ON THE ETIOLOGY OF BUBONIC PLAGUE:

AN EPIDEMIOLOGICAL CONTRIBUTION.

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OBSERVATION of two epidemics of plague which have occurred at Sydney among whites and in circumstances of population, laws, customs, housing, and food which closely correspond with European conditions has led me, as well as all the members of the public health staff over which I have the honour to preside, to entertain opinions concerning the diffusion of plague which differ fundamentally from those commonly advocated. What the latter are can be most concisely shown by a quotation from the latest work which deals with this subject. I refer to "A Manual of Plague," by Major W. E. Jennings, I.M.S., and I choose it for the purpose not merely because it has been published during the present year but also because it is most carefully written and reflects with great accuracy the views as to the causation of epidemic plague which are all but universally held at this date. The quotation runs as follows: "Our measures for the prevention of plague should be mainly directed against what the strongest evidence points to as the most important etiological factors. The disease is mainly spread by human beings, infected clothing or effects, and ship-borne rats" (chapter x., p. 200), and that the teaching thus indicated is not confined to the English school is testified sufficiently for the present purpose by reference to the extraordinary pains taken to isolate every person who had in any way come into communication with the subject of that case of laboratory plague which recently occurred at Berlin.

The teaching drawn from the first of the epidemics at Sydney directly opposed those views; it was that neither direct nor indirect communication with the sick had borne any share at all in diffusion of the disease, that place-infection almost certainly had not operated to maintain it, and that the sole source of infection for man had lain in rats affected with it. The evidence on which these opinions rested was given in my official report¹ which probably has fallen under the notice of most of those who are specially interested in this subject; however, its contents were also summarised in an article which appeared elsewhere.² Its importance lay wholly in positive exclusion of human intercourse and of place-infection as factors in the epidemic prevalence, a point which one author alone has perceived.³ It lay not at all in the assertion that the sole source of the infection consisted in diseased rats which, though clearly seen by us to be justified, was but imperfectly supported by examples.

Our observations of 1900 have been repeated at two places and, as far as the reports permit judgment, with similar results—namely, at Brisbane, Queensland, by Dr. G. B. Halford⁴ during epidemics which occurred in 1901 and 1902, at the same city by Dr. B. Burnett Ham⁵ during a further epidemic which occurred in 1903, and at Port Elizabeth, Cape Colony, by Dr. G. J. Blackmore⁶ during the earlier part of an epidemic in 1901. All of these writers seem to have been able to exclude the influence of human intercourse and place infection.

A second epidemic began about 15 months after the first had ceased. It afforded the desired opportunity of repeating, checking, and extending the observations recorded in 1900. A result was that the latter were in every respect corroborated. My account of this second epidemic, together with many interesting and important observations by Dr. Frank Tidswell who directs the laboratories of the board, has

¹ Report on an Outbreak of Plague at Sydney, 1900, by J. Ashburton Thompson (W. A. Gullick, Government printer, Sydney, 1900).

² Journal of Hygiene, vol. i.

³ Kolle: Zeitschrift für Hygiene, 1901, p. 397.

⁴ Report on 117 Cases of Plague occurring at Brisbane in Sporadic Form (Briggs and Morecom, Brisbane, 1902).

⁵ Report on an Outbreak of Plague in the State of Queensland (G. A. Vaughan, Government printer, Brisbane, 1903).

⁶ THE LANCET, Oct. 11th, 1902, p. 984.

also been published and is now available to epidemiologists.⁷ I do not propose on this occasion, therefore, to offer any summary of its contents; suffice it to say that the opinions thus far referred to have been carried into practice. I do not isolate the sick but allocate wards to them which form part of the infectious diseases division of a general hospital; there they are treated under the same conditions as are cases of measles or chicken-pox in other and adjacent wards in the same inclosure; the only extra precaution which has been taken is that of rendering all the wards rat-proof. Neither do I segregate those who have been in contact with cases of plague (though to this contact with cases of primary plague pneumonia would form an exception), in accordance with an opinion which I have elsewhere expressed—namely, that "contacts" are not such persons, but those who have been closely associated with plague rats.⁸ Hence disinfection of clothing, &c., is rapidly done; only the cleansing of houses from rats and the rendering of them less liable to infestation in future are subjects of careful attention. These modifications of the method of managing epidemic plague which elsewhere is still thought necessary have been attended with a great saving of time, anxiety, perturbation of the public mind, and of money, and if the result may be referred to as evidential in relation to the soundness of our conclusions, it can be said to have confirmed in practice the teaching we drew from the epidemiological facts observed in 1900.

Another consequence of these improved conditions was that the business of managing the second epidemic proceeded quietly and steadily, without excitement on the one hand and without opposition to necessary measures on the other. Hence, it became possible to direct attention mainly to examining much more closely than before into the nature of the relationship which existed between cases of plague and plague rats—a matter which, with us, had come to take that position of prime importance which before had been occupied by the questions whether human intercourse and place-infection were in reality among the causes of epidemic bubonic plague. Connexion between the neighbourhood of plague rats and cases of plague was discovered very much more completely than in 1900 and especially precedence of the disease among rats over its occurrence in man was shown by the events noted in many separate localities to have been practically constant. Starting then, from these two latter observations which, for us, have all the weight of facts, I proceeded to inquire by epidemiological methods how communication of the infection from the rat could be commonly effected under the conditions of daily life.

In the rat plague is a septicaemia; certainly some special means are necessary to transmission of the infection from it to man. Now, we know that plague rats can deposit the virus on external objects with the nasal secretion and with the excreta; it is possible, therefore, that it might reach man in one of two ways—(1) by contamination of his food and (2) by casual contact with his skin. The former of these must be summarily dismissed from consideration, for there is practically no pathological evidence that man is infected by feeding. If it be possible, as analogy from experiments made with the lower animals suggests, at all events it has happened so rarely that this mode can have no significance in relation to epidemics. But the pathological evidence that the infection is commonly received by inoculation in the skin is direct, uniform, and clear; all but a small minority of cases in every considerable epidemic are of the bubonic form and all such cases are so caused. Consequently that inoculation is the result of casual contact with deposited virus is at this date very generally held to be the most important cause of epidemics, although (altogether contrary to my experience) deposition is usually ascribed to man, and but rarely, or in quite a minor degree, and always with hesitation, to plague rats. This mode of infection—inoculation in the skin by casual contact with deposited infection—is a possibility which, it may be admitted, has occasionally come into play; but what evidence is there that it is a factor of any practical importance in production of epidemic prevalences of plague? This is the question which I propose to refer to a test of practical experience.

The proof that the infection of plague is communicated to man by inoculation through the skin in the vast majority of

⁷ Report of the Board of Health on a Second Outbreak of Plague at Sydney, 1902, by J. Ashburton Thompson (W. A. Gullick, Government printer, Sydney, 1903).

⁸ Report, Sydney, 1900, p. 42.

cases has been well stated in the report of the Indian Plague Commission.⁹ It is, "that primary buboes practically always develop in connexion with lymphatics which originate in the skin." The constant preponderance of "inguinal" or "groin" buboes over others was also shown in the same paper to stand in relation to the larger area of skin which drains through the lymphatic glands of the groin. Analysis of returns which represented 5442 cases of bubonic plague showed that the ratio in which buboes occurred in the lymphatic glands of the head and neck, the axilla, and the groin respectively was nearly the same as the ratio which the skin areas draining through the lymphatics of those regions bear to each other.

The proportion in which buboes are met with in the groin always preponderates over that in which the buboes occur in other regions of the body; this was very early observed among Eastern races who habitually go barefoot. Before long, however, the same preponderance was noticed among Europeans (though in small number) who did not go barefoot, but the hint thus given seems to have been overlooked and (perhaps in consequence) assumed infectiveness of ground surfaces became a prominent feature in writings on the etiology of plague. But at Sydney in 1900 I was able to show that in no less than 73 per cent. of the 283 cases which exhibited buboes the latter were found in the groin although all the patients referred to were white, clothed in European fashion, and invariably shod.¹⁰ The validity of the banal explanation of the preponderance of buboes in the groin thus seemed to be rendered more than doubtful. But I had pointed out in the same report that the infection of plague was received by man, for the most part, within buildings, which seems also to have been noticed in India.¹¹ Now, if the infection be usually received by man in houses, &c., the objection raised above to the banal explanation of the preponderance of groin buboes must be revised, for there is a time of day when nearly all people, whites as well as others, go barefoot—namely, when they are going to bed or rising from it. At that time, therefore, infectious material deposited on floors, carpets, &c., by rats (or otherwise) would be more likely to come in contact with the unprotected skin of the feet than with that of any other part of the body. So that, although conjunction between a recent breach of the epithelium and deposited infection in the requisite state of activity cannot often happen, here is the opportunity for it at all events, and plague was not so frequently contracted by our people in either epidemic but that possibly those persons who exhibited groin buboes might have received the infection in that way. Let us try, then, to ascertain what the fact was. Does the record contain any cases in which infection resulting in groin buboes was probably not contracted by casual contact with deposited virus of the unprotected skin of the lower extremities? If it turn out that it does the hypothesis under examination will be very greatly weakened; it must, indeed, be discarded as an explanation of epidemic bubonic plague.

Among the 139 cases which constituted the epidemic there were 106 in which buboes occurred in the groin. But it is necessary to speak more exactly. Our present concern must be with primary buboes, since these alone indicate the region of skin through which the virus entered; and in order to leave no room for possible confusion between primary buboes and secondary glandular swellings it is desirable to take into account only those cases in which the groin bubo was solitary. Then the expression "groin bubo" is loose. A bubo in one of the oblique set of inguinal glands indicates infection through the skin of the lower part of the trunk. Buboes in one of the vertical set of femoral glands alone indicate infection through the skin of the feet or of other part of the lower extremity. An examination intended to discover whether there were buboes betokening infection through the skin of the lower extremity must be limited, therefore, to cases which exhibited solitary buboes in one or other gland of the femoral chain. The total number of such cases was 60, but the majority are not available for the present purpose. Thus the place at which the infection was taken could not be determined in 16 of them; either the inquiries made failed to reveal any clue to it or else the evidence for infection at home and at work seemed equally cogent. These 16 must be thrown out, therefore, for they (or some of them) may have been infected at their place of

residence, where they were admittedly exposed to the risk ascribed to bare feet which is now under investigation. In 25 other cases it was considered that the patient did receive the infection at his place of residence, so that these too must be thrown out for the reason just given. In three others the place of employment probably was the place of infection but the details left room for some slight doubt; these are excluded by way of precaution. Three more were Chinese, concerning whom either information of the required kind could not be got at all or it was untrustworthy. Lastly, one was a woman whose movements could not be traced. So that 48 of the 60 have to be discarded. There remain 12 patients who had solitary femoral buboes and who are confidently adjudged to have received the infection away from home and therefore while fully clothed. Of these patients eight inhabited houses in neighbourhoods which were distant or even remote from the infected area and which were at all times free from suspicion of infection with plague (yielded neither indigenous cases nor plague rats); and while, on the one hand, 11 of the residences bore no traces whatever of infestation with rats (the twelfth alone having exhibited some slight signs), on the other hand, the places at which the patients were employed were badly infested and had yielded dead rats in number in ten instances at least. It is certain that these persons all fell into danger at their place of employment and apparent that they were not infected at home. At their employment they were fully clothed; their lower extremities were protected by boots, socks, and trousers, or (in one case) by stockings and petticoats, while their hands, arms, necks, faces, and in some cases, no doubt, their chests too were uncovered, exposed, and in no way protected from casual contact with deposited infection and therefore they should, *ex hypothesi*, have exhibited cervical or axillary buboes. In fact, however, they none of them did so. They were all of them inoculated in one or other lower extremity—in that very area of skin which beyond all doubt was well protected from casual contact with deposited infection. These 12 cases form but a small proportion of the 139 of which the second epidemic consisted, but the necessary rules under which they were selected, in circumstances where great caution was indispensable, led to rejection of several others which, were they to be cited as examples of a fact otherwise ascertained, might have been included. These 12 persons then were not infected by feeding for the reason drawn from the pathology of plague which has already been mentioned; it has just been shown that they could not have been inoculated in the lower extremity by casual contact with deposited infection and none of them had had any communication with preceding cases of plague. How, then, did they come to contract the disease? There is, in my opinion but one plausible—nay, but one possible—explanation. Inoculation in that situation and in those circumstances can have been brought about only by a suctorial parasite. On the whole the parasite which is best fitted to fulfil all the requirements of the case is the flea.

Several *a priori* objections have been raised during two or three years past to Simond's hypothesis of the flea¹² which, I may be permitted to add, I had myself accepted as a working hypothesis before I had had any practical experience with epidemic plague on the ground that it reconciled a much greater number of discrepant and even contradictory observations which had then been recorded than any other suggestion which had been made.¹³ I conclude, therefore, by mentioning first that the indirect objections raised by Nuttall¹⁴ on grounds furnished (a) by his unsuccessful experiments on the transmission of plague from animal to animal by means of bed bugs and (b) on the rapidity with which the bacilli of several diseases, among which plague was not included, lost their virulence after entering the stomach of the flea, have recently been met. Successful experiments on the transmission of plague from animal to animal by means of fleas have now been reported by Raymond and Gauthier working at Marseilles¹⁵ and by Elkington working at Bombay,¹⁶ both, be it remembered, thus confirming experimental results originally reported by Simond himself.¹⁷ Secondly, that the objection raised by

¹² Annales de l'Institut Pasteur, tome xii., p. 625.

¹³ Report, Sydney, 1900, p. 40.

¹⁴ Johns Hopkins Hospital Reports, vol. viii., pp. 17-21.

¹⁵ Abstract in Comptes Rendus de la Société de Biologie, December, 1902, tome liv., p. 1497. The paper is printed at length in La Revue d'Hygiène, May, 1903, p. 426.

¹⁶ Australasian Medical Gazette, August, 1903, p. 348.

¹⁷ Loc. cit. sup., p. 673.

⁹ Indian Plague Commission, paragraph 175.

¹⁰ Report, Sydney, 1900, p. 40.

¹¹ Indian Plague Commission, paragraphs 221 and 298.

Galli-Valerio,¹⁸ with him Nuttall,¹⁹ on insufficient grounds—namely, that “rat fleas” do not bite man—has also been met by direct experiments recorded by the observers just mentioned, as well as by Tidswell at Sydney. The species *pulex fasciatus* (Baker) and *pulex pallidus* will freely bite man and can even live on his blood alone for several weeks. I take this opportunity of pointing out that Tidswell²⁰ first observed *pulex pallidus* to be a species which most commonly infests the rats of the Australian coast line, an observation which was found to hold good with rats in India after he had communicated it to correspondents at Bombay.

The question which was asked above concerning inoculation by casual contact with deposited infection must now be put with reference to transmission of the infection by the flea. The former was there admitted to be a possibility which sometimes, in all likelihood, had been realised; but the chances against conjunction of the necessary circumstances are so many that it cannot be regarded as the cause of epidemics. The latter, also, appears to be a possibility; but does it take practical effect often enough to constitute that cause of epidemics which otherwise is still unrevealed? The answer cannot be drawn from laboratory experiments; it can be furnished only by epidemiological investigation and the evidence must always remain indirect. Much to the purpose has been recorded in my report on the epidemic of 1902.²¹ We are of opinion that the hypothesis of the flea fully explains its observed course but the evidence is too lengthy for inclusion in the present communication.

Sydney.

A Mirror

OF

HOSPITAL PRACTICE, BRITISH AND FOREIGN.

Nulla autem est alia pro certo noscendi via, nisi quamplurimas et morborum et dissectionum historias, tum aliorum tum proprias collectas habere, et inter se comparare.—MORGAGNI *De Sed. et Caus. Morb.*, lib. iv., Proœmium.

ROYAL SOUTHERN HOSPITAL, LIVERPOOL.

FIVE CONSECUTIVE CASES OF INTESTINAL OBSTRUCTION
SUCCESSFULLY TREATED BY OPERATION.

(Under the care of Mr. G. P. NEWBOLT.)

CASE 1. *Intestinal obstruction produced by a large gall-stone.*—A married woman, aged 54 years, was admitted to the Royal Southern Hospital, Liverpool, on August 4th, 1902, suffering from intestinal obstruction. She was a big, stout woman, weighing 18 stones and measuring 44 inches round the waist and was the mother of nine children, the youngest being aged 12 years. Her bowels were moved naturally eight days before admission and again two days later after an aperient. No flatus had passed for six days but an injection had brought away faecal matter. She had been vomiting brown, foul-smelling fluid for a week past, her tongue was dry, her pulse was 92, and her respirations were 28. The onset of the attack was sudden and the pain spread all over the abdomen. She had had similar, only slighter, attacks previously but had never been jaundiced and had never had pain in the region of the gall-bladder. Examination per rectum revealed nothing. The abdomen was somewhat distended but there was very little tenderness and the walls moved freely with respiration. When seen at 10.30 A.M. shortly after admission there was no vomit for examination and it was decided to wait for a while in order to get a specimen of the vomit before opening the abdomen. This was done as although there was some distension yet the woman did not seem to be in the distress which is usual when intestine is nipped or tied down. Her obesity did not invite laparotomy unless absolutely necessary. The trouble had lasted a week and she admitted having had similar attacks which she had got over. However, some hours later she had an

attack of faecal vomiting and there was no object in further delay. At 8.30 P.M., the patient being under chloroform, Mr. G. P. Newbolt opened the abdomen in the middle line with a four-inch incision, its centre being at the umbilicus. Some coils of distended small intestine escaped and on passing the hand into the abdomen a hard lump was at once felt in the intestine occupying the left iliac fossa. On drawing it up to the surface a large gall-stone was found to be blocking the gut. All the intestines were now returned except the portion containing the stone, and the abdomen having been shut off with sponges the intestine was tightly held on each side of the stone whilst the latter was removed through a longitudinal incision in the gut. The stone was squeezed out and the incision was closed by two layers of interrupted suture, one taking the edges of the wound and the other (a Lembert suture) prolonged well over each end of the incision. The abdomen was closed with through-and-through silkworm-gut sutures and the patient left the table with a pulse of 108 which soon fell to 92. She was kept under opium. Flatus was passed on the second day and she made a perfect recovery, leaving the hospital five weeks after the operation. The stone was egg-shaped, one end being fractured, and this lay uppermost or nearest the duodenum. It measured two inches in length, one and a half inches in diameter, and three and a half inches in circumference; it weighed 303 grains when dry. Around the broken end was a ring of dark pigment which looked as if this portion of the stone had been gripped by encircling mucous membrane and stained. The fracture was apparently a recent one. Mr. Newbolt saw the patient on June 5th last and she was perfectly well.

CASE 2. *Intestinal obstruction produced by a band.*—The patient, a single woman, aged 31 years, was first seen by Mr. Newbolt in consultation with Dr. W. Murray Cairns on July 16th, 1902. Her previous history was good: she had never had any digestive trouble but had had scarlet fever six months before and on examination a mitral murmur was present. On July 14th, two days before Mr. Newbolt saw her, she ate an ordinary midday meal of beef, &c., and one and a half hours later she was seized with acute pain in the umbilical region which doubled her up on account of its severity and made her vomit. Her bowels acted twice after the pain began, the second motion being attended with a good deal of griping. She was not collapsed; her pulse was 60, her temperature was normal, and her illness simulated an attack of colic. Mustard poultices were applied to the abdomen. On the next day she was no better; there were constant pain, vomiting, and no passage of flatus. Dr. Barr saw her at this stage of her illness and diagnosed obstruction by band. On July 16th, when Mr. Newbolt was called in with a view of operative proceedings, the patient was still vomiting, no flatus had been passed, and slight distension of the abdomen appeared; the vomit was becoming feculent and the pain was constant. On examination her pulse was 70 and her temperature was normal; her face was of a good colour; the urine was scanty; the abdomen was not moving with respiration and was tender all over with a marked tympanitic area. Mr. Newbolt had her removed in an ambulance to the Royal Southern Hospital. Here he opened the abdomen in the middle line by an incision four inches long with the umbilicus at its centre. Distended small intestine at once presented and was allowed to escape. Contracted gut appeared in the left iliac region and tracing it back Mr. Newbolt came upon the seat of the obstruction which was low down in the ileum. A thin white fibrous band exactly like a vocal cord was attached at one end to the mesentery; it encircled the gut and was again attached to the mesentery on the opposite side. It was a separate structure and not a thickening of the peritoneal coat; there was no trace of old peritonitis and the band looked as if it might be of congenital origin. It was cut away close to its origin and insertion. The abdominal wall was closed in layers and the patient was given a saline injection per rectum. Flatus was passed on the third day and the patient made a good recovery. During convalescence she had an acute urticarial rash all over the body followed by some bronchitis. She went home five weeks after the operation. Her further history, for which Mr. Newbolt is indebted to her brother-in-law, is interesting. In September, 1902, she went to Palestine and did well till early in November when whilst at a hotel in Damascus a recurrence of the symptoms of obstruction suddenly supervened. She was taken to the Scotch Mission Hospital in

¹⁸ Centralblatt für Bakteriologie, Band xxvii., p. 1., Band xxviii., p. 842; Journal of Tropical Medicine, vol. v., p. 33.

¹⁹ Op. cit., p. 65.

²⁰ Report, Sydney, 1903, p. 72.

²¹ Op. cit., Paragraphs 295-300.