play together and walk in the streets without any precautions, no care being taken of their treatment and yet terminating favourably. It is the benign plague of authors which is observed when the disease commences and when it is at its end, though it is rarely seen in the middle period, which is entirely

devastating, but it is not less plague and it no less merits the attention of physicians and magistrates." Again, in all recent epidemics in Tripoli, Mesopotamia, and Astrakan non-fatal, mild bubonic disease occurred both before and after the epidemic. We have it on the authority of Dr. Dickson that in Mesopotamia, previously to the manifestation of plague, glandular swellings free from fever prevailed. All this evidence seems to indicate a close connexion between benign and malignant polyadenitis; but we have some evidence of the separate existence of the two diseases. 1. In Mesopotamia Tholozan records: (a) that mild non-fatal bubonic plague existed from 1856 to 1865, during which period (nine years) no malignant type was observed; and (b) that after the epidemic of malignant plague (1866-67) passed over the same form of mild plague existed. Did it exist during the epidemic? This is not known, for it is not likely that persons suffering from so mild an ailment would present themselves for treatment to foreign medical men at a time when all swellings were viewed with suspicion. There is, therefore, no evidence to show that the two diseases did not co-exist seeing that it existed nine years before and perhaps as long afterwards. The present belief is that the mild develops into the malignant type, and when that subsides it is once more deprived of its malignancy and becomes benign. We have no evidence of any disease behaving in such a manner to such an extent as claimed for plague. 2. In Calcutta six years ago an outbreak of mild, non-fatal plague occurred in the fort amongst the troops, but it was not followed by the malignant variety. 3. At the present moment in Calcutta there is reported to be a form of fever with glandular swellings, and also a form of glandular swellings without fever, and it is further stated that these are constantly met with in medical practice in Calcutta. 4. In 1890 the variety of idiopathic buboes, of glandular swellings with fever, and of mumps met with in Hong-Kong before referred to was not followed until four years afterwards by plague. These records go to prove:—1. That long periods of time may intervene between the prevalence of specific glandular swellings and the outbreak of malignant plague. 2. That glandular swellings may exist for nine years before malignant plague appears, and continue for years after it has disappeared. 3. That independent outbreaks of glandular swellings (pestis minor) may come and go and no

The latest evidence we have from Calcutta is conflicting in the extreme. We have it to hand on trustworthy evidence that people are suffering from fever with glandular enlargement, that the disease is regarded as pestis ambulans—a mild bubonic plague—and that the *diplo-bacteria of plague* are found in the blood. We have hitherto regarded the presence of the bacillus in the blood as the one token of the disease being malignant in type; but we have now contrary evidence to consider, and if we are to accept it, we must believe that specific glandular swellings in the plague belt may be caused by a definite bacillus, and at the same time admit a varying degree of malignancy to that bacillus. Professor Cunningham—a most competent observer—declares that the bacillus found in the Calcutta cases is non-infective, and that probably it is not the same bacillus as that described by Kitasato in 1894. This is stated in the face of positive evidence by careful observers which cannot be set aside thoughtlessly. Time alone will answer this question. In the meantime, however, it is feasible to suggest that there may be but one bacillus, and that according to its state of development, or in consequence of its evolution, different types of the disease may prevail in the human body, each associated with a definite train of symptoms, characterised in the one by glandular swellings merely and in the other by a toxic infection of extreme intensity.

The bacillus. — The bacteriology of plague began with the advent of Kitasato from Tokio during the Hong-Kong epidemic of 1894. On June 14th, 1894, Kitasato demonstrated the bacillus, which was confirmed afterwards by Dr. Yersin of the Pasteur Institute in Saigon, working in an independent manner. The bacillus has been variously described as (1) a diplococcus; (2) diplococcus enclosed in a delicate capsule; and (3) a short bacillus with the ends somewhat rounded and a clear space or band in the centre. Professor Woodhead writes:—"The size varies considerably according to the staining method employed, the organism appearing rather smaller in the watery gentian violet solution than in the glycerine fuchsin solution. It is plain that the size of the capsule and its staining capacity vary very considerably, and it is quite possible that this appearance of a capsule is like that which is sometimes met with around the bacillus of anthrax stained in special solutions in that it may be simply a refraction from the albumin from the constricted bacillus due to the drying

and other processes of preparation." In the figures drawn by Woodhead an organism was met with in some specimens which seems to indicate a development after death. The last statement may be the most important of all, and its confirmation will be anxiously looked for during the present outbreak in India. It may be the first step towards a knowledge of the life-history of the bacillus in the same manner as the flagella of the plasmodium malaria are found to develop in the field of the microscope some half hour after removal from the body. Dr. Yersin undertook some experiments to test the truth of the infection of the soil, the results of which elucidate the toxic nature of the bacillus. He found (1) that a bacillus in some respects resembling in its appearance that met with in the human body was found in the soil of Hong-Kong during the plague epidemic; (2) that the bacilli of the soil and body occasionally behaved similarly in culture experiments; and (3) that the culture specimens of soil bacilli were, however, innocuous, possessing no virulence when injected into mice or guinea-pigs. Dr. Yersin further found that in his cultures of human plague bacilli there were microbes of different virulence, some mildly powerful and some altogether powerless to infect either mice or guinea-pigs. Dr. Lawson, in conjunction with Dr. Takaki, Dr. Kitasato's assistant, found a bacillus in the soil of the garden near his house which closely resembled the human plague bacillus, possessing most of its microscopic attributes, but showing none of its toxic properties. The district whence the soil was obtained lay within the plague-stricken area, but considerably removed from any infected house.

The conclusions to be deduced from the above rehearsal are:—(1) That plague bacilli vary in size; (2) that cultivation experiments produce bacilli identical in appearance but possessing different toxic and infective powers; and (3) that a somewhat similar bacillus is found in the soil (whether at all times or not is not known) which resembles the non-virulent form the result of bacteriological culture.

The Recognition of Plague.

The whole question of Propriety ^{in Asia} depends upon whether it is a native or a European that is attacked.

During the 1894 epidemic the deaths amongst natives amounted to the extraordinary percentage of about 95.

The ordinary Chinese belief was that five persons out of a 1000 recovered.

During 1895. Some 50 persons had plague & 50 died

During 1896. The numbers

are well nigh the same
as in 1854 - a few a very few
recovered.

For Europeans the death
rate in 1854 was two deaths
out of 11 seized.

It would seem that not only
the well clothed & well fed portion
of the community have but
little chance of taking the
disease; but when they are
seized their chances of recovery
are infinitely greater.

Very few of the better class of
Chinamen were afflicted & the 3
Japanese Doctors who were
seized 2 recovered.

As to infection and contagion, direct contagion, that is, transference of the disease from the sick to the healthy by bodily contact would appear at the first glance to be an element in the transmission of plague. How otherwise can we explain the circumstance that, when a case of plague occurs in a house, those dwelling in the house and brought in contact with the sufferer are almost certain to be seized, and not only so, but few, if any, of the other inmates escape. The next in order to be seized by plague are the relatives of the sick and the visitors to the house, who, going to their own homes, set up a fresh focus of infection. Of the community, medical men, clergymen, and attendants, who are brought in contact with the sufferers, more readily acquire the disease than others who are alien to the family and house. These facts would appear to prove conclusively the contagious nature of plague. But there is another side to the argument. As long ago as 1835, when plague was epidemic in Egypt, out of ten French medical men who attended the sick only one contracted the disease. Further, we have the well-known case of Balard during the same outbreak, who, anxious to prove his contention that the contagious nature of plague was much exaggerated, for two days wore the shirt of a patient who had died from plague. Yet Balard did not contract the disease. More recently we have the evidence of the Hong-Kong epidemic in 1894. None of the European medical men in attendance on the plague patients, in all, some fifteen in number, were attacked by plague. Those men were freely exposed in the wards and at post-mortem examinations, yet none were infected. Further, none of the British or Italian born nurses—these were the only two European nationalities represented—were attacked by plague during 1894. But a more interesting fact still is that none of the Chinese students of the College of Medicine were attacked. For six weeks these men were on duty in

turns night and day in the Plague Hospital. Eight of them acted as ward attendants and clerks throughout the period and were consequently in constant contact with the sick. A racial immunity might be claimed for Europeans, but in face of this fact it cannot hold good. If there be any truth in the acquisition of disease during an epidemic by fear or nervousness the case of these students refutes the legend. They saw that the Europeans escaped and how terribly their countrymen suffered; they had to contend with the superstitious beliefs of their country, inherited and acquired; they were breathing the same air, eating similar food, hearing daily, hourly even, of the deaths of their friends, relatives and acquaintances, yet none of them succumbed to plague. We have, therefore, two opposite conditions before us—namely, plague entering a house and seizing practically all the inmates; and, on the other hand, medical men, nurses, and native students acting as clerks and dressers escaping completely. Where, then, lies the truth of the contagious nature of plague?

As usual, there is a channel of escape from either of these two extremes of argument. The immunity above related does not extend indefinitely. The Europeans attacked during the 1894 epidemic were chiefly soldiers of the Shropshire regiment, then stationed in Hong-Kong. When plague required sanitary interference beyond the power of the local board these soldiers came forward and volunteered to help in the work of cleansing and purifying the city. The work assigned to them was of the most laborious order and they were exposed to a concentration of the contagion, for the field of their duty lay amongst the dwellings of the poorest class of Chinese who were suffering from plague. These houses were of the vilest kind of habitation mankind is acquainted with; small, windowless, low-ceilinged, reeking with filth and excretions, they presented infection in its most concentrated form. Further, the soldiers had to use spade and shovel to dig down into the accumulated layers of dirt on the floors of the houses and throw it into the street. It was this occupation more than any other to which is attributed the infection of the soldiers. During the 1894 epidemic two of the sisters of the Italian Convent contracted the disease and died, but they were neither of them Italian born; they were both Eurasians and in very humble circumstances. In 1894, also, three Japanese medical men contracted plague, of whom one died. The two who recovered were both considered to have

inoculated themselves at a post-mortem examination on a case of plague. The facts recorded would seem to show that, during the 1894 epidemic in Hong-Kong, English practitioners and nurses enjoyed an immunity which did not extend to the Japanese medical men or to the nurses of Eurasian blood.

The evidence of the subsequent outbreak in Hong-Kong in 1896 goes to show that Europeans were much more frequently attacked than in 1894, and two of the nurses in attendance on the sick contracted plague. One of the nurses was a British-born nursing sister at the Plague Hospital, where she had been for many months. She never was ill until the very end of the epidemic, when she got a sharp attack of plague, from which, however, she fortunately recovered. The sister (Katherine McIntosh) believes she was infected by a little child she nursed, and whom she frequently carried about in her arms. The other European nurse was an Italian-born sister in the convent, who believed she was infected in the same way—viz., by nursing and carrying about a child sick from plague. The Italian sister died from her disease. It is interesting to note that the patient from whom the English sister contracted her disease recovered, whereas the patient of the Italian sister died. Was the recovery of the English sister due to a lessened degree of infection, seeing that her patient recovered, and the death of the latter due to the extreme malignancy of the emanations from the body of the dying child? is a pertinent question. Nor is this all, for yet another sister in the convent died, presumably infected whilst nursing her companion. These three deaths seem to point markedly to contagion rather than infection as the means of spread, for although there were some 600 persons in the convent none of the other inmates were afflicted thereby.

Gathering this evidence together and trying to focus it we find that the soldiers who were affected were exposed—if there is any truth in intensity of contagium at all—to the most concentrated form of the poison. They worked not in the streets in the open air, but in reeking hovels for several hours together. Intimate and prolonged contact with the contagium in an active condition seems to be the explanation of their infection. The same is to be said of the two European nurses who were seized. Both attributed their ailment to intimate and prolonged contact with a generating source—viz., a child carried in the arms not for a few minutes,

but for several hours daily extending over several days. That there are, moreover, degrees of power in the contagium, even when emanating from the human body, seems to be supported by the nurse and infecting child both recovering in the one instance, and the nurse and infecting child both dying in another. Bulard sleeping in the dead man's shirt proves nothing further than that the plague-infected garment did not generate poison of an intensity sufficient to infect. The poison grew every moment more dilute, but a nurse carrying a child, throwing off contagium continuously, is an exposure of a different stamp. The immunity of the medical men and Chinese students does not annul the effect of prolonged and intimate contact as being a means of spread; for it must be remembered that the medical man "visits" only, he does not stay for a day beside one case as a relative does, nor does he sleep or eat under the same roof as does a nurse. In this way also the immunity of the Chinese students is to be accounted for. They performed their task in a newly-built, commodious, and roomy mat-shed. Freely ventilated by being raised three feet from the ground, and its lofty roof constructed so that air swept across between the side walls and the roof with large apertures in the roof itself, the chance of contracting the disease from so diluted a form was reduced to a minimum.

The practical lesson to be learned from these statements is that free ventilation, and attention to ordinary hygienic rules, lessen the chance of contracting the disease to a very great extent, not only for Europeans, but also for natives; but that all are liable to contract the disease when either from the calls of duty or wilful neglect exposure is extreme.

What has been termed the "miasmatic" method of infection bears distinctly upon the maintenance and epidemicity of plague. Miasmatic infection implies a soil-bred disease—a disease existing, as we assume malaria does, in the earth itself, in the water, or in some particular form of fermenting or decomposing material in which the germ finds a nidus. In a neighbourhood so infected the disease is endemic, as manifested by the fact that again and again the disease recurs—that after a few months of seemingly complete disappearance the disease crops up and runs a more or less severe course. The time of its disappearance may be merely one of hibernation, or the climatic conditions may be unsuited to the activity or the evolution of the bacillus of the disease; but when a sufficiency of time

Plague is infectious chiefly by the dust
arising ^{during} ~~from~~ the cleaning of a room
~~where~~ ^{wherein} Plague patients have occupied.

~~Contagion~~

Plague is contagious by ^{prolonged} intimate
contact with the Plague stricken or
with the case of persons sleeping in the
same room as ~~carrying~~ ^a nurse
carrying a diseased child in her arms.
Infection ~~by~~ ^{from} a dwelling can
be (well nigh completely) prevented
by thorough disinfection (by Chlorine);
Contagion by thorough ^{ventilation} ~~disinfection~~
of the Hospital ward & ~~avoiding~~ ^{avoiding} touching
~~the intimate & prolonged contact with~~
~~the Plague stricken~~ avoiding carrying
the ~~the~~ sick ^{diseased} in the arms

Differential diagnosis.—In all cases nowadays the microscope is regarded as the one test whereby to test the presence or absence of plague. Rightly or wrongly this conclusion has held good ever since Kitasato discovered the bacillus in June, 1894. The names given in an earlier part of the paper indicate the diseases held to be closely analogous to plague. They are "typhus," "adynamic typhoid," and "intermittent fever"; but the word "bubo" is affixed to all. Still the name gives the drift of its applications and enables us to apportion its place in the category of disease.

Typhus fever.—The resemblance to typhus fever, in many signs, symptoms, methods of spread, and circumstances of development, is worthy of more than a passing remark. It has been noted how overcrowding, whether in town or country, is a potent factor in plague as in typhus fever. Hemorrhages—so characteristic of typhus fever—are so frequent in some outbreaks of plague as to occasion the use of the synonym of "hemorrhagic fever." The infectious nature of the two diseases appears so similar, so markedly "catching" to all inmates of an infected house, so sparing in their seizure of visitors. Such instances of the resemblance between the diseases might be multiplied with the result that we might become converts to the opinion expressed by Murchison that "Plague is perhaps the typhus of warm climates, the two diseases being generated from similar causes and differing only in intensity

from the effects of climate and other collateral circumstances." The specially characteristic sign of plague is the presence of buboes; the specially determining sign of typhus fever is the rash. Both may be absent in either; or we may find buboes in typhus fever and petechial eruptions in plague. The eruptions met with in the Hong-Kong epidemic were never characteristic, although many cases showed spots on the skin. These in most instances were no doubt mosquito bites, but the occurrence of a pink blush around them gave a specific appearance to these petechiae. Further, the distribution of typhus fever corresponds in many ways with that of plague. Typhus fever has a southern limit, not being met with within the Tropic of Cancer in either the old or the new world. Typhus fever has never reached Australia or New Zealand, or appeared south of the Equator. Typhus fever, however, is accredited to have reached the United States and Canada, where plague has never been. With this solitary exception the distribution of the two is synonymous. The pursuance of this subject would lead me far beyond the limits of time at my disposal; but there can be no doubt that we have two diseases generated by a common cause, overcrowding and personal filth; and, if we assume the supervention of a specific microbe upon such states, typhus fever or plague, as the case may be, may arise.

I have yet another disease to add to the list which may be mistaken for plague. On June 26th, 1894, when the plague was at its height, I saw a Parsee patient dwelling in a house in which plague existed, suffering from fever (104° F.) dry tongue, headache, backache, and large swollen glands in the left groin which had suddenly appeared. Plague seemed the only diagnosis, and the man, much against his will, was sent to the plague hospital. In two days he came back again quite well, and on examining him I found his urine thick and milky. That night I found filaria in the man's blood, and knew I had made a mistake in the first instance. Of course, the mistake is most likely to happen, but nevertheless it is not pleasant to think that we had subjected the man to the terrible danger of plague infection.

The conclusions drawn from a study of the spread of plague are:—I. Varieties: 1. The varieties of plague known under the names (a) fulminant, (b) typical, and (c) pestis minor, are allied. 2. The cause of fulminant and typical plague is a diplobacterium in the blood and tissues. The cause of pestis minor may be an allied diplobacterium, but with a lesser toxic power. 3. An appropriate name for the fulminant and

typical plague is "malignant polyadenitis." An appropriate name for the mild variety (pestis minor) is "benign polyadenitis." II. Infection and contagion: 1. Plague is infectious chiefly by the dust arising during the cleansing of dwelling houses which plague patients have occupied. 2. Plague is contagious by prolonged and intimate contact with the plague-stricken, as in the case of a nurse carrying a child ill of the disease. III. Distribution: 1. Plague is met with in a definite area of Asia which may be termed the "plague belt." 2. That the home of plague at the present day is Mesopotamia and the countries adjacent. 3. From Mesopotamia as a focus the plague may spread northwards to the Caspian Sea, westwards to the Red Sea, southwards as far as Bombay, and eastwards as far as (Formosa) the China Sea. 4. During the present century plague has shown a western retrocession and an eastern accession of virulence. IV. The bacillus. 1. Typical plague (malignant polyadenitis) is associated with pestis minor (benign polyadenitis). 2. A bacillus of somewhat similar appearance microscopically is reputed to be found in both. 3. The bacilli differ in their toxic powers only (?). 4. A benign polyadenitis may run its course without being preceded or followed by the malignant variety. 5. Malignant polyadenitis may run its course without being preceded or followed by the benign variety. 6. The bacillus of the benign variety attains malignancy by passing through some intermediate host, possibly, but not probably, the rat.

Name.

Age.

Address.

Disease.

Vol. and
Page.

Bubonic Plague

Nomenclature.

Neither the word Bubonic nor the word Plague are unsatisfactory as being proper descriptive terms of the disease under consideration.

The term Bubonic implies a sympathetic swelling of a gland - in Plague it is an idiopathic swelling - a true ~~but~~ bubonicle that we have to deal with.

The glands in syphilis are not of the same kind although the term may be correct; ~~the same way~~.

The ~~word~~ word Bubonic is correct ~~and~~ ~~not~~ if infection can be traced to a wound in the area over which the gland presides, but this is not proven.

Plague. The term ~~and~~ ~~is~~ ~~not~~ ~~due~~ ~~to~~ the class of diseases to which it belongs it is a more popular expression unworthy of scientific nomenclature.

Disease
4.
Bubonic

Post-mortem naked eye appearances afford but little information as to the nature of the disease. On opening the chest and abdomen the lungs are observed to recede abnormally in the chest, leaving the heart and pericardium peculiarly prominent. The pleurae and peritoneum are here and there occasionally ecchymosed. The lungs are pigmented; the sub pleural region presents occasional ecchymosis the engorgement of the lung with blood gives a dark reddish brown hue to the organ. On opening the bronchi the mucous membrane is pale for the most part with here and there injected patches. The membrane conveys the impression of a submucous oedema; there is but little frothy mucus present in the tubes. The lung when cut into oozes dark non-aerated bloody fluid at every probe. It seems although the blood had sagged only, that the whole lung is so affected and not the posterior part merely. No consolidation is to be found as a rule, nor is congestion of the most dependant part marked. The fluid in pleural cavity is normal in quantity and appearance. Occasionally a slight increase in the quantity of the pericardial fluid was noted. No sign of recent pericarditis has been observed. The substance of the heart was firm and the muscle of a healthy colour when viewed externally. The inner third however

of the wall showed a definite pallor, a yellowish-white hue as if from rapid granular change in the heart tissue commencing from within. 1000 cases showed signs of very recent endocarditis, but as a rule the valves were healthy. The blood in the heart and large vessels was uniformly fluid & except immediately around the mitral valves. No red staining of the endocardium was noted. The tongue seemed swollen at the base the granular structures over the posterior third being large, bulgy, and oedematous. The laryngeal mucous membrane seemed pale as if from submucous oedema, with here and there flecks of brightly stained injection. The stomach, empty as a rule was streaked longitudinally by ridges with injected crests the intervals being pale and the tissue rather firm. The ileum showed Peyer's patches distinctly in younger subjects but although the contents of the gut seem to cling unnaturally to the surface of the patches there was no congestion or injection. The gut otherwise was normal. The liver becomes prominent and firm, but actual increase in bulk is not so great that it could be of clinical value. The colour is paler than natural the cut surface are of a whitish pink tinge, as though blood had been expressed from the surface, and an

oedematous condition of liver cells left the appearance which is best stated as that of cloudy swelling. The gall bladder is full in all a distended in a few cases. The bile is rather darker than usual. The spleen is invariably firm purplish in colour and increased in bulk. The cut surface are firm rather catarrhal and presenting none of the appearances met with in death from high temperature. The kidneys present the appearance met with the cloudy swelling, and perhaps that term conveys the best idea of their state. Supposing the death to have occurred in a patient suffering from femoral bubo it will found in cutting down upon the gland that the surrounding tissues are full of serous fluid & which infiltrates the periglandular area for 2 or 3 inches around. A large patch of subcutaneous hemorrhage frequently extend outwards as far as the gluteal region. The gland when cut into shows a marked capsule, and a reddish pulpy splenic-looking tissue within according to the stage of the disease is the gland uniformly red mottled or pitted out with white or yellow points where suppuration is actively threatened. If the patient has died late in the disease pus may be met with may be met with as in other suppurating glands. No is one gland affected a chain may be diseased

leading up beyond Poupart's
ligament to the iliac fossa and far
beyond the disease tract may be traced
to the lumbar and retroperitoneal glands
generally. Cervical gland, axillary
glands mediastinal, and even
mesenteric glands may be affected.
The enlargement of the latter is
perhaps the most interesting as without
intestinal lesion these glands are
affected in common with others as
proved these by the presence of the
bacillus in all.

Quarantine.

The questions in connection with this subject are many:-

The incubation period is perhaps the ~~primary~~ ^{primary} one. What leads me to guess ~~at~~ ^{is} us.

1st That during an epidemic 3-6 days is the period of incubation. This may be regarded as a scientific truth.

Beyond this however the evidence is not so conclusive. Take the most recent record.

a). The case referred to as occurring in Calcutta namely the boy who had travelled from Bombay on whom glandular swellings had pre-existed, did not develop typical plague until many days after. "He left Bombay on the 23rd Sept 1896 & arrived in Howrah on the 26th Sept. About 15 days before he left Bombay he noticed the gland at the left groin to be enlarged & painful, then the gland in the right groin began to enlarge, but he felt quite well until he reached Nagpore on the 24th September" That would make 16 days in all from the time of noticing swellings in the groin until he felt ill, ^{but} it was not until the 28th or 20 days after he first noticed the swelling that fever set in.

It cannot be said that the disease did not begin to show itself for 20 days, for we have the evidence of glandular swellings long before, but the invasion of fever did not begin until that late period.

Either the glandular swellings were due to causes other than Plague, or he became infected ^{by Plague} ~~but~~ just before leaving Bombay; or the incubation period of the disease for glandular feverish symptoms are not co-incident. ~~can~~

Another ^{instance} ~~case~~ ~~that~~ I would refer to is the case reported to have occurred at the Greenwich Hospital. If the ^{Calcutta details} ~~above case~~ is correctly reported, the Greenwich Hospital case is readily understood; as a vessel can come from Bombay to London under the 20 days.

In a previous part of the paper I stated that there was no evidence of Plague in Bombay earlier than the 9th of Sept^r, or 10 days after the time that it was possible for the patient at Greenwich Hospital to have left Bombay. But I have evidence that may elucidate this point. A writer in the ^{in the present epidemic}

The period of infection :- During the 1894 epidemic the patients were regarded as infectious so long as any bacilli could be found in the blood. It was a good practical working basis and it seemed to answer admirably. In most instances the bacilli disappeared in a ~~few~~ 5 to 6 weeks. ~~Here I to recommend any test of~~

I do not think a better test could be selected, ~~but~~ it ought to constitute the basis of infective ^{power} a freedom ^{from the disease} ~~from the~~ ~~the power of contagion~~

of the possibility of being infectious or of freedom from the disease.

Of course the blood does not always show bacilli even during the height of the disease, but we may ~~however~~ lay ^{it down} as a useful

maxim, lay it down as a maxim that their presence in the blood renders the person ~~unfit to mix with the~~ ~~unfit to mix with the~~ a possible source of contagion.

Practically, Quarantine should be determined by the presence or absence of Plague Bacilli in the blood. or if

still further test be needed the sputum should be examined as well.

Cordons: - At present cordons
~~are established~~ in India, rail
way stations are watched, all
travellers by ~~each~~ vehicle or on
foot are inspected, practically
medical inspections ^{in fact} ~~cordons~~ are established around
the infected districts. This is a totally
different thing from a cordon.
The justification for ~~it~~ ^{establishing a cordon}
in modern times ~~that~~ is perhaps based upon the
fact I said to have arisen in
Russia on several occasions during
the present century by the estab-
lishment of military cordons.
Curiously enough also we have
in this country ~~some~~ ^{special} quarantine
laws against Plague & Yellow fever.
~~still remaining in force~~ They would
no doubt be put in force were
plague to ^{seriously} approach our shores.
Are these laws retained merely
because no one has been so far
troubled to remove ~~them~~ ^{the} remains
a land mark or are they of
any practical value.
By a study of cordons we ^{ought} ~~could~~
to be able to arrive at some notion
of the spread of infection.
If a military cordon Russian
can prevent Plague travelling
beyond an infected village
then whether the soil or the
rats or the atmosphere or
the clothing are at fault but
man only can be the carrier.

(and can
be used
only.)

~~The amount of~~

This would seem definite & conclusive.
Cordon, however fail but their
failure may be caused by them
being imperfectly carried out.

~~Nothing is more difficult than to~~
carry out than a strip cordon;
apart from direct bribery, the
necessity for an individual to
break through ^{may be} so great that the
death may be preferable
risk of life a punishment in so doing
may be desired.

I can not enumerate ~~the~~ what
is apparent to every one how
a cordon may be set at naught
by travellers, ~~by assassins, by~~
merchandise, food, clothing, ~~boats~~
streams of water, ~~animals~~ four footed
animals, snakes & birds. A perfect
cordon is an impossibility, unless
man alone is the carrier, & he
be prevented exit & entrance through
the cordon. A Russian guard round
a Russian village especially if
now lies in the front of an open
country ~~that may be~~ ^{possible} ~~possible~~; but a European cordon
of natives round a tropical
country round a native ^{Town} ~~city~~
is perhaps one of the best means to
ensure spread.

At the present moment we
believe by intelligent observation

How do they bear in Quarantine
Although the evidence is limited
I would hold that it points to
this; namely, that mere absence
of fever is not sufficient to
settle the point whether ~~or not~~
the period of incubation is
ended or not; 2/ that a ~~patient~~
person coming from an infected
area with a glaucomatous swelling
of non-venereal origin is to be
viewed with suspicion, even if no
fever is present, & if ~~so~~ even 20 ^(or more)
days have elapsed between
the exposure & the date of exam-
ination.

This is not a comforting
statement & I only hope that
my information is wrong; but
it is difficult to understand
the Greenwich Hospital ^{cases} ~~Cases~~
otherwise. ^(+ similar)

Further
I

He has referred frequently to
a virile & a non virile bacillus
but if these cases are true it
would seem as though the in the
same patient ~~as bacillus~~
~~bevirgin~~ can become a virulent
a bacillus may ^{pass from} ~~pass from~~ being
a non-virulent but virulent
form in the same person ^{offer an} ~~even~~ ^{which}
^{we know of} ~~the~~ ^{disease of} ~~the~~ ^{person}

