

Transactions of the
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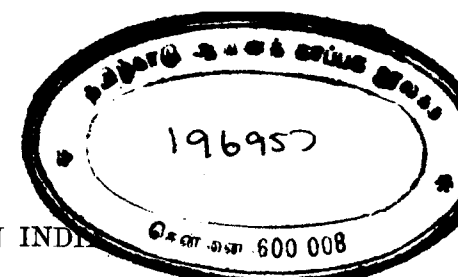
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MADRAS:

G. PLUMBE, AT THE LAWRENCE ASYLUM PRESS, MOUNT ROAD.

1900.



PLAGUE IN INDIA

The origin of the 1896 outbreak of the plague in India must always remain a matter for conjecture without any possibility of definite proof. That it was imported from one of its endemic centres, or from one of the places in which it was then prevailing epidemically, has been assumed as beyond refutation, but, as will presently be seen, it has been epidemic so often in the old days within the limits of the Bombay Presidency that it might without a strain of meaning be said to be endemic there, in which case the recent severe outbreak can be placed on the same level as epidemics of cholera, small-pox, and such like diseases.

Areas in which plague is known to be endemic exist in Yunnan, in China, Mongolia, Persia, Assyr in Arabia, and perhaps Thibet and Siberia. There is also a small tract on the southern slopes of the Himalayas, known as British Garhwal and Kumaon, where a disease called Mahamari, said to be identical with plague, breaks out at frequent intervals. From Yunnan plague spread to Canton in 1894, and to Hong Kong in 1895 and perhaps Bombay in 1896.

Other possible routes are from Persia *via* the Persian Gulf and from Kumaon. The latter is the shortest and most practicable route, but it is not looked upon with much favour by the Bombay authorities, and doubts are still expressed concerning the identity of plague and Mahamari.

The earliest record of the existence of this disease in Kumaon is in 1823, and from that time until 1897 there have been localised epidemics every year or two in some of the tiny villages on the mountain sides. Rodgers¹ has expressed an opinion that the real endemic area is not in British India, but away over the mountains in Thibet or beyond, and that each Kumaon outbreak is due to fresh importation from without. Be that as it may, from the hills Mahamari used to descend occasionally to the plains, and small breaks have continually been reported or described by the Civil Medical Officers in charge of the neighbouring plain stations. Relation of these outbreaks have been directly traced to the mass cannon men.

¹—See page 452, Special References.

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Assuming the identity of the diseases, and certainly the majority of medical men in India believe in it, what could then be more likely than the transportation of plague from the hills to Bombay during 1896. There is no need to be sceptical on the ground that it has not occurred since the existence of Mahamari was discovered in 1823, for the origin of the Pali plague in 1836 is wrapped in mystery, and none can now say that it was not imported from Kumaon. Moreover, these hillmen live a life apart from the rest of mankind, holding but little communication with the inhabitants of the plains; and it has always been their habit to evacuate a village in which plague made its appearance and camp out for a while on the hill sides. The Bombay Police reported that certain hillmen from that region had arrived in Bombay in July 1896 which adds plausibility to the supposition.

I will now briefly bring to your notice those outbreaks, which contemporary records describe sufficiently clearly to leave no reasonable doubt that they are identical with the plague of to-day.

A pestilence is mentioned² as having occurred in Guzerat in 1575 and being very severe for six months. The people all fled away to avoid it. It was called by one of the names usually applied to plague, namely, "Waba," but no description is available to identify it. For the first reliable³ account we are chiefly indebted to the Emperor Jehanghir, whose personal reminiscences are most graphic and interesting. As many strange and incredible tales of its virulence were told then as one hears in the bazaar to-day, and the panic caused was evidently prodigious; all Hindustan and the Punjab were affected, and the disease was said to have been very prevalent in Kashmir. It lasted altogether eight years—1611-1618. Terry in his "Voyages"⁴ remarks that the English suffered heavily on this occasion, and were in no way more fortunate than the natives.

After an intermission of about 60 years another outbreak recorded, the focus of which was Ahmedabad. Thence it spread southwards to Goa in 1684 and Bijapur in 1689. In 1690 severe in Tatta in Sind. Ovington in his "Voyage" mentions that the English escaped infection to a great extent, this epidemic, which spread also to Bussorah by sea

There is mention in Elliot's History⁵ of a localised outbreak at Berhanpur, about 300 miles north-east of Bombay, a few years later, and it is possible that there was another in that neighbourhood in 1770.

From 1812-1821 there was another severe visitation in Guzerat, Dutch and Radhanpur. This also reached Sind. It was investigated by Dr. Whyte, who submitted an excellent report to Government on the subject.

Fifteen years later, in 1836, came the Pali disease, which was very limited in area, and in consequence some doubts have arisen whether it was really plague or not. A disease of a similar nature was at the same time prevalent in Bareilly and other places in the North-West Provinces, and is described in Dr. Guthrie's report to Government. This brings us to the present epidemic which has already lasted more than 3½ years, and, if any reliance can be placed on a comparison with the duration of the older outbreaks, it is not likely to cease for another four or five years.

In reviewing this series of well-authenticated epidemics of plague, it is a striking fact that they should have prevailed in nearly the same districts, and that they have recurred at longer or shorter intervals for several centuries. This is surely as clear an instance of endemicity as can be brought forward. Though there are no records to support the view, it may be supposed that there were persons or animals in whom the specific bacillus contrived to exist, while its virulent properties remained sufficiently in abeyance to allow of the continuance of life in its host, and that, in obedience to the periodic law, which seems to govern all nature, epidemics included, after the lapse of 20, 40, 60 or 80 years, it has again and again assumed a virulence, which rendered its presence incompatible with the life of its host.

If this is considered too problematical, the following alternatives remain:—

1. That the bacillus vegetates somewhere outside the bodies of men or animals, or lies dormant as an encapsuled spore till roused to activity by a fitting change in its environment. The fact that all the working bacteriologists deny that the bacillus is capable of sporulation, and that it cannot become encapsuled, is easily got over by the supposition that it is easily got over by

assuming that it is now passing through its active, vegetative stage, during which it does not sporulate. When this stage is drawing to a close, perhaps after lasting eight years, then only will resting spores be formed.

2. That the bacillus was freshly imported on each occasion from its real endemic focus, an assumption which does not explain the life history of the bacillus, but merely shifts the scene of action.

It is chiefly on account of its comparative novelty to Europeans that the current epidemic has attracted so much notice, and not because it is new to India, or because the type has changed greatly since the last. Europe has known nothing of the plague since it was overrun in the seventeenth century; and to most medical men the name conveyed but little, until this opportunity was afforded of studying an ancient pestilence with the aid of methods, which never before have been available. The estimated plague death roll in the Bombay Presidency has not yet (April 1900) quite reached 310,000, which is considerably below the limit reached by some epidemics of cholera. During 1898 alone 302,000 persons died from "fevers" in the Bombay Presidency, but malaria and cholera pass unnoticed, because the one kills daily and every year, and the other yearly, though with periodical waves of intensity. Long familiarity, the absence of any very stringent or disturbing Government regulations, and the associations of religious notions with their attack, make them almost acceptable to the mass of the people. (Cholera is due to the freaks of a devil, and small-pox to the visitation of a goddess to an honored mortal.) People are gradually coming to hold a similar view of plague, and are ceasing to care so much about their risks from it, though they retain their rooted objections to any interference with their customs in the way of preventive measures. Possibly after plague has existed for seven or eight years it will be as carelessly observed as cholera. Exception has

made by science of recent years. Nevertheless, the mortality, except in a few small places, where for various reasons the usual measures could not be adopted, does not bear comparison with the appalling devastation accredited to plague in the past. It is only fair, therefore, to assume that the fight has been comparatively successful. It is forgotten also that there is a very good reason why diseases should now spread over larger tracts of country now than they did formerly, both in India and out of it. Railways in India, though comparatively few in number, afford a rapid means of transit from district to district; more and more natives are travelling about every year intent on trade, or to perform religious ceremonies or pilgrimages, and races and tribes are becoming more and more commingled and have freer intercourse with each other, though they may not intermarry. It is along the great lines of communication by sea and land that the plague has travelled; and it would almost certainly have spread both faster and further but for precautionary measures. It is becoming clearer, the longer the epidemic prevails, that quarantine has not been successful enough in checking the dissemination of the bacillus to justify its continuance, except in special cases. Though it would doubtless succeed if it could be enforced in such a way that there would be no possibility of the escape of travellers, and if the export and import of infected articles could be absolutely regulated, yet, when huge crowds of ignorant and credulous people have to be dealt with, it becomes impossible to safeguard every loophole, even presuming that the channels of distribution were now defined with such accuracy as to enable the necessary instructions to be issued—a presumption which falls far short of realisation.

Inoculation by M. Haffkine's method, which statistics have proved to be of the utmost value, though not a perfect guarantee of immunity, combined with the isolation of the sick and segregation of the contacts, as far as practicable, and with the surveillance of all arrivals from an infected area for a period of 10 days, now form the lines on which the system of control is conducted. Whether these measures would be a complete success in a civilised country is a matter of opinion; in India, at least, many sick escape isolation, contacts evade segregation, and travellers from infected areas cannot be traced with certainty (In Madras City 4.5 per cent.

are untraced), and notwithstanding the utmost care of all the officials concerned, it is a safe prophecy that the system of control will to the end of time fall short of perfection.

One's original views of the course of plague not unnaturally become insensibly modified as time goes on, when the forecasts made by eminent men are found not to have been carried out in their entirety, and one's own expectations have failed to be realised.

A short review of the epidemics as they occurred in some of the larger towns, when compared with the accompanying charts, may not be altogether valueless.

Bombay has never been entirely free from plague since the first case was diagnosed in September 1896; the traffic is too great, the city too crowded and extensive, and the inhabitants too heterogeneous and unruly, to allow the measures adopted to meet with great success. Each of the four epidemics, from which Bombay has suffered since 1896, began to be evident during November or the first week in December, and rose to its maximum in February and the beginning of March, and declined again during May. The second was more intense than the first and the third than the second. The fourth only appears to have been a little worse than the first, but all the authorities in Bombay unite in admitting that the registration was probably even less accurate in this than the three preceding ones. This marked seasonal incidence looks as if it ought to throw some light on the Etiology of the disease; but, though most elaborate charts have been published in several of the numerous plague reports showing the annual variations in temperature, pressure, humidity, mortality, &c., nothing definite can be arrived at, except what is evident to the most unskilled observer, namely, that the plague increased when the cold months came on, and declined when it became hot again. It may be mentioned that Bombay is a damp place where the temperature rarely rises much above 90° F. or falls below 70° F. and that the rainy season is from June to September. Baldwin Latham⁴ has recently advanced another theory, namely, that plague is in some way related to the vapour tension. When the temperature of the air is above the temperature of the ground, the dew point is also above the temperature of the ground. Then condensation takes place, not evaporation. The cessation of plague is coincident

with the cessation of exhalations from the ground. When the exhalations begin again, i.e., when the air temperature is lower than the temperature of the ground, plague begins again. He concludes that plague should always be epidemic at the same time of year in the same place. Further observations must however be made.

The primary epidemic in Bombay was at first most severe in the densely-populated and dirty quarters of the city, spreading gradually to the suburbs and more or less isolated villages as it subsided. The second epidemic followed a similarly radial course. The third occurred after a period of comparative freedom and exceeded the others in its extent and daily mortality, which was possibly in some way dependent on the relaxation of the preventive measures. From all accounts efforts have been still more slackened during the fourth epidemic.

Total mortality in Bombay	1897 ...	58.28 per mille.
	1898 ...	63.23 "
Bombay City population	1891 ...	806,144
Total plague to Sept. 1st, 1899.	Cases ...	50,457
	Deaths ...	43,636
Case mortality	...	86.4 %
Total actual mortality for three normal years before plague	...	80,541
	...	169,550

The difference between the total actual mortality for three years previous to the plague and the total actual mortality for the three plague years should indicate approximately the total number of deaths to be attributed to plague during those three years. This number is 79,009, but the number of reported plague deaths during the period was only 43,636. Deducting the plague deaths, there remain, therefore, 35,000 deaths in excess of the usual figures to be accounted for during the three plague years. Relapsing fever and small-pox had their share, but by far the majority of the above deaths must be considered to have been unreported and undiagnosed plague.

From September 1896 to April 1900 there have been 384,609 cases and 301,872 plague deaths reported in the Bombay epidemic. This gives a case mortality of 78.4 per cent.

Poona was infected from Bombay late in December 1896. The course of the disease has been most irregular (*vide* chart) both as to season and intensity. It followed the example of Bombay in so far as each outburst was severer than the preceding ones, but up to date it has escaped the fourth. The first reached its height in March 1897, the second in November 1897, then followed a period of a whole year, during the greater part of which there were no indigenous cases at all but only imported ones, and the third reached its height in July 1899. This last was the most remarkable of all. The total population of Poona before the plague was about 120,000. The first two epidemics reduced this by some 8,000 persons and a large number had left the city. Yet for two weeks during the third epidemic people died at the rate of 200 a day, which is equivalent to over 663 per 1,000 per annum. Never for a single day did Bombay, Karachi, or Bangalore approach this rate by 200 per mille.

The prompt and vigorous measures adopted in Poona during the first epidemic were probably not without effect, and in about 2½ months the city was practically free. Then came the disturbances, and murders of officers, that created so much discussion, and the precautions were relaxed. Within a very short time the disease re-gained its ground, and all the work had to be done over again. The hiatus of one year that succeeded the disappearance of the second epidemic does not admit of satisfactory explanation. Karachi was more fortunate. The first outbreak was severe while it lasted, but, aided by the naturally-isolated position of the town and by the existence of desert country all around, the strict segregation enforced, without doubt, materially shortened its duration. The inland extension was confined to towns on the only railway line, but here too preventive measures were successful in absolutely exterminating the disease, though doubtless the extreme heat of Upper Sind was highly unfavourable to its continuance. The climate of Karachi closely resembles that of Bombay with regard to temperature and humidity, but the rainfall is much less, though there seems to be no reason for supposing that the bacillus failed to permanently establish itself there on that account only. After between four and five months of complete immunity a second outbreak occurred at Kotri, which is supposed to have originated in the arrival of two persons from the Jullunder District of the Punjab

which had by then become infected. These persons did not themselves get plague, but certain numbers of their family did, and it was supposed that the bacillus was re-imported in their clothes.

There are two other possible hypotheses—(a) that infected clothing had been imported up the Indus in boats from Cutch; (b) that the bacillus had been in Kotri all the while in a dormant state, and suddenly resumed its activity.

Immediate precautions were taken with the result that no other place became infected. About two months after the last case in Kotri a new case was discovered in Karachi, which was quickly followed by others in different quarters of the town; and in a few weeks, after having had absolute immunity for a space of seven months, a much more virulent outbreak than the first was raging in the town. By means of strict segregation—a system of passes and railway examination—no extension of the disease occurred beyond the first railway detention camp, which was about 10 miles from Karachi and situated in a narrow neck between some rough hills and the sea. This neck, about 15 miles wide, was guarded by a cordon of police. Over 100 cases of plague reached this camp from Karachi, and apparently not a single one escaped into Upper Sind, judging by the fact that there was no epidemic in Sind at that time except in Karachi. There were two theories put forward to account for the second outbreak in Karachi—(a) the rejuvenation of the bacillus which had been vegetating for seven months; (b) the importation of a large quantity of cotton waste from Bombay which was at once made up into thin mattresses called resais. These commanded a ready sale owing to the destruction of so many of the same kind during the previous epidemic. Thus both in the case of Kotri and Karachi we are compelled to believe that the bacilli were vegetating somewhere for periods of four and seven months respectively, until some change in their environment allowed them to re-assume activity and virulence, if credence is not put in the re-importation theory, which in both instances is merely a speculation to account for known facts, as no scientific proof beyond coincidence is adducible in its behalf. The consensus of opinion now in India certainly concludes that infected articles of apparel, or merchandise, are not the usual means of starting an outbreak in a new locality, but that transmission by human agency or by rats is the more

likely; and knowing as little as we do of the causes which contribute to the rise and fall in virulence of epidemic diseases it is unwise to be dogmatic or accept any one apparently plausible explanation to the exclusion of all others. No means have hitherto been devised by human beings to control the transport of the bacillus by rodents, and, though it is unlikely that it would ever naturally be carried many miles across country by direct infection from rodent to rodent, yet, unless very strict censorship is exercised over the exports from an infected town, there is every reason to suppose that it can be conveyed to a fresh centre by this agency and nullify all the care and trouble taken in preventing human contacts from having free access to uninfected districts. In the second Karachi epidemic nearly the whole city was camped out on an extensive maidan, whereby the duration of the outbreak was probably shortened. During the succeeding six months the town was never declared free from plague, one or two cases a week kept cropping up, and every body fully expected a third outbreak, so much so that the natives who had been camped out preferred to leave their huts and everything standing, considering that they would shortly be again required. Many, indeed, never re-occupied their town houses at all, but came into their daily business from the camps by tram, a temporary line having been laid down for their convenience. When the expected recrudescence came, therefore, the city was evacuated in a few days, and, though the disease prevailed to a certain extent in the camps, it was promptly dealt with and never got entirely out of hand. In October 1899 Bangalore was beginning to suffer from an outbreak, which, with the exception of the third at Poona, is the severest up to date. After most of the surrounding country had been freely sown with the seeds of the disease it declined in the usual way in about three or four months. Since then though neither the native city nor the military station has ever been entirely free, there has been no second epidemic outburst up to date, a period of 21 months. No adequate explanation can be offered of this peculiarity, and sooner or later when the necessary conditions prevail, if the example set by all the other towns is followed, a second outbreak, even more severe in character than the first, will occur. The first has at least thoroughly disproved an idea which prevailed during 1897 that plague could not really flourish south of the 15th parallel.

There was a widespread belief, though it has no further foundation than a comparison of the distribution of the epidemics of former years, that plague cannot thrive on the east coast of India. This was confirmed by the failure of the disease to spread in Calcutta in 1898, when about 150 cases occurred, followed by a complete intermission. One well qualified to give an opinion stated to me that this was not in any way the direct consequence of the measures employed for its suppression; it appeared simply to die out. Further discussion of the aspects of plague in Calcutta are at present unprofitable, considering the confusion of opinions of which is still rife there. Some deny that the plague is identical with the Bombay disease, others that it is plague at all. A few assert that if the disease now prevailing is plague, then plague has prevailed in Calcutta ever since they began to practise there. Even the bacteriologists are not agreed. In Madras again there are found those who believe that plague can never find a footing in their town because it is on the east coast. It is true that there have been 22 imported cases discovered up to date, and that no indigenous ones have followed in their train. But this, however, I can personally assert was practically impossible owing to the immediate and thorough action taken. That in former times plague did not spread to the east coast was quite a fortuitous circumstance, and in a large measure determined by the absence of communications, I am convinced; and it would be a most imprudent policy to rely on ancient history and the Calcutta episode as a surety that the bacillus of plague finds the environment of the east coast unsuitable for its development. On the contrary, my personal experience together with a survey of the course of the disease during the past 34 years leads me to the belief, almost amounting to a conviction, that Madras will in its turn suffer, and that Burmah too will not escape.

The beginning in Karachi.—Karachi was undoubtedly infected with plague by passengers arriving from Bombay by sea, direct communication between the two places by land being impossible except by a most circuitous railway journey, which no one in his senses would ever think of undertaking, unless extremely hurried by avoiding detention. Perhaps, in a big mercantile port, it could have been impracticable to have prevented the importation of the disease without the entire dislocation of the trade, which, of course,

would have been vehemently opposed by the Chamber of Commerce and all the merchants of a city jealous of, and longing to obtain an advantage over, Bombay. After the plague had got a good hold in Bombay, Karachi began to fear for herself, and sea quarantine was instituted during November 1896 for two or three weeks. No cases having been detected by this means, it was allowed to lapse, and free communication again followed.

On December 10th the first case was diagnosed in the native town, and the total weekly mortality in that quarter during the four weeks of December read as follows: 21, 24, 45, 106. At this time nothing could be done, as the Epidemic Diseases Act had not yet been framed, and local authorities had consequently no powers to segregate, remove sick, or even to enter houses without the permission of the inmates.

The prevailing native theory of the origin of plague was that it was due to the influence of the Queen-Empress. Deeply offended by the desecration of her statue in Bombay, she had sworn that the insult should be avenged by the sacrifice of 100,000 lives, and had either provoked the pestilence by occult means or had instructed her emissaries to poison off the required number of natives and to send her their hearts in token of the accomplishment of her will. Hence arose a deeply-rooted prejudice against hospitals and *post-mortems*.

The difficulties to be contended with were greatly enhanced by the rumour, started by some rascal in Bombay, and quickly conveyed to Karachi, that Government servants had secret orders to poison off every one stricken with plague in order to prevent the disease from spreading. This at once received confirmation in the minds of the natives in Karachi by the death of most of the patients who had been visited by Europeans. Some of the local medical officers considered a dry tongue to be a valuable diagnostic sign in the early days, and were therefore always wanting to touch tongues—an action which was misinterpreted by the ever-suspicious native.

Government officers, they supposed, carried some subtle poison about with them, and having requested the sick man to show his tongue, either touched it with a poisoned finger or squirted the

poison into his open mouth. For the same reason the civil hospital and dispensaries, to which some hundreds of natives formerly had daily resorted for treatment, were now almost deserted.

Coupled with the usual dislike of interference, which is inherent in the native, caste rules, the purdah system and religious scruples generally, this mistaken idea made it almost impossible to deal with the epidemic in any way without special legislation. Acting on the notion that the disease could not flourish inland and that heat was inimical to it, no effort to stop an exodus was made, either in Bombay or Karachi.

Indeed, in the latter town large sums were distributed by the Municipality to the poorer inhabitants of the most densely-populated quarters to enable them to pay their railway fares and go up-country, or else to start a small colony in the jungle. This is how the disease spread into Upper Sind. Not till the middle of March 1897 was the Epidemic Diseases Act passed and put into force by Government, and segregation and removal to hospital made legal. Before that time I used to go about seeing sick in their own houses and trying to gradually disprove the poison theory. After a few venturesome or intimidated persons found that they did not necessarily die after a proper examination, or after drinking my medicine, confidence began to be restored and freer access to the sick became possible. Still, there were always plenty of ignorant ones, chiefly women, who having once got the notion of poison into their heads were unable to dismiss it, not to mention those who wilfully spread false stories for their own ends or to foment disturbances. As my dealings were chiefly with Musalmans, the theory of *kismet* had also to be combated. "If God wills it my brother will die, if not he will recover. In either case what is the use of medicine and why should the *Sahib* trouble himself." Annexed is an extract from the *Times of India* showing the state of native feeling as late as 11th April 1898:

Resolved by the *hakims of Lahore*—(1) "In the opinion of this meeting the bubonic plague is not a contagious disease. It originates from poisoned air, and this poison is created in the air on account of atmospherical germs and the excess terrestrial humidities. This poisoned air affects and poisons the blood of such human bodies only as have a predisposition for its action. (2) This meeting is of opinion that the rules embodied in the Resolution of Government (regarding isolation and disinfection of clothes and houses) are unnecessary under the principles of Unani Medical Science."

The press of executive work, other than strictly medical, prevented much note-taking and the opportunities for scientific observation were, for that and other reasons, but few. The following medical details are based on about 1,000 cases seen and 350 personally treated.

Type of disease.—The usual division into two types, named Pestis Major and Pestis Minor, did not hold good in Karachi. Whatever may have occurred before the first diagnosis of plague was made it is pretty certain that during the height and decline, and subsequently to the cessation of the epidemic, there were no persons attacked with glandular swellings accompanied by little or no fever and no severe general symptoms. Pestis Minor, therefore, was non-existent during the first epidemic in Karachi. As in every acute specific disease there are gradations from the fatal to the very mild case, so in plague are very mild cases occasionally seen, but that is no reason why they should be dignified by a distinct title. The disease is identical in both cases, the mild attack being merely due to either the resistance of the individual or to lessened virulence of the microbe.

Prodromal stage.—There was no prodromal stage observed in Karachi, such as the newspapers made great capital of, talking of plague patients being taken up as drunk and disorderly by the police, owing to dizziness preceding the attack; but the persons, who were occasionally apprehended wandering about in the streets in a state of delirium, were in an advanced stage of the disease. I have, however, learnt from other medical officers that they have taken persons out of trains, on the strength of a shuffling gait and some inco-ordination of speech, who a few hours later developed fever and buboes.

The attack.—The first thing noticed was generally a rigor or a feeling of general *malaise*. The temperature quickly rose to 103° F. or higher, and the patient was often prostrated. He sometimes, however, retained sufficient strength and resolution to walk to a hospital or elsewhere, but the exertion was not unfrequently fatal.

Lately in Bombay a witness in a criminal case was noticed by the Magistrate to be scarcely able to stand in the box and to

be breathing laboriously. Suspecting plague, an ambulance was fetched and the man placed in it. He was dead before he had been taken 50 yards from the Court.

Varieties.—There were three well-marked varieties of the disease, two of which at any rate have been distinguished before. Writers describing the effects of plague in certain French towns, notably an Abbé of Avignon, make a difference between the type primarily affecting the lungs and the type accompanied by buboes. Dr. Whyte also distinguishes the spitting form in 1820 in Guzerat. Old English accounts of people falling dead within a few hours of infection may be taken to indicate the septicæmic or toxic type. The three varieties may be named

1. The bubonic, the commonest.
2. The pneumonic.
3. The toxic.

These have been still further sub-divided by different observers in India, but no particular purpose is served by introducing unnecessary complications. An intestinal type with profuse diarrhoea and no buboes has been mentioned as prevailing in the south of the Bombay Presidency, but nothing was seen of it in Sind.

Bubonic form.—All kinds of cases were seen under the heading of simple bubonic plague from the fatal to the mildest. In an ordinary case the patient had a rigor, the temperature quickly rising to 104° F. or 105° F. Occasionally he vomited once or twice or passed a motion, but constipation was the rule at the beginning. In a few hours there was tenderness over a gland somewhere, followed by the formation of a bubo at that spot in the course of 48 hours. More rarely the bubo did not appear for 4-6 days, the patient having irregular fever meanwhile. The tongue was coated with white fur on the dorsum, edges clean and red. In children the tongue seemed to me typical; thick white fur on the dorsum with red papillæ showing through; the central line, tip and edges, clean and red. In adults the papillæ were not always so evident, and the tongue was usually more thickly coated. I never saw the blue or black tongue one hears of, and it only got dry and brown in a few bad cases of pneumonia. The skin was hot and

dry, sweating was not the rule till three or four days had passed. The pulse, full and rapid at the start, quickly became feeble, rapid, and of low tension, and not infrequently dicrotic.

In old men with hard arteries and a bubo, the disease often took the following course:—

First day—High temperature, strong and full pulse, pain in bubo.

Second day— Do. do.

Third day—Temperature lower, pulse fair, seemed better.

Fourth day—Temperature lower, pulse much weaker.

Fifth day—Cardiac failure and death.

In most cases there was delirium at some period and not infrequently profound coma, sometimes lasting for 48 hours. While comatose there was retention of urine and faeces. In other cases insomnia or diarrhoea were prominent features.

Whether the bubo would suppurate or not was generally evident by the 6th or 7th day; and, if the patient were going to recover, the temperature would then rapidly fall and the pulse regain its strength.

Death usually took place about 3rd-5th day, rarely later than the 7th. Sometimes a secondary pneumonia would come on about the 6th or 7th day and rapidly kill a patient who seemed to be doing well. Other symptoms included a severe frontal headache; injection of conjunctivæ, which rarely went on to a severe conjunctivitis; slight jaundice and enlargement of liver and spleen with tenderness over the latter; intense pains in the lumbar region and limbs not coming on till the patient had passed the severer stage; paralysis of bladder, palate, phonatory muscles; a muttering delirium lasting several days; nausea and vomiting [rarely bilious; and very rarely suppression of urine.

The urine was scanty and full of urates, but in the few cases examined no albumen or sugar was found. No external evidence of nephritis. Sometimes a papular rash appeared all over the trunk and limbs fading in a few days. No hæmorrhages were noticed, with the exception of profuse hæmoptysis in some pneumonic cases,

thus marking a difference between this particular outbreak in Karachi and the older accounts, and also reports received from other parts of the Presidency. The one or two instances of slight epistaxis and mæna were probably accidental.

Necroses of skin and subcutaneous tissues—badly named carbuncles—were sometimes seen.

Primary Pneumonia.—In the pneumonic form both lungs were generally involved. The onset was indicated by a rigor, rise of temperature, and often somewhat severe pain over a limited area of one lung, where a pleuritic rub might be heard. The pulse rapidly weakened, respirations became shallow with apparent dyspnoea and the patient sometimes died, without the development of further symptoms, in 48 hours. In other cases the process spread till the whole lower lobe of the first infected lung became involved and then the other lung followed suit. The physical signs are not the same as in ordinary pneumonia. The dulness though evident was not so complete and typical, tubular breathing was seldom heard. There were abundant rales over the whole affected area, rather coarse in nature. Rhonchi were also heard. The fine crepitation of ordinary pneumonia was not audible. The most striking feature of these cases was the bad condition of the patient when compared with the physical signs presented. The latter were rarely as marked as one would expect, and even early in the history of a case a patient would be in a state of extreme prostration with nothing to be found but a tiny patch of pneumonia in one lung. If the patient recovered, these physical signs cleared up with astounding rapidity after the fall of the temperature about the 7th or 8th day, and the patient felt practically well though somewhat weak. The sputa were bronchitic in appearance and contained numerous bacteria and diplococci. The specific bacillus could only be identified by cultivation. Cough did not trouble the patient much, but insomnia, pain in the chest, delirium and diarrhoea had all to be combated in different cases.

In certain instances the sputa were streaked with blood or rusty coloured, and, although more pneumonic in appearance, never had the characteristic viscosity. The plague bacillus was readily seen in cover-glass preparations of this coloured sputum. Some of these patients died and others recovered.

Another variety was invariably fatal. Within 24 hours of the attack the patient began to spit what looked like pure frothy blood. It came up easily and with a slight cough; about a saucerful being expectorated in a couple of hours. After this the flow was more scanty. Breathing became more and more difficult, both lungs were œdematous throughout, and death occurred in 48-72 hours. The bloodlike sputum contained pure cultivations of the plague bacillus mixed with red corpuscles, and it can be imagined how easily other persons can acquire plague from close contact with such a case. The German Plague Commission found that the plague bacillus retained its activity for 10 days in some of this pneumonic sputum kept in a vessel, but that it died after 16 days. The temperature kept up to 104° F. or 105° F. and the patients remained sensible till just before the end. This is the most awful form of plague; one can never forget the aspect of the patient—an expression of wild despair. He sits up breathing rapidly and in gasps, his eyes rolling about and shifting his head from side to side; speechless, yet capable of vigorous muscular efforts in response to a question, and recklessly spitting in any direction. His minutes are numbered, and he obviously knows it.

Secondary Pneumonia.—Secondary pneumonia, *i. e.*, pneumonia occurring about the 7th day in an ordinary bubonic case, was not so common, but was none the less fatal. Like the primary pneumonia it may be slight, severe or hæmorrhagic; and there was nothing to distinguish its course from that.

Toxic Plague.—The least frequently observed form of plague was one in which the patients died in 8-20 hours without bubo or pneumonia having had time to develop. It was apparently due to the absorption of an overwhelming dose of the poison, or else the sufferers happened to be extraordinarily susceptible to the usual amount. It was chiefly seen amongst old people aged 60-80 and young adults of the Sweeper and Sidi castes. The attack was marked by a rigor followed by high temperature, orthopnea, weak pulse and extreme fear of death. No other complaints were made, but rales appeared at the bases of both lungs during the last few hours from the failure of the circulation. At first the senses were very alert, but the pulse disappeared and a semi-comatose state preceded death.

The Bubo.—The bubo itself may appear in any lymphatic gland or set of glands in the body. At first there was no swelling or enlargement to be made out, but only tenderness on pressure and shooting pains. Within a few hours one gland was usually distinctly enlarged and could be distinguished from the rest. The pain was often so severe that sleep was impossible and the limb was kept at rest to ease the gland. In other cases, curiously enough, palpation merely gave uneasiness, and pain was scarcely complained of.

In a few hours more, the original swollen gland became indistinguishable owing to the infiltration of surrounding tissues, and a lump, the size of half an egg, was visible.

No further change may occur in a very mild case, and in the course of three or four weeks the bubo will be entirely absorbed. Otherwise, the swelling goes on till the size of a large orange is reached, the skin over it being red and tense with œdema all round.

Sometimes, instead of taking three or four days to reach its height, a huge bubo was developed in a few hours—a less satisfactory condition than the usual one. Enlargement of several other sets of glands may follow the first, a second bubo being quite common. In one of my cases there were five, one in each groin, one on each side of neck, and one in the right axilla. Three of these suppurated.

Bubo in the groin.—Of the glands in the groin either the femoral or the inguinal sets may be enlarged separately, or together form a single bubo. If it was the inguinal set, then in most cases the iliac glands in the abdomen were also affected and a huge mass could be felt reaching half way to the umbilicus. If the femoral set alone was inflamed, the abdominal lump did not appear. When the bubo was left alone and resolution did not occur, the mass softened about the 6th-8th day and after a while opened externally by two or three small holes; an immense time elapsing before the patient was fit to go about again on account of the difficulty the sloughs found in getting out without assistance. If opened up after spontaneous rupture and the sloughs pulled out, a cavity was revealed corresponding in size with the original bubo. After the removal of the sloughs the cavity was clean and readily granulated up, if the habits of the patient were such as to allow it.

As a rule, all the subcutaneous tissues sloughed together with glands, nerves, and vessels, leaving the fascia covering the muscles bare. The worst of these cavities holding from $\frac{1}{2}$ –1 pint of water completely filled up in 6–8 weeks unless the patient was an opium eater or debilitated by other disease. Then they sometimes died with a freely suppurating wound and no healthy granulations.

Occasionally the bubo got larger and larger and an oedematous swelling rapidly extended over the surrounding area. This in my experience was invariably fatal. I saw a small axillary bubo suddenly increase in size; then the whole side from axilla to iliac crest became thickened with oedema, so tense that it would hardly pit and very painful. Incision was useless.

In another case there was a bubo in the groin; the solid oedema appeared and spread to the umbilicus and half way down the thigh before death occurred. Fortunately, this was not a frequent complication.

No buboes were seen in the popliteal space.

Bubo in the axilla.—The axillary bubo generally began and ended in the set of glands beneath the edge of pectoralis major. It was rarely as severe as a bad groin bubo, and the glands towards the apex of the axillary cavity were not often involved. Only once did I see the whole of the loose tissues of the axilla slough. Once a lump appeared in a woman's breast at the same time as an axillary bubo. The two masses were connected by a thickened chain of lymphatics, and though the axillary bubo did not suppurate, I had to incise the breast and extract a large slough. It healed in a week. The supracondylar gland was not infrequently enlarged, but was generally accompanied by buboes elsewhere. Only once did I see it suppurate.

The cervical bubo.—The neck is the most dangerous place for a bubo to be formed on account of the proximity of important structures and the freedom of lymphatic communication in the head and neck. It may begin in any of the sets of glands, submaxillary, submental, superficial cervical, anterior and posterior, or even in the substance of the parotid. The deep sets did not seem to be primarily involved. A single or even a double cervical bubo may follow the usual course, either resolution or suppuration. But very

often there was a good deal of oedema, which, if limited to one side, sometimes passed away harmlessly; but if it spread to the other side, put the patient into the utmost danger. The swelling would begin on one side, and, spreading beneath the chin, reach the ear on the other side. Then the tissues got infiltrated with serum till the collar was completed at the back of the neck, descending to the sternum and clavicles in the front. By this time the patient could not move his head, swallow or speak, and respiration was exceedingly laboured. The whole neck and parotid region was one tense mass and the condition of the patient hopeless.

Death, if cardiac failure did not intervene, was due to asphyxia from oedema of the larynx or pressure on the trachea, and in this way a cervical bubo might kill directly without reference to the toxins in the system. The deep cervical glands were probably also involved in these cases.

Between the simple bubo on one side of the neck and this extreme swelling, there was, of course, seen a number of gradations. Free incision would relieve the tension of the swelling for the time being considerably, but had no influence in controlling its spread.

One woman of about 60 survived who had not been able to swallow for 48 hours. She was allowed to lie in a semi-conscious state during that time on her back, and when I was at length permitted to take her in hand, there was a slough 3 inches in diameter on her occiput. I opened two huge sloughing abscesses in her neck and she eventually recovered after a illness of 2½ months, passing through an attack of facial erysipelas, but had rather a stiff neck.

Internal buboes.—Certain patients complained of severe abdominal pain in various situations, but kept their muscles so rigid, that nothing could be felt. Cardiac depression forbade the employment of an anaesthetic and no autopsy was possible in the one or two that died. It is a question whether there was or was not a bubo of the abdominal glands in these cases. Such a bubo could only be diagnosed when there was swelling of the iliac glands and bulging above Paupart's ligament, which might or might not be accompanied by a bubo in the groin or elsewhere.

Temperature.—No uniformity or typical tracing could be distinguished in the temperature charts of plague.

A sudden rise to 103° F. or 105° F. was almost invariable, then irregular intermissions, remissions, or steady maintenance of a high-level of 105° F. for several days, were all observed. The charts of tertian ague, typhoid fever and acute pneumonia were all represented.

A remission about the 5th day and subsequent rise sometimes indicated the onset of secondary pneumomonia.

The fever terminated by both crisis and lysis, more often the former.

The highest temperature I observed was 106.8° F., the highest I heard of 108.3° F. The temperature was invariably subnormal for several days after a crisis. Incision of the bubo was often followed by a permanent fall in the temperature.

Theory of the disease.—Exactly how the poison enters the body is not yet definitely decided. Two ways are obvious, namely, Inoculation and Inhalation.

No one who has seen a bad case of pneumonic plague and examined the sputum under a microscope will doubt the possibility of the latter mode and both clinical experience and laboratory experiments confirm it. In Bombay monkeys were infected by being made to inhale a spray containing virulent bacilli by Simond and others. Whether primary pneumonic plague can result from a cutaneous inoculation is not yet proved, but a case to be quoted later seems to point in that direction. Bubonic cases at any rate frequently follow a cutaneous inoculation, which is easily proved in the laboratory, and has unfortunately been decided on the persons of several medical officers and assistants while performing *post-mortems*.

Early in the epidemic it used to be asserted that the position of the bubo indicated with certainty the point of inoculation. However fascinating, and even probable, this view may be, it has not been entirely borne out by experience. The frequency of femoral groin buboes was accounted for by inoculation of the naked and unprotected feet of most natives, and the inguinal bubo was connected with their habit of squatting and thereby inoculating an abraded buttock. The loin cloth of many natives also conceals a circle of itch. Next comes the axillary bubo, consequent on a

scratch of the hands or arms. Finally, the cervical bubo was said to show that the point of entrance had been on the head or neck or possibly the tonsil. The German Plague Commission remarked that children who naturally stuff everything they can into their mouths suffered from submaxillary buboes. The simultaneous appearance of three or more buboes in different situations can only be explained on this hypothesis by the assumption that the patient had numerous abrasions, and by literally dabbling in virus had infected them all at the same time. Wilm⁸ stated that of 300 English soldiers who were on plague duty at Hong Kong and worked with protected feet and legs, the 10 who had plague had mostly buboes in the groin. Simond, working in Bombay, said that coolies who handled dead rats as often got groin buboes as axillary.

Besides direct infection of an abrasion other agencies have to be considered, such as insects. As yet however, though many experiments have been made, nothing definite from a practical point of view has been arrived at. In India everybody looks at the swarms of flies that occasionally present themselves with suspicion for many reasons, and it was only natural to suppose that they might have something to do with plague. Nuttall found that *flies*⁹ fed with plague organs could carry about virulent bacilli for several days, but they ultimately died. Still they would undoubtedly be able to remove bacilli from a pneumonic expectoration to another spot. *Bugs and fleas* next claimed attention, and attempts were made to localise the point of infection from a bite. The Austrian Plague Commission reported that they never saw a case that must have been caused by an insect bite, nor a recognisable entrance place in the skin. Simond¹⁰ in October, 1898 considered that fleas and other vermin were frequently the means of communicating the disease, and that a dead rat might be handled with impunity after the fleas had deserted it, but he adduced no evidence in support of his assertions. Nuttall¹¹ on the other hand has allowed bugs and fleas to bite infected animals and then healthy ones afterwards. No case of transference occurred. He also found that both bugs and fleas digest pathogenic organisms, and though their dejecta may contain virulent microbes for 24 hours, it is not so later. A bug, however, may be crushed on the skin of a healthy man and, if it contains any virulent organisms, these may be inoculated by scratching.

The German Plague Commission doubted whether insects can convey the disease, and are emphatic that the mosquito cannot. Nevertheless Sticker, one of their members, considered that insects (ants, pediculi and acari) were the chief agents in the transference of infection from the sick to the healthy, except in the case of pneumonic plague. Hankin found that *ants* would carry about bits of plague organs, but did not themselves die. It is evident then that further experiments must be made before the precise rôle that flies and parasitic insects play in a plague epidemic can be determined, but personally, even though the possibility of the transportation of the virus in or on insects be granted, I do not consider that it is a very important point as far as preventive measures are concerned.

As regards infection *via* mucous membranes without abrasions, the evidence of different observers is again conflicting.

Simond¹² obtained negative results by feeding rats, mice and monkeys with plague cultures, but Yersin, Calmette, Borrel and Hankin all succeeded.

The German Plague Commission reported that rats are easily infected by touching the conjunctival or nasal mucous membrane with a pure culture of the plague bacillus. They also acquired the disease by eating the bodies of other rats which had died of it.

To infect monkeys however by the intestinal canal, large quantities of cultures must be used. They died in a few days with intestinal hæmorrhages.

Batzaroff¹³ also found that without excoriation a plague culture placed on the nasal mucus membrane of rats produced pneumonia. The conjunctival, buccal, intestinal, rectal and vaginal mucous membranes also absorbed infection.

Lustig informed the Municipal Commissioner of Bombay that he had communicated plague to rats by feeding and that he had found the bacillus in the fæces and also in the urine. It must be concluded, therefore, that plague can be acquired by man by cutaneous inoculation of an abraded surface, by inhalation and possibly by absorption from mucous membranes. Whether human beings suffer by reason of infected food is not known, the German Plague Commission at least did not meet with a case of intestinal plague.

Having somehow obtained entrance into the body the bacillus has to overcome the local resistance before it can do much harm. If inoculated into a limb, it has to pass through the nearest set of lymphatic glands, where acute inflammation is set up by the struggle and the tissues involved usually perish. Whether in mild cases the bacillus ever gets further than the primary bubo is not known, nor whether it is to be found in the spleen and distant lymphatic glands. At any rate if any bacilli at all reach the general blood current they are destroyed before general infection can occur. In a fatal case large numbers enter the blood, reach the spleen, are cultivated there, and, overpowering all opposition, are deposited by the blood all over the body starting other foci of growth. The same thing probably happens in pneumonic plague but the lungs do not afford so good a protection to the system as the lymphatic glands and further extension is therefore more frequent.

The disease is a septicæmia. If the tissues can prevent the bacilli from reaching the blood in large numbers and can confine them locally, the patient recovers. But if quantities do reach the blood and multiply all over the body the individual is quickly poisoned by the toxins liberated. There is no local reaction at the seat of inoculation unless the so called carbuncles are a manifestation of it. They are certainly generally distal to a bubo. I have seen them on the foot, calf, arm, abdomen and nose. The skin becomes red, then blackens and a swollen cedematous ring forms round it. The necrotic area rapidly increases if not treated. Skin lesions of this nature seem to have been more common in the earlier epidemics, according to the records, than they are in the present one. The plague bacillus has been seen in these necroses, but many others are present as well, and it is not certain which is the prime cause. A few cases were seen which began with severe tonsillitis, tremendous swelling, some sloughing, and of course inability to swallow, but there were no means of determining whether this was due to a local infection of the tonsils by the plague bacillus or by ordinary cocci.

No adequate explanation can be put forward to account for the supervention of secondary pneumonia in an ordinary case. It may be supposed either that a new dose of virus has been inhaled into the lungs before any immunity has been established by the

first, or that a mass of bacilli have escaped from the bubo and gained access to the lungs through the venous system. The so-called flattening of the bubo, which has been quoted as a certain sign of death, is partly due to the diffusion of the effused fluid on the failure of the circulation. I have never known a bubo to totally disappear in a day or two though others say they have, even when the patient did not die. The cessation of local resistance, when the bacillus has overcome the organism would also perhaps cause a diminution in size. The diminution before death is by no means an invariable or even a common symptom.

The connection between rats and the plague remains to be considered. Though perhaps more has been written and talked on this point by both doctors and laymen than on any other feature of the epidemic, yet still the fundamental idea that rats are a necessary factor in the spread of plague is under dispute.

Rats, mice, bandicoots, squirrels and monkeys are all readily susceptible while dogs, cats, pigs and birds are either absolutely refractory, or so much so as to enable them to be neglected.

Rats may or may not become infected before men in a new focus. At any rate, their death in numbers has often preceded an outbreak in a new town; while in others it is the men who have begun to die off the first. It is probably a matter of chance whether an infected man or an infected rat happens to be the first to arrive in a town. If the former then probably the epidemic affects the men first and the rodents only later. If the latter, then many rodents will die off before human beings begin to be attacked. The importance of the rôle of the rats probably differs in different Indian towns, and from evidence given before the British Plague Commission, plague may be very severe in a town without a concurrent increased rodent mortality.

The disease can easily spread among rats from the habit they have of devouring their dead companions. Many human corpses also were found in Bombay to have been gnawed by rats. The Plague Research Committee found evidence¹⁴ that infection had been carried by rats to human beings who could not have been contaminated by other means.

In Bombay of 20 coolies who were employed to collect dead rats in a godown, 12 got plague within three days. In short, though it cannot be doubted that plague can exist epidemically in a community where there are no rats, still it is obvious that if the rodent population of a town is also affected the bacillus must be widely disseminated through a town by their means. Infected rats whether living or dead must also be able to carry the bacillus hundreds of miles to fresh foci either by rail or by steamers. Considering the danger run by a community if the plague gets among the rodents it has been thought advisable to destroy as many of the rodents as possible to lessen the available pabulum if the disease is accidentally introduced. With this end in view in Madras City alone,

249,905	...	...	Rats,
61,787	...	...	Bandicoots,
51,399	...	...	Mice,

were destroyed between July 1898 and March 1899, but they appeared to be just as plentiful and easily caught at the end as at the beginning, so unless some other means of extermination can be introduced, such as a microbic disease fatal to rodents and harmless to mankind and domestic animals, there does not seem to be much prospect of successful prophylaxis in this direction. This measure has again been recently highly extolled by Dr. Manson.

Anomalous Cases.—Certain peculiar and unaccountable features occasionally presented themselves during the conduct of a case, such as partial paralysis of the palate for a few days with regurgitation of fluids, and partial paralysis of the œsophagus with great difficulty in swallowing. These were seen when there was no cervical bubo to cause pressure symptoms. Articulate speech was sometimes interfered with owing to some inco-ordination of the phonatory and laryngeal muscles, though the patients were always able to make a loud noise. Recovery was delayed much longer in the phonatory paresis than in the palatal and œsophageal. Retention of urine only occurred during coma. In two or three cases, the eyes went wrong. In one the eye-ball was protruded till the lids had to be divided in order to prevent sloughing. The cause could not be ascertained and the patient died while the pressure

was diminishing. In another the sclerotic bulged out and burst within 24 hours, allowing some vitreous to escape, but the patient recovered and retained useful sight.

Simple corneal ulcerations sometimes occurred which healed as the patient recovered. During convalescence, secondary abscesses were several times seen in the scalp and limbs.

The pains in the limbs, which sometimes gave much trouble after the acute stage has passed, seemed to be due to a real neuritis, though they did not last long enough for any muscular wasting to be evident. Dr. Whyte in his Guzerat Report mentions that lesions of the eyes were not uncommon in that epidemic.

Concurrence of Diseases.—One patient took plague while covered with discrete small pox papules and developed a groin bubo. His temperature ran up to 105.5 F. and he died on the second day of the disease. I came across about 50 cases of small pox while looking for plague. Most of them were segregated and no other was attacked by plague. One patient got erysipelas while two suppurating buboes in the neck were being treated. Her features were greatly swollen and unrecognisable for a few days, but she soon recovered. A boy of 10 had tubercle of one lung. He just escaped with his life from plague, but never recovered strength and the tubercle made rapid advances. He had to be discharged from hospital and I lost sight of him, but death probably followed. The German Plague Commission noticed that after plague, tubercle of lungs rapidly becomes worse. Dr. Pearse¹⁵ has tabulated a long list of concurrent diseases from the Bombay Hospitals, showing the influence exerted by plague on their course.

Two of my patients, one a man of 40, the other a boy of 4, after safely passing the acute stage and after their buboes had healed, failed to recover strength and gradually got thinner till they succumbed some two months after the beginning of the attack. They made no complaints and no trace of organic disease could be found during life, nor were *post mortems* permitted, so it remains doubtful whether they died from the effects of plague or not.

Bacteriology.—Though much has been discovered with regard to the bacteriology of plague during the past three years we are still just as much in the dark, or even more so, as we are in respect

of most of the other specific diseases when it comes to dealing with them on a scientific basis on a large scale. Even the bacillus itself is not finally decided on.

Yersin describes an organism which does not stain by Gram's method, Kitasato one that does, and they each consider the microbe they are dealing with to be the specific organism of plague. Kitasato¹⁶ goes so far as to say that further research is necessary before preventive inoculation can be put upon a rational basis, though the organism dealt with in Bombay is one that does not stain by Gram's method. The bacillus seen in both Bombay and Karachi was short and thick, stained easily with aniline dyes, and showed, when not too deeply stained, that the poles of each rod took the colouration more deeply than the centre. This characteristic bi-polar staining gives the microbe the appearance of a diplo-coccus, but cannot be obtained when the cultivation has been artificial on solid media. Preparations from agar cultivations, on the other hand, generally show characteristically elongated forms which are but rarely found in the tissues of man.

The crucial differentiation test of the plague bacillus¹⁷ is the stalactitic growth found, when it is cultivated in fluid media in the dark, by Haffkine. The best mixture is a neutral or faintly alkaline peptone bouillon with a few drops of oil on the surface from which the stalactites can depend.

The bacillus survives the process of freezing but is killed by moist heat at 80 C. in 10 minutes and in 100 C. at 1 minute. It does not long survive desiccation or exposure to sunlight.

When sheltered from heat and direct sunlight the bacillus, if spread on moist articles of clothing, will survive only a few days. Opinions differ considerably, however, on this most important point of survival on articles of clothing, for, while Hankin¹⁸ and the German Plague Commission limit the time to a few days, Gladin extends it two months.

Little or no success has attended the search for the plague bacillus in earth, air or water, and so no definite line of action can yet be formulated for sanitary officers. Gladin states¹⁹ as a result of his experiments that the plague bacillus when cultivated in the presence of other organisms is overcome by almost all of

infection might follow from allowing the bacillus access to the blood vessels; nevertheless recent experience in Glasgow has shown this fear to be unfounded.

Treatment. There is no doubt that drug treatment is somewhat unsatisfactory. Of the numerous vaunted specifics—chiefly native in origin—not one has been proved to be of value, and though it may seem to be illogical to condemn anything one has not tried, no one who has seen the disease in all its virulence could possibly imagine any beneficial influence being exerted by the majority of them. Starting with the idea that the whole organism, especially the central nervous system, is being poisoned by virulent toxins, which cannot be directly neutralised, and that death occurs from cardiac failure of central origin, stimulation seemed to be the most rational line of treatment to follow.

Though of the number to respond to stimulants only about 5% really appeared to be tided over the critical period by them, yet many were temporarily benefitted, and in the absence of other indications this method was therefore pursued.

Surgeon Major Deane in Bombay treated some cases on homœopathic principles with snake poison: at first some success was reported but latterly I have heard that the venom was an absolute failure and in fact did harm. There is certainly some similarity between the symptoms of both poisons. If drugs are to be used and the case is seen early enough it is advisable to start with Calomel gr. x, followed by a pleasant diaphoretic mixture. Later when the pulse begins to fail the time arrives for stimulants. It is quite useless giving them at first, before the pulse weakens.

Brandy was the most generally used. Given freely and at the right moment it often improved the condition of the patient and rendered a pulse again perceptible, but the result was disappointing, as a relapse not seldom followed in a few hours and the brandy soon had no further influence.

Caffein has been thought much of, but I found it of only temporary value.

Ammonium Carbonate was still better, and was particularly indicated in the pneumonic cases. Inhaled from a bottle, when the

patient was pulseless and unconscious, it often rapidly roused him and even brought back a strong pulse in a few minutes. Its effect however was very evanescent.

Digitalis was quite useless as its action is too slow: moreover a drug was required, which would stimulate a poisoned nervous system, and not one which acts to a large extent locally on the heart.

Strychnine was decidedly the most satisfactory drug of all. It strengthened the pulse and its effect was more lasting than anything else. The doses that could be borne without any ill effect were very large, 3 ii of liquor strychninæ being quite a usual quantity to order, and I have seen more than double that quantity administered without spasms.

Hypodermically strychnine, brandy and ether were found of value in the order named.

External reflex stimulants were always at hand. Mustard plasters over the cardiac region, hot bottles, and massage with mustard oil or croton oil were all tried.

For the frequent cerebral excitement, delirium and insomnia nothing acted so well as hyoscine hypodermically. It quickly quieted down a noisy patient and induced several hours of sleep without appearing to depress the heart in any way. Indeed the pulse of a wildly delirious patient often sensibly improved after an injection.

Of the other hypnotics *sulphonal* was the only one I tried, it acted very well in two doses of gr. xx each, when the patient simply was unable to sleep and there was no pain.

Morphia was seldom wanted as the pain in the bubo was not persistent enough to justify its use.

Starch & Opium enemata were used for diarrhoea, which was sometimes very troublesome towards the end. Feeding was not so very important as the acute period did not last many days. The usual liquid diet was taken together with a certain amount of rice. Care had to be taken to prevent patients from distending their

stomachs. Occasionally vomiting stood in the way of mouth feeding and in such cases, together with those in which a cervical bubo interfered with deglutition, nutrient enemata were resorted to.

For general rules the following must be adhered to. Keep the patient lying flat and allow no sitting upon any account for fear of syncope.

Give food out of feeding cup, and always use the bed pan.

Each patient must be provided with a spittoon, containing some antiseptic, and induced to use it.

Care is required in keeping the bed clothes dry and clean, for unconscious patients will often pass everything under them and a change may be necessary 6 or 8 times daily. A waterproof sheet is desirable on each bed.

The bladder must be regularly examined in unconscious patients particularly women, to guard against retention, and the catheter used when necessary.

Smoking should be strictly prohibited, since most natives use tobacco, which often contains some drug, to attain intoxication, a condition which cannot fail to be dangerous when the heart is already depressed.

The most noticeable effect of plague on the system is the profound depression of the cardiac centre, and the object to be aimed at is the avoidance of all exertion or emotional disturbance. If unattained, sudden death may ensue.

I saw one man well on the way to recovery sitting up and talking to a circle of friends, whilst devouring a large bowl of curry and rice. Regardless of remonstrations he insisted that he was quite well and felt very hungry. Half-an-hour later his death was reported. Either his uncalled for exertion or his distended stomach had proved too much for an already weakened heart.

Another man walked outside his hut during the temporary absence of his attendant, but fell down at the threshold and died in a few minutes.

Sometimes patients would walk to hospital to avoid being carried in a dhooly, of which they had a most unaccountable horror. Several of these died of cardiac failure within an hour of admission.

Treatment of the bubo. The local treatment of the bubo was the most satisfactory thing about the disease. The early pain was usually quickly relieved by frequently renewed applications of glycerine and belladonna.

Later, when there was great swelling and tension, hot fomentations soothed. Excision of the primarily infected gland and injection of pure carbolic acid I did not try. They failed in Bombay and indeed it is difficult to imagine what benefit could be looked for from either procedure. On the other hand allow the bubo to become tense and then, before suppuration has begun, make a free incision right through it, allow it to bleed, and dress with plenty of carbolic lotion. Other things being favourable all pain is relieved by this means and the temperature often falls to normal while the incision fills up and becomes well in a week or so without any suppuration in a fair proportion of cases.

If incision be delayed too long by a day the whole bubo will probably be found to have sloughed and the patient spends a weary time waiting for the cavity to granulate up. There is no suppuration to speak of from the cavity after the sloughs are removed, and I never saw the granulations become too exuberant, a troublesome feature in the Hong Kong epidemic.

If such a bubo is left to itself, and not incised, it bursts after a time through the skin at one or more points, and suppurates for a very long time, till the openings ulcerate to a size sufficiently large to permit the passage of the necrosed tissues.

Under these circumstances, the pus, not having free exit, may burrow and the patient may eventually die of septicaemia with his whole thigh a stinking bag of pus, or of septic pneumonia, the extension having taken place from the neck beneath the clavicle. Indeed while few things turn out more satisfactorily than a bubo incised at the proper time, there are few more horrible to see than one that has been left to itself for a month with the parts simply enveloped in leaves and rags. One thing to remember is that the

incision must be very free, from end to end of the swelling, and right down to the deep fascia. Otherwise, when the tension is relieved, what at first seemed a large cut is found to be really quite short, and a second operation will probably be required if suppuration begins.

A bubo never merely suppurated without some portion at least sloughing. The temperature, which often kept up to 105° F. for three or four days without a drop, required energetic treatment as it was associated with delirium, and the patient undoubtedly failed more quickly than when it remained at the more reasonable height of 103° F. or when intermissions gave temporary relief.

All substances like *antipyrin* are quite useless and tend to reduce the already feeble heart. *Quinine* may be given when the patient has a huge spleen and evidences of malaria, but I have not observed any marked improvement arising from its use. It cannot at any rate do harm, which is a very desirable property when careless Hospital Assistants are about.

Cold Sponging (70—80° F) only reduced the temperature by about a degree, and it would rise again in half-an-hour, so the sponging had to be repeated at frequent intervals. It was always very comforting and pleasant to the patient, so I used it in most cases even when the temperature was not very high with the result that sleep or quietness would often succeed restlessness.

The *wet pack* was more effective, but had sometimes to be applied for many hours at a time, the nurse of course taking the temperature and watching for signs of collapse. Cold baths when practicable would undoubtedly be useful.

The ice cap to the head was found very grateful, relieving head-ache, calming delirium and reducing the temperature much better than sponging. When the acute period had passed no further treatment was as a rule necessary as the patient was not generally greatly reduced and quickly recovered strength. Only the bubo required attention. Some patients however in whom the fever continued for three weeks or more and those in whom there had been profuse suppuration lost a great deal of flesh and strength and wanted a month or so of tonics and good diet before they were fit to be discharged.

Inoculation. The advantages of inoculation with Haffkine's prophylactic, the success that has attended its employment on a large scale, and the dangers that may result from its injection, have been recently thoroughly investigated by the British Plague Commission who have come to the following conclusions:—

1. Inoculation sensibly diminishes the incidence of plague attacks on the inoculated population, but the protection it affords is not absolute. It is impossible to express the measure of protection numerically.
2. The fatality of plague attacks is diminished among the inoculated.
3. The full protection is not acquired for some days after inoculation.
4. The maximum duration of protection may be a considerable number of months, but can only be determined by further observation.
5. There appears to be a definite quantum of the fluid which gives the maximum amount of protection. So the best results will only be obtained after an accurate method of standardisation has been devised.
6. There is no ground whatever for asserting that death has resulted from inoculation and in the overwhelming majority of cases no evidence was adduced to show that any injurious effect has supervened upon inoculation.

The prophylactic is prepared by cultivating the bacillus in a fluid medium of animal or vegetable origin for 6 weeks in the dark, destroying the organisms by heat and bottling the fluid without filtration and with care to avoid contamination. The dose to be injected varies from 2 c.c. to 10 c.c. and two injections have been considered more efficient than one, but when the optimum dose is known this will be unnecessary. A full account will be found in M. Haffkine's lecture before the Royal Society ²³.

With regard to its effects they vary in different individuals, but the most usual are fever beginning about 6 hours after the inoculation, lasting for some 24 to 48 hours, and pain and swelling

at the seat of the operation, lasting for perhaps a week. General malaise and vomiting are not unusual, but the majority of persons are fit in about 3 days.

Theoretically, all the inhabitants of a threatened town should be inoculated before the disease has established itself, but, practically, the apathy of the average native, combined with the absence of accurate information on the subject of the duration of the protection, makes this course impossible to be followed. Small towns of 20,000 inhabitants have been entirely inoculated, but in large ones of over 1,00,000 it is doubtful how many could be induced to avail themselves of the chance. When Madras (population about 5,00,000) was threatened 5,800 persons came forward to be inoculated in a month, but they ceased to present themselves directly the scare passed away and no indigenous cases came to light. Lustig is preparing a prophylactic on a different principle from Haffkine's from which he expects great results. He considers the fluid part of Haffkine's fluid is useless and that the virtue lies in the nucleo-albumens contained in the dead bacilli, so the substance he will issue will be pastelike or solid. No results are as yet to hand. The curative serums prepared by Yersin and Calmette have not so far proved to be of particular value.

Post-mortems ²¹. Owing to religious difficulties coupled with extreme pressure of executive work I was unable to do more than 6 *post-mortems*, none of them being cases which had been under my own observation during life.

No. 1. There was nothing found to indicate death from plague, no bubo, pneumonia, no bacilli, in any of the organs. It had been certified as plague.

No. 2. There had been signs of pneumonia before death but no hæmoptysis. A small pneumonic patch was found in the lower lobe of right lung, over which the pleura was adherent. The lobe was deeply congested on section and the patch full of bloody fluid, containing no air, and sinking in water. Bacillus not found, possibly owing to decomposition having set in.

No. 3. Also had pneumonia during life. A small patch was found at the *post-mortem* closely resembling No. 2. Bacillus very abundant in the fluid. Spleen enlarged and friable; bacillus found in small colonies. Liver slightly enlarged and studded, both on the

surface and within, with what appeared to be minute abscesses, 1-3 mm. in diameter. These proved on examination to be pure cultures of the plague bacillus.

No. 4. Bubo in axilla. Bacillus found both in this and in the spleen. The intestines were deeply congested throughout their length, as if they had contained some highly irritating substance.

Nos. 5 & 6. Each had a bubo. No bacilli found. Intestines very pale. In none of these cases were the bronchial or mesenteric glands enlarged or inflamed as one had been led to expect, and in one case only, No. 4, were other glands besides those in the bubo enlarged, namely two iliac glands.

After death the previously tense bubo flattens out to a certain extent and the glands found in it were not so large as might have been expected. In each case the tissues were deeply blood-stained for some distance around the bubo, and in one the extravasation followed in the track of the lymphatics for several inches. In fact the *post-mortem* appearances of a bubo are quite unlike any other pathological state of lymph glands and their surroundings that I am acquainted with.

Infectivity of Plague.—The disease is not very infectious, at least in the bubonic form, and people can be in daily contact with cases over a period of some months without taking it. No immunity is thus established, for I know of three cases in which nurses were attacked after 2 to 4 months of hospital work. One died and two recovered.

In Karachi no paid hospital attendant or servant was attacked, with the exception of one of the above mentioned nurses. Until the bacillus has obtained access to the blood it is difficult to understand how the patient can disseminate it at all. I believe that plague cases could be treated in the general wards of a hospital without danger to the other inmates, provided that the supply of fresh air was unlimited, that the nursing was first class and care was taken in properly dealing with the excreta and expectoration. It would, however, be unjustifiable to experiment in this matter till more is known. Government, acting on the advice of certain of its Medical Officers, has issued a circular recommending the isolation of pneumonic plague in separate wards where possible.

Dr. Whyte in his report on the Guzarat epidemic of 1820, remarks that "although this disease in a close ill air situation is highly contagious, in an open space it is quite the reverse. It has never remained long in any except the large and walled towns".

If this were not so, if plague were as infectious as small pox, then it would be hard to understand how any natives at all escaped in the early days before segregation was enforced, owing to their pernicious habit of crowding into a sick room and sitting seven and eight on the patient's charpoy to watch him die.

A sharp distinction must be made between the simple bubonic cases and the pneumonic from the point of view of infectivity. Mere proximity to the former does not usually lead to bad results, but living in a confined space with a pneumonic patient is highly dangerous by reason of the necessary inhalation of the air he has already breathed and the probability of inoculation with his dried sputa. A nurse in Bombay, whose conjunctiva was accidentally inoculated by a patient coughing as she was bending over him, took the disease.

Plenty of fresh air in a sick room, strict cleanliness, attendance to all abrasions, good food and regular exercise will almost certainly, barring accidents, preserve attendants from infection. On the other hand, sitting for hours at a time with the head within a foot of a coughing pneumonic patient in a stuffy hut, carelessness with the sputa and excreta, eating with unwashed hands, &c. will just as surely invite it.

How long a patient should be kept in hospital before he can be declared free from infection is still a matter of opinion. The following periods seem to me to be sufficient.

1. Pneumonic plague. Till lungs are quite clear and the patient is otherwise fit; perhaps four weeks.
2. Incised bubo. Till the wound is healed and the bubo well on its way to absorption.
3. Sloughed and suppurating bubo. As bacilli are not contained in the discharge it is sufficient to keep the patient till he seems fit to go, say four or five weeks.

4. Spontaneously absorbed bubo. As it may be several months before all traces of enlargement have disappeared, and the patient has no means of discharging bacilli, probably four weeks from date of attack would suffice.

Other cases presenting peculiar features may require longer treatment but only the minimum safe periods are named above. Patients are usually unduly anxious to leave a plague hospital and should therefore be allowed to depart as soon as a due regard for the safety of their neighbours will permit their return to ordinary life. The incidence on the various castes, races and communities will not be known till all the statistics are collected and made available.

Incubation Period. In no case that came under my notice, did the incubation period exceed a possible seven days, and generally it was much less, but other medical officers have told me of cases coming by train from an infected to an uninfected area which developed plague 10, 12 and even 15 days after arrival. Still the factor of inoculation during that period from infected clothing, hair, &c., is always difficult to eliminate.

It may be that the incubation period is shorter in pneumonic than in bubonic plague, just as the former is the more virulent, still I cannot help thinking that 10 days quarantine is unnecessarily long for the majority of cases and that eight would suffice: but until all the results of the epidemic can be compared no law can be laid down and many people must suffer in order that the few with abnormally prolonged incubation periods may be caught.

Disinfection. In the early days of the epidemic, in the absence of exact knowledge and without the means of experimenting, it seemed highly important to disinfect all bedding and wearing apparel both before and after segregation. This was effected by the immersion of all goods that would be uninjured by the process in a solution of Jéye's fluid or by boiling for half-an-hour. Things that would have been injured by water such as silks and velvets were hung out in the sun for half-an-hour. The strength of Jéye's used varied from 1 in 20 to 1 in 60 of carbolic and the immersion was but momentary. However the prolonged exposure to the sun

and air that a soaked mattress got, together with the subsequent washing in water to extract the carbolic, were, judging by the results, sufficient to destroy bacilli if they were present. The chief objection to Jeye's was its expense but the rapidity with which things could be disinfected in this way more than counterbalanced the cheapness of the lengthier process of boiling, which was a matter to be considered when one had to disinfect the kits and persons of 1,200 people in a day in addition to other work. No case of plague came to my knowledge which could be in any way ascribed to inefficient disinfection.

Houses were thoroughly cleaned of all dirt, then sponged with a solution of crude carbolic or perchloride of mercury, saturating all corners and crevices and including the ceiling. After the lapse of a few days to let them get quite dry again, lime washing, tarring and painting finished the process.

Windows were made where none existed and holes were broken through walls and roofs, when necessary, to admit air and light. The matting of mat huts was all burnt and the bare poles and floor disinfected.

Subsequently an acid solution of perchloride of mercury was found to be the cheapest for general use, and according to Hankin the most efficient germicide.

The acid serves a double purpose, it is inimical to the plague bacillus and it prevents the formation of insoluble albuminates of mercury which otherwise nullify the power of the fluid when it comes into contact with organic matter such as a cow-dunged floor. The researches of Hankin, the German Plague Commission, Marsh and Pitchford all indicated the superiority of perchloride of mercury. Dilute acids also kill the bacillus readily, but it is somewhat resistant to the action of carbolic acid and alkalies.

Chloride of lime and permanganate of potash are also valuable on occasions. The disinfection of the persons of travellers from infected districts is not a measure of much value. As practically applied it was probably useless; and in addition entailed much discomfort and annoyance on the people who had to undergo it. Kurth examined suspected hides, cotton, jute, &c., but found no

evidence of the likelihood of the importation of plague thereby. The German Plague Commission confirmed this view with regard to hides.

Disinfection of merchandise on a large scale is impracticable, and to play at disinfection is a most expensive and futile game, which can have no value except to enable the authorities to impress the ignorant with the idea that something tangible is being performed. If importation is feared then either all goods from an infected port should be rejected, or only such of them, as waste and rags, as can reasonably be suspected to be dangerous. To scatter carbolic powder over Mail bags as they do at Brindisi, or to sprinkle passengers, luggage or bales of goods about to be shipped from an infected port, is pure and simple waste of money.

Mortality. In my division in Karachi there were 1,190 cases of plague and of these 891 died giving a case mortality of treated and untreated together of 74 %.

Of the total number of cases treated in my hospitals, viz: 341, 160 died giving a case mortality of 46·6 %. This is too low, and is accounted for by the fact that the hospitals were all opened about the same time and all cases found at that time, whether convalescent or not, were sent into them.

Of 246 cases treated from beginning to end 156 died giving a case mortality of 56·6 %. Subtracting number of persons treated from total number of cases, and the number of deaths amongst the treated from the total number of deaths, we get

1,190 — 276	914 cases.
891 — 156	735 deaths.

giving a case mortality amongst the untreated of 80·4 %.

This difference of nearly 24 % between the treated and the untreated is in my opinion as much due to the removal of the former from dark, damp, dirty and unventilated huts, where no sort of care, in our sense of the word, was taken of the patient and he was worried all his time by being made the object of curiosity to as many neighbours as the hut could accommodate, to light, dry, clean and airy hospital huts where he was kept quiet and well attended to.

The mortality varied in different hospitals, thus in one hospital there was a case mortality of 58% while in another it was 48%.

So a proper estimate can only be formed of the effects of hospital treatment by taking large numbers. In Bombay, the hospital returns show mortality varying from 50%—70% and, as before mentioned, the total returns for the whole city show a case mortality of 86.4%.

As stated before the toxic form proved to be most fatal, the pneumonic next and the bubonic least. Pregnant women usually aborted; sometimes the child lived, but showed no signs of plague, the mother generally died of hæmorrhage. Young children under three years did not very often suffer, babies in arms still more rarely. Old people too were remarkably exempt, but if they got the disease they succumbed.

The following analysis of 247 cases in which full notes were taken shows the site of the bubo in the sexes and the prevalence of primary pneumonia.

	Bubo in groin.	Bubo in Axilla.	Bubo in Neck.	Primary Pneumonia. No bubo.	No bubo. No Pneumonia.	TOTAL.
Male	50	15	5	28	9	102
Female	16	17	8	11	2	54
M. Children	17	6	18	1	3	55
F. Children	13	8	15	...	...	36
Total ...	96	46	46	35	14	247

This table bears out the prevalent idea that groin buboes are commonest in males, axillary in females, and cervical in children.

Primary pneumonia was equally common in both sexes while children were almost exempt. It formed about 13% of the total number of plague cases.

Of the 35 primary pneumonias five recovered, three of these having had slightly rusty sputa for a short time. Those that had hæmoptysis all died.

Table of the case mortality among the 247 cases analysed above:—

90 Males with buboes	C. M....	...	...	...	57%
47 Females	"	...	...	...	38%
29 M. child	"	...	...	...	34%
31 F. child	"	...	...	...	35%
22 Males with Primary Pneumonia	C. M. ...	...	...	...	91%
13 Females	"	...	...	...	85%
9 Males	} No bubo	} Toxic	} Plague C. M.	100%	
2 Females					
3 Children					

The average duration of fatal cases was males 5.9 days, Females 4.0 days.

This male average is considerably raised by one or two cases which did not die for 3 or 4 weeks.

Diagnosis. A case cannot be diagnosed with certainty at the outset, when there is only high fever, so all such suspicious cases must be isolated till the appearance of pathognomonic symptoms. As mentioned before it sometimes happens that the bubo does not make its appearance until after 3 or 4 days of irregular fever, but this is distinctly unusual. There is no difficulty in distinguishing old, chronic, enlarged glands from a recent bubo. The doubtful cases are the ones with slight pneumonia with no blood spitting, bubo or great depression. If other members of the family are ill at the same time with the bubonic form, it is enough to clinch the diagnosis, but otherwise it can only be made certain by cultivating the bacilli from the blood or sputa, as ordinary cover glass preparations do not suffice. A great point in distinguishing plague pneumonia from the ordinary form is the case mortality, which is much higher in plague than even in the so-called epidemic pneumonia, but of course this is of no value in diagnosis at the time aid is required.

Severe remittent fever is about the only thing that can well be confounded with plague during an epidemic, but the lapse of

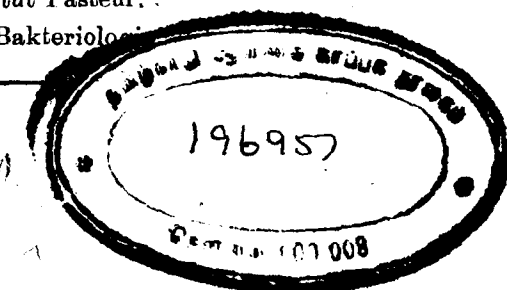
time soon removes the doubt. Sometimes concurrent diseases such as paralysis, cedemas and heart troubles may cause confusion, but a little time and care will readily determine the immediate cause of illness.

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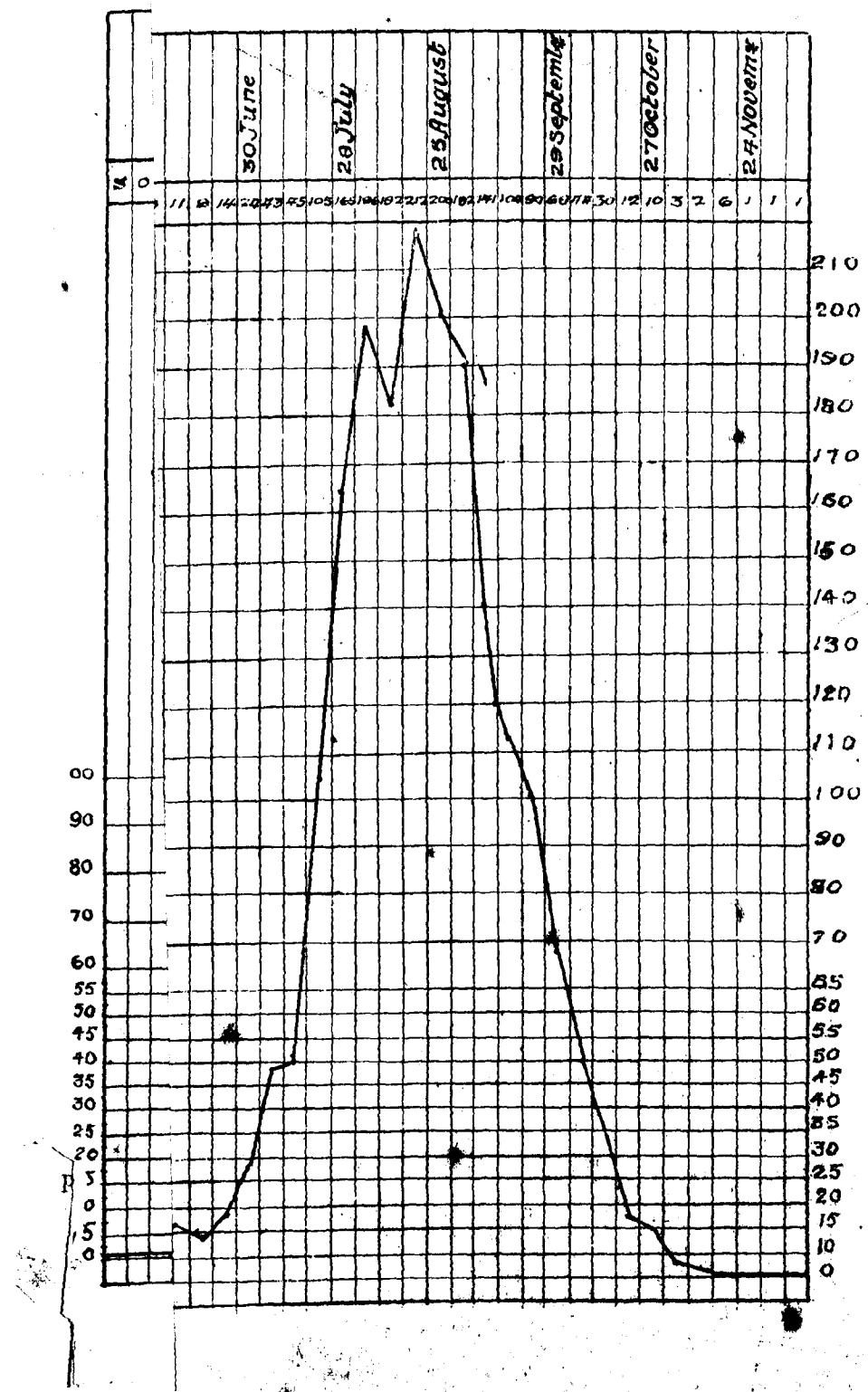
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PERIOD WEEK ENDING	11 Dec 98	18 Dec 98	25 Dec 98	31 Dec 98	7 Jan 99	14 Jan 99	21 Jan 99	28 Jan 99	4 Feb 99	11 Feb 99	18 Feb 99	25 Feb 99	3 Mar 99
AVERAGE DAILY CASES	50	45	40	35	30	25	20	15	10	5	0	complete Freedom	



As there was nothing very pressing, interesting or important just then in Chingleput I decided to revisit the East Coast between Ennur and the Coromandel and make a thorough search for the anopheles. I found that the different species were as easily distinguished in their larval and pupal forms as they are as full grown insects and so armed with a few bottles and a saucer I spent a week on the Coast. The result of extensive enquiries were roughly that

- (1) the Coast was quite healthy and malaria of a severe type unknown before 1895;
- (2) that from August to November were the worst months for fever;
- (3) that the Salt Department found their factories had become so unhealthy that Ennur had been practically abandoned by the Assistant Commissioner and all his Office;
- (4) that it was extremely difficult to get men to work for the ordinary wages at the Puldivak and other factories on that Coast.
- (5) that some villagers stated that persons who cut down the trees in casuarina plantations were always badly attacked by fever;
- (6) that casuarina plantations existed long before 1895;
- (7) whilst fever was very bad in Ennur in 1895 it did not get bad in Carimanul villages until 1897.

The question seemed to me to narrow itself down to this—

1. Are malaria-carrying mosquitoes prevalent?
2. Why should casuarina plantations be malarious?
3. Granted that casuarina plantations are now very malarious, why were they not so before 1895?

I claim to have completely satisfied myself on the first two points, but to have no practical explanation, such as I dare bring before the meeting, of the third point.

I would venture to remind you, Mr. President and Gentlemen, that the anopheles mosquito is known to breed in pools and puddles, in paddy field water (if not flowing too rapidly) and in wells (if unused and undisturbed) and in tanks and ponds, especially

small ones that contain no fish or frogs. I would further venture to remind you that the stretch of coast running from Ennur to the Coromandel is practically a strip of sand dunes and paddy fields, separated along its entire length from the remainder of the Chingleput District by the canal—this canal opens into the sea at Ennur at Karinkali and at Pulicat. A great deal of this Coast is only slightly higher than the sea level and is to a large extent wooded with casuarina plantations in various stages of growth. As far as I could judge from the shallow wells which exist in all the villages there was fresh water to be got everywhere in the dry season at about 6 to 12 feet below the surface. I learned that the casuarina trees require for the first five years of their life to be carefully and constantly watered, but that after that age the roots have penetrated to the water level and watering becomes unnecessary. The water in the casuarina plantations is so near the surface, even in the dry season, that the coolies do not trouble to make real wells in the plantations, but simply dig out or scoop out shallow holes in the sand in which about one foot of water daily accumulates. These pits or shallow wells are not filled in when the trees no longer require artificial watering. It is also a noticeable feature of many of the small villages along the Coast that being poor people, the inhabitants find that a costly brick-lined well is unnecessary and that large holes dug in the sand round the outskirts of the villages supply sufficient water for men and animals. I have now practically told you the result of my investigation, for it only remains to be said that anopheles in hundreds, anopheles in thousands exist in almost every village pool and well and in every one of the casuarina pools I examined, also beyond Adyar; except those that had only just been made or from which water has been daily removed. I am, therefore, in a position to answer the first two questions.

(1). Malaria carrying mosquitoes are extraordinarily prevalent even at the end of the cold season.

(2). Casuarina plantations (after the trees have grown too old to require watering) are particularly suitable places for the growth of malaria carrying mosquitoes.

Question 3. As to why malaria has only been bad since 1895, whilst casuarinas existed from 20 years before, I do not pretend to

explain. I feel that this question is one that can only be answered by the gradual accumulation of scientifically accurate facts and by a fuller knowledge of the life history of the malarial parasite in relation to the anopheles, not forgetting that to start an epidemic of malaria there must not only be anopheles on the scene but diseased, malarial parasite infected anopheles, and that these must have been infected from man and so it seems to me that either (for some reason as yet unexplained) anopheles mosquitoes bringing with them the malarial parasite invaded this strip of Coast in 1895, or some person or persons at a particularly favourable moment came to that Coast and infected the anopheles already existant there. Here, however, I begin to feel the ground very unsafe beneath my feet, partly as I am dealing with theory, always to me distasteful when the foundation of facts begin to be scanty, and the superstructure of supposition disproportionately heavy, and partly because being myself a recent convert to the mosquito malarial theory, I feel that it requires some boldness for me to address you on this subject at all. I can only, in conclusion, ask for the same mercy as is usually accorded to indifferent performers in the music saloons of the Far West notified by the placard "Do not fire at the performer he is doing his best."

I have since learned from Officers of the Salt Department that similar waves of malaria have spread over several of the other salt factories along the Eastern Coast from Ganjam to Cheyoor. It seems that the virulence of the malarial parasite varies and even vanishes sometimes in much the same way that cholera microbes suddenly spring into vigorous growths and produce an epidemic, although to us who do not as yet know or fully appreciate the conditions of the struggle for existence and the hourly hurly burly of microbe life, it would seem that cholera once started should not stop until it had made a clean sweep of the Eastern world. So, I take there are restraining influences constantly at work killing off or rendering slow of growth both the malarial parasite and its host the anopheles mosquito. Perhaps, some day there may arise again a Ross among the Indian Medical Service of Southern India who, by dint of laborious observation, may grasp the life history of the pest and understand the conditions that favour or retard the growth of both the mosquito and its parasite and that epidemics of the kind

may be stamped out or greatly lessened in violence; in the meantime it would seem to be the legitimate duty of this Branch to stir up all its Members to carefully record in the light of the mosquito theory all facts, however apparently divergent, connected with malaria and to educate public opinion on this subject. Captain Donovan and Captain Cornwall are about to follow me with a description of the development and characteristics of the various forms of mosquitoes, a knowledge of which is now-a-days essential to all medical men practising in tropical climates.

G. G. GIFFARD,
CAPTAIN, I.M.S.

NOTES ON CASES OF MEDICO-LEGAL INTEREST.

This paper is, as its heading implies, an attempt merely to place on record a few of the more interesting cases of chemico-legal interest which have passed through my hands. One of the most salient features to my mind is the large number of cases in which the motive has been of trifling importance when compared with the gravity of the crime. In the recorded history of poisoning in other countries we find the poisoner playing for high stakes and even from before the Christian era poisoning was much used, *e.g.*, as a political engine. In the 15th century we find secret assassination by poison actually receiving State support, for in the days of the famous "Council of Ten," the trade of the Venetian poisoner was in such a flourishing condition that he could command a lucrative tariff, exclusive of travelling and other expenses; the farther the journey, and the more eminent the man, the heavier was the payment. Detection in those days was well nigh impossible for even dissection was not availed of. Since then the art of poisoning has declined, not so very much, perhaps, because of an altered state of society, for even in modern days we have had such dastardly deeds as those which effaced President Garfield, M. Carnot, the Empress of Austria, and King Humbert; the modern weapons of such crimes are lead and steel. It is to the general advance of the art of dissection at first, by which we have acquired knowledge of normal and abnormal signs in the tissues; and subsequently to a gradual improvement in chemical methods which are now able to unearth the deadly agent even after it has been buried for months—it is to these agencies that we may fairly ascribe the

gradual decline of the poisoner's art in European countries. But the motives in civilized countries even now may be said to be "lofty;" suicide, the acquirement of a large fortune, are some of the motives which now appear prominently; and the crime of administering poison, for the sake of murder or attempt to murder, is comparatively speaking one of extreme rarity. Statistics for 10 years ending 1892, show that out of 6,616 deaths from poison in England and Wales, only 42 were traced to murder, the rest being suicidal and accidental. I am unable to give you comparative figures for Madras Presidency, because it is only since recently that judgments and results of judicial enquiries have been communicated to the Chemical Examiner, and there is always a certain proportion of undetected and false cases which are difficult to eliminate. But the following figures compiled from known results of enquiries will give you some idea of the small proportion of suicidal and accidental poisonings.

Years.	Total cases in which poison detected.	Accidental.	Suicidal.
1897 ...	89	5	8
1898 ...	99	6	11
1899 ...	72	8	10

There is no absolutely certain method of verifying whether all the remaining cases ought to be classed as homicidal or not, but my experience convinces me that it would not be far from the truth to assume them to be so.

Here then we have a high ratio of homicidal poisonings, and as I remarked at the outset the motives are in many cases quite disproportionate to the gravity of the crime. Poisoning in India dates from very early periods of Asiatic history. It was not an uncommon practice even in early days to settle a trifling conjugal quarrel by resort to secret assassination by poison and the ancient practice of suttee has been ascribed to the necessity of checking this tendency to domestic poisoning.

Now, I will quote a few cases picked up at random to show you how trivial are often the motives which actuate homicidal attempts:—

(1) In order to obtain money which was refused on loan, a man contrived to poison the food of thirteen persons and to be sent for to treat them for Rs. 4. His treachery was fortunately discovered, and he was sentenced to ten years' rigorous imprisonment.

(2) Two men quarrelled about water, which they shared in turns for irrigation, and the villagers interfered and settled the dispute. One of the parties not satisfied with the settlement took up a handful of dust, showed it to the sun and dropped it—a form of curse indulged in by Hindus during a quarrel—and he declared that whoever was guilty of injustice would lose his son by next morning. The same afternoon, this man contrived to administer poisoned jaggery to the other man's son, who fortunately recovered under timely treatment in the dispensary. The vomited and purged matters contained arsenic; the purchase of jaggery that day by the accused was proved, and he was sentenced to four years' rigorous imprisonment for attempting to commit murder.

(3) A man visited another's paramour on the strength of a former intimacy with her, and she received him with favour. This led to a quarrel between the men, who vowed vengeance against each other, declaring that each would convert the other's "house-site into a field of castor oil plants," meaning extinction of the family; while the one engaged a sorcerer to work spells, the other stole a march on him by poisoning his son with an arsenical jaggery ball.

This last case it seems to me is one which called for the immediate purchase of a new whip; instead of this it seemed more natural for the aggrieved party to invest in arsenic.

The following case could hardly be surpassed for the diabolically cruel method of removing an objectionable husband.

(4) A man was seized with vomiting and purging soon after eating a meal served as usual by his wife. He continued for six days gradually getting worse. The wife, who was all the time in illicit love with another man, is reported to have "sat on his chest on the seventh day, throttled him with one hand and with the other

squeezed the testicles," which were described in the *post-mortem* notes as in a "pulpy condition." The final injury, no doubt, accelerated the death which was approaching from the effects of arsenical poison. The woman was tried for murder and sentenced to death, but the sentence was, I believe, subsequently reversed on appeal.

The surprise here seems to me that she did not save herself all this trouble by simply deserting him. By way of contrast, I will give you a case of *feigned poisoning*, under somewhat similar circumstances:—

(5) A young woman who was in intrigue with a man refused to live with her husband on the plea that she was invariably seized with vomiting, which regularly ceased as soon as she left him and returned to her own village. She was induced by the friends of both parties to return and "try a last chance", but the vomiting recurred. While her husband was asleep she wounded him grievously with a mamooty, in order to sicken him of her, and she then set up the plea that she was ill-treated and made to vomit by the forcible administration of nauseous substances. No poison was found in the vomited matter, and from the evidence the Judge concluded that she forced herself—mechanically or by drugs—to vomit, so as to make herself as unpleasant as possible or to arouse sympathy, and to have a decent pretext for returning to her lover. She was sentenced to five years' rigorous imprisonment.

A common enough class of cases is that in which the object cannot be said to be murder; but bodily hurt is certainly intended, and they show to what lengths the dabbler in poisons will go. I refer to cases in which the *Mantara-karah* is called to detect the culprit in cases of theft:—

(6) A woman lost some jewels from her house, and a reputed magician offered to recover them for a reward of Rs. 2. He gave her some sanctified rice to distribute among the neighbours, which, he said, would compel the thief to confess; but this *ruse* failed. He then prepared some balls, which, he said, would give the thief a pain in the belly; and of the neighbours who came by invitation to witness the trick, twelve persons ate the balls which were

distributed by him. One of these was soon after seized with the usual symptoms of arsenical poisoning and died. It appears that deceased was more strongly suspected than any one else of having stolen the jewels, which were mysteriously restored to the house of the owner during the night of his illness and death. The magician was sentenced to seven years' rigorous imprisonment for his successful performance.

(7) A theft occurred, and a magician with a reputation for detecting thefts was employed to discover the thief by sorcery. At a preliminary *séance*, at which rice was thrown on a winnow supported on the points of a pair of scissors, certain persons were suspected by the winnow shaking when they threw rice. At the next performance, the stolen property was to be restored; before a large crowd of people a hen was first whirled about over a diagram on the floor and caused "to fall down dead"; then an image was moulded out of flour and from it little balls were made and distributed to the suspected persons, one of whom became violently ill in about fifteen minutes, lingered in pain for eighteen days from mercurial poisoning, and died. The magician was sentenced to three years' rigorous imprisonment.

(8) A quack with a reputation for detecting thefts was engaged to trace a stolen jewel. All the villagers were summoned, given each a pill and told to re-appear next day; but an additional pill was administered to the deceased, who suffered from intense symptoms of aconite poisoning. Meanwhile, the quack was going through a sort of hypnotic performance with a boy who was made to stare at a light and to whom leading questions were being put as to whether he could see in the light a person resembling the deceased. The appeals of the suffering woman for an antidote were met with the reply that if she would restore the jewel she would be all right. When the quack at last yielded and gave her some gruel, it was too late, she could not swallow and she died in a short time.

There is happily another side to all this sorcery and quackery, from which it will be seen that the advantages are not always on the side of the sorcerer and the quack. Here is a somewhat amusing case of the biter bit:—

(9) A man suffered from loosening of teeth, which he believed to be the consequence of medicine prescribed by his family physician,

and so he determined to have his revenge. He obtained a fresh bolus of the medicine and awaited his opportunity. The quack was one day passing the house, and having been invited to enter it he was requested to swallow his own pill. Persuasion having failed, the quack was seized by the neck and the bolus was violently rubbed into his mouth. The aggressor in this case very properly received rigorous imprisonment for committing such an indignity on a professional man!

The next case shows that even sorcerers are not safe among people whose motives are actuated by strong superstitious beliefs:—

(10) A man who was a reputed sorcerer was asked to visit a sick child; on his entering the house four persons fell on him and floored him. Then one of them held him down with a rope twisted round his neck; another forcibly held open his mouth; the third a goldsmith knocked off 12 teeth with the end of a stick; and the fourth filled his mouth with dirt. They then carried him out into the road and left him half dead from shock and bleeding until he was discovered and removed to the dispensary. There is a superstitious belief in the district from which this case was reported that the utterances of a sorcerer became ineffectual by knocking off his teeth. There were reasons adduced, trifling enough in all conscience, for resorting to this barbarous but orthodox method of rendering the sorcerer's words powerless.

The most common tool of the poisoner in India is WHITE ARSENIC, and so the majority of cases met with are those of arsenical poisoning. I will record only a few of those which appear to have some points of special interest attaching to them. The large quantity of white arsenic found in the body is the leading feature in the following three cases:—

(1) A corpse was found in the bed of a river, and at the *post-mortem* examination some suspicious particles were observed in the stomach. In the visceral matter sent I found altogether over 133 grains of white arsenic, some of the partially digested pieces being as large as marbles.

(2) A man, who was reaping his varagu crop, remonstrated with a crowd of about 40 persons, who came and attempted to

remove it by force. He was then tied up to a post and beaten and left in an unconscious state. From the stomach were picked out coarse particles of white arsenic, some of them as large as a cherry stone, and weighing in all 98½ grains. Six more grains of it were extracted from the viscera sent, making a total of over 104 grains of white arsenic found within the body. I pointed out in my certificate that such large pieces of white arsenic could not have passed into the stomach during life, unknown to the deceased, and that he must have swallowed them with suicidal intentions or have been forcibly made to swallow them. The Magistrate found "no evidence of how the white arsenic got into the stomach of the deceased and that probably it was administered to him in order to get up a false case of murder."

(3) In a case which ran a fatal course in about six hours, from the vomited matter sent up I picked out over fifty grains weight of white arsenic, some of it in pieces about the size of a pea; similar pieces weighing over four grains were picked out of the purged matters and from three cloths soiled with excreta. The Medical Officer also forwarded similar pieces weighing over 68 grains which he had picked out of the interior of the intestines at the *post-mortem* examination; and, in addition to all this, I extracted from the viscera the equivalent of over 17 grains of white arsenic. The exact amounts were:—

In the vomited matter	...	...	60.5	grains As, O.
In the purged matter and soiled cloths	...	...	11.48	"
Total ejected			71.98	"
In the stomach and its contents, portion of liver,				
one kidney and part of intestinal contents	...	...	17.82	"
Picked out of intestines alone at the <i>post-mortem</i>				
examination	...	...	68.54	"
Total retained			86.36	"

The total quantity which this woman took must have been over 158 grains, much of it in coarse lumps which had in a short space of time been conveyed and scattered throughout the whole alimentary tract. The tenacity, with which these particles were found imbedded in the mucous lining of the intestines, was sufficient to convince any one of the utter hopelessness of attempting to dislodge and

eliminate them by any form of treatment during life. [*Specimens of the white arsenic obtained from these three cases were handed round for inspection*].

In some cases of arsenical poisoning there are very slight symptoms of gastro-intestinal irritation, or even none at all, while the nervous system becomes affected, the patient presenting the symptoms of coma, as in opium poisoning. The following cases appear to be examples of this uncommon form:—

(4) Two boys were given some jaggery by an old woman, and one of them immediately "vomited, went off to sleep and died in six hours."

(5) Another fell asleep in fifteen minutes and died in five hours without any other symptoms.

(6) A nautch girl was treated to a glass of brandy and subsequently to "some other liquor," shortly after drinking the latter she became insensible and died in a short time without regaining consciousness.

In all these cases arsenic was found in the viscera; while in a fourth case in which convulsions preceded coma, I found as much as 32 grains of white arsenic in the stomach. The need for caution in certifying to the cause of death in such cases is evident.

Death from *secondary effects of arsenical poisoning* are rarely met with, although the majority of fatal poisoning cases in this Presidency are due to arsenic. The next case is of further interest in its bearing upon the period of complete elimination of this poison from the body.

(7) Seven persons suffered from symptoms of irritant poisoning and one of them died. Suspicion was subsequently aroused that they had by mistake eaten sugar, which had been poisoned with arsenic in order to kill vermin. Traces of arsenic were found in the exhumed viscera. Another of the attacked party died a fortnight after all active symptoms had subsided, having complained of continual abdominal pain during this period. Traces of arsenic were detected in the liver and kidney, the poison not having been completely eliminated from these organs even after so long a period.

The next three cases are illustrations of *well-poisoning*, and happily they are not of common occurrence:—

(8) Several shallow wells had been dug in the dry bed of a tank, and over twenty persons who drank the water from one of these wells were poisoned. On baling the water out in order to clean the well, abundance of a yellow powder was found sticking to its sides, which was sent for examination and turned out to be impure arsenious sulphide (containing white arsenic).

(9) A woman, T... e, who had quarrelled with her paramour refused to return to him, but received the visits of another man. A friend with whom she was living suffered from purging, vomiting and sore eyes, all characteristic symptoms of over-dose of arsenic; and she herself suffered from similar symptoms and died; about the same time a neighbour also died. It seems that all three persons obtained water from the same well, and the paramour was suspected of having poisoned the well. There was no evidence against the accused and he was discharged; but arsenic was extracted from T... e's organs equal to three-fifths of a grain of white arsenic and also in about a pint of water taken from the well equal to one-hundredth of a grain. In the judgment, the insolubility of white arsenic in cold water is given as a reason against the possibility of deceased having been poisoned with arsenic through the well water, and this is done on the authority of Taylor, though the latter admits that "cold water allowed to stand many hours on this poison will dissolve about half a grain of it in a fluid ounce."

(10) Nineteen persons after drinking well water suffered from vomiting, purging, and burning sensation in stomach without a fatal result. Two grains of white arsenic were obtained from six quarts full of water.

Arsenic is a constant ingredient in the composition of "love quenchers," and not long ago I found 45 grains of white arsenic in one of these—a most efficient quencher; but curiously enough it is as often as not an ingredient also of philtres which guarantee unfailing love; but a novel effect was claimed for arsenic in a case in which a woman narrowly escaped death after drinking milk in which "something" had been mixed, which was said to have "the virtue of imparting a good complexion to her child *in utero*."

These cases only further illustrate what slight provocation makes people fly to poison in order to avenge an injury, and how great is the need for restricting the possession and sale of poisons.

The next group of cases are those of *Mercurial poisoning* :—

(1) A case of more than ordinary interest was that in which a man, who was not on good terms with his wife, devised a peculiar method of punishing her by surreptitiously introducing a piece of corrosive sublimate into the vagina after having an intercourse with her. The drug not only caused local inflammation but it also gave rise to all the symptoms of general mercurial poisoning, including ulceration of the mucous membrane of the mouth and the stomach, from the effects of which the poor woman died. Twelve days after, the visceral matters were found to contain mercury equivalent to a large poisonous dose of mercuric chloride. A contention arose during the trial, as to whether the symptoms of mercurial poisoning could have been the result of swallowing the drug; but no evidence was forthcoming in support of this contention. On the other hand, the whole of the evidence went to confirm the fact that the external application of corrosive sublimate caused inflammatory changes in the alimentary canal of about the same intensity as when the poison is swallowed. The accused got 10 years' rigorous imprisonment.

(2) As an example of wanton cruelty, perpetrated apparently in the absence of provocation, the following could scarcely be surpassed. Some persons forcibly held down a woman, and rubbed some perchloride of mercury into her mouth and private parts. She consequently suffered all the agony of severe mercurial poisoning with sloughing of the mucous passages which came in contact with the poison, the *post-mortem* examination revealing a shocking state of things. The woman died after nine days, and traces of mercury were obtained from the liver and kidney.

(3) A small phial containing some fried Bengal gram was sent for examination, because a person who ate some of this suffered from severe vomiting followed by a metallic taste in the mouth. The gram was found to be intimately mixed with metallic mercury, and over thirty-two grains weight of the metal was mechanically separated from the stuff. Liquid mercury is said not to be a

poison, though there are a few recorded cases which show that metallic mercury sometimes does produce poisonous effects, as it did in this case.

(4) A hospital servant, having been dismissed for misconduct, drank off some corrosive sublimate lotion and died from the effects of gastro-intestinal irritation. Although the organs were separately removed and examined separately in the laboratory, the poison was found only in the stomach, colon and urine, none being found in the small intestines or kidneys. The curious distribution of the poison is noteworthy and the Medical Officer who forwarded the case offered the following suggestion as a probable explanation :— that violent peristalsis of the small intestines drove the poison into the large bowel where on account of the slower motion it was deposited, and that there was probably time for it to accumulate in the bladder while a rapid process of elimination carried it quickly through the kidneys. It was pointed out at the time that this curious distribution of the poison could be accounted for by a physical process independent of vital acts like absorption and excretion or peristalsis. Crystalline bodies diffuse readily through animal membranes; and the contiguity of the stomach and the colon would favour the passage of the poison by diffusion from one organ into the other. It might also diffuse from the small intestines into the peritoneal cavity, and so escape detection in the *small portion* of intestines which is usually sent for examination. Why then does it not escape altogether from the stomach also? Because the bulk of a soluble irritant like corrosive sublimate is *directly* absorbed into the tissues of the stomach, while other organs receive it in much smaller quantities *indirectly* through the general circulation; and the retention of the poison in the stomach is favoured by the altered state of the circulation through this organ brought about by the irritant action of the poison. Once the poison has diffused into the peritoneal cavity, it can taint the bladder and possibly its contents; while the kidneys which are usually covered by a thick layer of fat might, within certain limits of time and other circumstances, be protected and as in this case be found to be free from poison. There appears to me to be no reason why large amounts of a crystalline poison retained in the stomach and its walls should not diffuse during life into the transverse colon and into the peritoneal cavity. It certainly can

do so when introduced into the alimentary tract *post-mortem*; and I have already communicated a note on the subject to this Branch.

Colocynth.—A boy was reported to have died of cholera, and to clear the doubt a chemical examination was required. When searching the contents of the stomach in the laboratory, 10 seeds of the colocynth fruit were found; and from the mixed material was extracted a substance which gave the chemical re-actions of colocynthin. The *post-mortem* examination revealed intense irritation of the rectum, the portion of the alimentary canal upon which this drug specially acts.

Trichosanthes palmata.—A woman is said to have eaten the seeds of this fruit with suicidal intent, suffered from vomiting and purging and griping, and died collapsed. The fruit sent for examination was identified as that of the *Trichosanthes palmata*. No "alkaloid" was found in the viscera, and a portion of the fruit was found to be non-poisonous with a guinea-pig, the quantity sent being too small for further investigation.

According to Roxburgh this fruit is "regarded as poisonous and is employed for destroying crows," while a variety known as *T. amara* is used in Jamaica "as a rat poison." In the Bengal Dispensary it is stated that numerous trials were made with the fruit to ascertain "whether it had purgative properties, but without effect." It belongs to a natural order (*Cucurbitaceae*) which furnishes such strong purgatives as colocynth and elaterium. In this connection it is interesting to note that the edible vegetable-marrow, cucumber and water-melon, of the same natural order, belong to a species (*Cucumis*) which furnishes many fruits having strong cathartic properties; and it is said that cultivation has so modified their properties as to render these fruits edible, for even the cucumber in a less highly cultivated state is known to act as an aperient in some individuals. In the same way the fruits of several species of *Trichosanthes* are said to be in common use among natives, while the *Trichosanthes palmata* is credited with medicinal properties and is looked upon as a poison.

Mannihot utilisima has been the cause of accidental fatal poisoning in several cases which have come to my notice. The nourishing farina, known as tapioca, is prepared from the tubers of this plant; the expressed juice contains hydrocyanic acid, which is

dissipated by roasting, when the starch becomes fit for food. It is a plant which seems to be widely cultivated in Southern India, and appears to be a staple article of food in Travancore. The possibility of people being poisoned by incautiously eating the roots, before they have been subjected to the preparation necessary for volatilising the poison, is proved by the occurrence of these cases. This plant it is which supplies the deadly "mandioc" arrow poison of the South American Indians. Fresh samples of the root when cut give out the peculiar odour of prussic acid, and responds to the sulpho-cyanide test. [*Samples passed round for inspection.*]

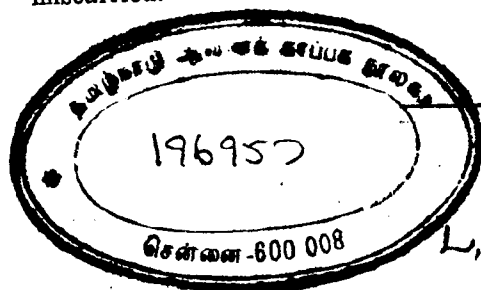
Abrus Precatorius.—Four persons suffered from vomiting and purging after eating raggi porridge into which "some red substance" had been put. The suspected substance sent was identified as the seeds of this plant. Seeds of the white variety were identified mixed with nux vomica seeds in another case of suspected attempt to poison. This is the well-known "Gundamani" employed in *Poisoning cattle by "Sui."*—This method of poisoning, though common in some parts of India, had not (as far as I could discover) been recorded in this Presidency until a suspicious case from the Anantapur district attracted my attention a few years ago. It was sent with a note to this effect:—Buffalo poisoned by introduction of some noxious substance beneath skin of neck with the aid of a pointed instrument. Dissection revealed a punctured wound of the part, which contained some foreign substance, which was unfortunately not sent for examination in this case. The attention of Medical Officers having been called by a Circular to this method of poisoning, I have since had several cases in which the agent of destruction has been verified as the "Sui." The mode of poisoning is as follows:—The seeds of the *Abrus Precatorius* are powdered into a paste with water and a small sharp spike about half an inch long (called "sui" or "sutari") is made from the paste and hardened by drying in the sun. A piece of stick about four inches long, which can be conveniently concealed in the palm of the closed hand, is taken and a small hole is bored at one end of it. In this hole is fitted the base of the pointed sui, and a blow is struck with it, when the protruding point of the sui enters the skin and remains there after the handle is withdrawn. The animal invariably dies in about 30 hours; much cedema is found at the seat of injury, with

marked injection of the vessels and hæmorrhagic points in the mucous membrane of the stomach and intestines, [*A specimen of a "Sui" was handed round for inspection.*]

Lastly a case of fatal poisoning by *Strychnine* seems worthy of record:—

The corpse of a woman was dragged out of a well, and as the cause of death could not be ascertained at the inquest, the body was conveyed to the local dispensary by the police; on getting ready for the *post-mortem* examination, a dead fœtus dropped out of the winding sheet to the astonishment of the by-standers, *post-mortem delivery* having occurred on the way unnoticed by the bearers of the corpse. There were no signs indicating that any attempt to cause abortion had been made, there were no signs of drowning, the fœtus was seven or eight months old, and its delivery was accompanied by total inversion of the uterus. Under these circumstances, the woman also being a widow, it was suspected that she died from the result of a drug taken to procure abortion and that her paramour had cast the corpse into the well to veil the cause of death. *Strychnine* was found in the viscera sent. The occurrence of childbirth after the life of the mother had become extinct, without the aid of art and indeed even after interment, has been recorded and avouched by many observers of established credit; the independent contractile power of the uterus, or cadaveric rigidity, being stated to be the chief factors in the production of this accident. So-called "Cadaveric spasm" also is known to occur at or after death by strychnine poisoning and to persist till true cadaveric rigidity comes on, disappearing only with it. In the present instance the body must have lain for about eighteen hours in the water, cadaveric rigidity had all but passed away at the time of examination (only the upper limbs being slightly stiff), and, no doubt, expulsion of the fœtus occurred by the pressure of the gaseous products of putrefaction which filled the abdomen. The possibility of this accident occurring is a point in obstetric jurisprudence which should not be forgotten when examining the dead body of a female alleged to have miscarried.

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