

A HISTORY OF TROPICAL MEDICINE

Based on
THE FITZPATRICK LECTURES

Delivered before the Royal College of Physicians of London 1937-38

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VOLUME II

CHAPTER XI

CHOLERA

1. GENERAL ACCOUNT

Of all pestilences cholera is perhaps the most awe-inspiring; it may run so rapid a course that a man in good health at daybreak may be dead and buried ere nightfall. Again and again has the fear of cholera been the beginning of sanitary wisdom. Incidentally, Public Health legislation in Great Britain owed much in its earliest days to cholera. It was not a medical man, but a lawver-sanitarian, Sir Edwin Chadwick (1800-90), who was chiefly responsible for initiating the public health era, and it was largely through his efforts that the Public Health Act of 1848 came into existence. He was helped in attaining his ends by outbreaks of cholera in England which brought home to Englishmen the dangers to which their fellow-countrymen and others abroad, in India and the Colonies, were exposed at all times. A General Board of Health was established but met at first with no little opposition because it conflicted with vested interests and because local authorities resented central control-we see the same in some colonies to-day. The Board survived for ten years only, but did good work, for, as one result, Liverpool appointed a Medical Officer of Health, and was the first city to do so, and soon afterwards John Simon was appointed to the corresponding position for London.

It was cholera that helped the Sanitary Act of 1866 to become law and in consequence a more sanitary atmosphere began to prevail. A cholera survey yielded much information as to the sanitary state of many districts and the means employed to prevent infection entering from abroad.

In Chadwick's day the surroundings amid which the poorer people lived were highly insanitary and favoured the spread of cholera when once it was introduced. Seeing that cholera is one of those diseases which formerly were much more widespread and occurred commonly in temperate climates—but is now of a limited distribution and mainly a disease of warm climates—it will be

instructive to glance for a moment at the conditions in England a century or so ago when the disease was raging there and we shall see that they were similar to those associated with the disease abroad at the present time, and it is but natural to infer that measures which have resulted in freeing Great Britain from cholera in the past will, if applicable and applied abroad, mutatis mutandis, have the like happy results.

In the eighteen-thirties cholera was raging in Bethnal Green and 'summer-houses' in the gardens of weavers were occupied as dwellings, though situated on undrained soil and with mere holes in the ground for privies, while the water-supply was obtained from surface wells into which the refuse and ordure from the houses gained entrance. Larger houses were tenement dwellings in which an entire family might live in a single room, and since the ordinary privies were very soiled and dirty and, moreover, access to them implied exposure, the excreta were often kept, temporarily at least, in the room until the atmosphere became almost unbearable.

A few years later, in 1847, Lambeth had common sewers, but most of the houses had no drains leading to them, and even the large houses had cesspools only. Further, many of the so-called sewers were little more than elongated cesspools, the water-supply was inadequate to flush them, they might overflow and their contents come up through untrapped gullies. In October the following year the Common Council of the City of London met and a recommendation of the Commissioners of Sewers was passed after much discussion, that a medical officer of health should be appointed for the City and Liberties of London forthwith to hold office till January, that was for three months. He was to be paid £150 for this. Who was appointed we have not been able to find out, but a few days later the Lord Mayor reported that every effort was being made by the Commissioners of Sewers to enforce the sanitary recommendations of the Board of Health and that he had received a letter from Dr. J. A. Paris, President of the Royal College of Physicians, informing him to his great satisfaction that the College had appointed a Standing Committee to consider the subject of cholera.

It is not within the scope of these lectures to discuss whether the $\chi o \lambda \xi \varrho a$ of Hippocrates (460–370 B.C.) or the disease so called by Celsus, who lived in the reign of Tiberius, or, again, that described some eighty years later by Celius Aurelianus, by Aretæus of Cappadocia and other ancient medical writers was the same as

Cholera asiatica (should it not rather be named indica?) of modern times. We may certainly take it that ξηρα χολέρα of Hippocrates had nothing to do with cholera as we know it, for the term was used for obstinate obstruction, not intractable diarrhea. The etymologically corresponding Latin term cholera sicca is used in modern text-books for a very fatal type of the disease in which the patient succumbs to an overwhelming dose of poison and dies in a few hours without any reaction or symptoms of diarrhea, vomiting.

Apart from Hippocrates and Roman medical authors, it is mentioned in Chinese and Hindu writings. The first European writer in more modern times to describe it was Bontius or Bonsseus, a physician to the Dutch East India Company, in Batavia in 1629.

Medical records of historical value may be taken as starting from what is known as the First Pandemic of the early years of the nineteenth century, though the earliest description of the disease in epidemic form was probably the outbreak which devastated Ahmed Shad's military forces in A.D. 1438.

Four such pandemics occurred during the century: the first from 1817-23, the second from 1826-37, the third from 1846-63, and the fourth from 1865-75. The first is always spoken of as starting in 1817 because reliable information of the dates of its diffusion begin at Kishnagur on the Hooghly in May that year; there is little doubt, however, that in 1816 cholera was already becoming endemic in Behar. There is no need to describe its extension in detail or its ravages in the different districts attacked; suffice it to say that by 1818 infection had crossed the borders to invade Ceylon in 1819, then Mauritius and on to East Africa in 1820, the Philippines, China and Japan in 1822. In another direction it passed to Arabia in 1821, thence, in 1822, to Persia and Syria, and in 1823 Russia. Briefly, in this period it had extended over a territory of nearly a hundred degrees of longitude. from Nagasaki in 147° East to the coast of Syria in 52° East, and upwards of sixty-seven degrees of latitude, from Bourbon (now known as Réunion) in 21° South to Astrakhan in 46° 21' North. This pandemic, it will be observed, did not cross the frontiers of Europe, though it came very close.

Professor Major Greenwood, when speaking of the difference between this pandemic of 1817 and outbreaks of the three preceding centuries in his work on *Epidemic and Crowd Diseases*, a work full of interest, of instruction and of stimulating ideas, says: "This difference may be summarized in a phrase as a difference

in dispersiveness" and quotes the following passage from Georg Sticker's Abhandlungen aus der Seuchengeschichte und Seuchenlehre (1912):

Was neu in der Geschichte der Indischen Cholera war und was die Gemüter dort wie hier mit Recht erregte, war die weitere Nachricht, dasz jene verherrende Seuche nicht mehr örtlich beschränkt blieb und zur gewohnten Jahreszeit an vielen Orten zugleich auftrat, sondern sich in Bewegung setzte, unter einem geheimnisvollen Antriebe zu wandern begann und ohne Rücksicht auf die Jahreszeit, sich an den Menschen verkehr von Menschenleben fordernd. Noch in selben Jahre einen groszen Teil der Halbinsel erobernd kann sie schon im folgendem Jahre über die Grenzen Vorderindiens hinaus, durchzog ostwärts und westwärts die nordlichen Länder Asiens, berührte im Jahre 1823 im Kaukasus und am Volgaflusz die Grenzen Ruszlands und bedrohte so

Europa.

[What was new in the history of Indian cholera and rightly caused apprehension there as well as here was the further intelligence that this destructive epidemic [of 1817] no longer confined itself to a particular area and at the accustomed season of the year appeared simultaneously in several places, but set itself in motion, under the influence of some mysterious impulse began to travel, and, without paying attention to the season of the year, attaching itself to the lines of human intercourse, spread widely in various directions, exacting everywhere hecatombs of victims. In that very year a great part of the peninsula was conquered; in the following year cholera crossed the frontier of Upper India and travelled east and west through the northern lands of Asia. By 1823 cholera, in the Caucasus and on the Volga, reached the frontiers of Russia and thus menaced Europe.]

In order to retain a chronological sequence we will here state the general opinion relative to this disease at the time of the first pandemic as given by Leonard Rogers. Neither the proximate cause nor the remote causes were known but negative propositions were affirmed that it was not due to improper food nor to irregularity of seasons, though climatic influences, especially a moist east wind, might play some part. The observed route of extension along rivers was ascribed to commerce. Since removal of inhabitants to a new site often ended an outbreak the disease was believed not to be contagious, and quarantine measures were regarded as useless.

Reginald Orton held that cholera was "due to deficient nervous action brought about by a lessened aeration of the blood, which is itself dependent on unsettled weather consequent on deficient electrical fluid in the atmosphere"—a view about as enlightening as Abram's box.

The second pandemic, 1826-37, also started in India and after the lapse of only three years from the ending of the first. It extended over an even larger area and in several directions: From India to China, from India to Russia, and thence successively to Poland, Germany, Great Britain and France, to Belgium, Holland and Norway. It was of this epidemic as Heinrich Heine saw it in France that he wrote in a letter dated 9th April, 1832, how on 29th March, the night of mi-carême, a masked ball was in progress when one of the harlequins collapsed with cholera. In a short while carriage-loads of people were hurried to the Hôtel Dieu to die, and to prevent a panic they were thrust into rude graves without even removal of their dominoes. Soon the public halls were filled with the dead, sewed up in sacks for lack of coffins. Long lines of hearses stood en queue outside Père Lachaise. The rich fled from the town and over 120,000 passports were issued at the Hôtel de Ville.

Other parts of the Continent appear to have become infected vid Great Britain, rather than directly; thus Portugal was almost certainly so invaded and after that Spain, Italy, Sicily and Switzerland. From Britain it is probable that Canada was invaded and thence the United States, Cuba, South and Central America. This pandemic died down in 1837 and for the next decade no records of cholera in Europe, Africa or America are found.

During this extensive outbreak quarantine was in force in various countries but did not avail to prevent the spread of infection; a triple cordon of troops was instituted and was found equally useless; in Spain the death penalty was imposed for infringement of rules, but it, too, proved futile. At this time the air-borne theory of propagation gained more adherents. The influence of climatic variations and chills was believed to play a large part in initiating outbreaks, but the capriciousness of the distribution was thought to be due to generation of a poison in the soil which "checked perspiration, congested the bowels and led to inflammation." Contagion was not believed to take much share because it was noticed again and again that those attending on the sick in hospital did not often contract the disease. During this period it may be noted from perusal of reports of cases that copious bleeding found fewer advocates; also intravenous injection of salines were first tried-by Latta and Mackintosh (see later).

The third pandemic, 1846-63, and the fourth, 1865-75, do not call for any detailed description, the accounts of them would be very like those already given. Suffice it to say that during the third, infection spread over the whole northern hemisphere and

to 25° South in the Old World, and to 30° South in the New World. The fourth is particularly noteworthy, first on account of its course, for it passed to Southern Europe by sea from the coast of Arabia instead of the former route viâ Afghanistan, Persia and Russia, and second by reason of its unexampled rapidity of spread, taking only a few weeks to reach Europe. These two are interconnected, the rapidity of spread being brought about by the shorter route to travel. Since then there have been no such widespread outbreaks.

Sir James Ranald Martin has some interesting remarks on these epidemics and particularly in comparing the rate of spread of the infection in the second and third. That of 1817 started on the shores of the Ganges, and then passed to the Indus and Euphrates, Nile, Danube, Volga, St. Lawrence and the Mississippi. Since then, he states, epidemics "have always originated in and issued forth from India, but not, to my knowledge, been imported into India by ships from infected countries." Martin held that "communication from person to person was very rare and even when the disease is presumptively contagious, it cannot be localized by quarantine."

The following table shows the spread and its rate in the outbreaks of 1830 and 1847, as regards Russia, Europe and Great Britain.

| | | | | 1830-1. | | 1847-8. |
|--------------|-----|----|-------|--------------|-------|----------------|
| Astrakhan | * | | 1830, | 20th July | 1847, | June |
| Moscow | | | ,, | September | ** | 18th September |
| St. Petersbu | ırg | | 1831, | 16th June | 1848, | June |
| Berlin . | | | 22 | 31st August | 53 | June |
| Hamburg | * | | 2.9 | October | ,, | September |
| Sunderland | | 96 | 2.9 | 24th October | 22 | 4th October |
| Edinburgh | | | 1832, | 22nd January | 33 | 1st October |
| | | | | | | |

It is worthy of note that each successive 'pandemic' reached a wider distribution than its immediate predecessor, and interesting also to see how certain districts, even countries, escaped. Thus, it did not invade Australia or the Pacific Islands, nor East Africa south of Delagoa Bay, the Cape, the interior and southern parts of Africa to the Sudan, parts of the West Coast of Africa, or St. Helena; in South America, the Falkland Islands, Tierra del Fuego, Patagonia and Chile; North America above the fiftieth parallel, the Bermudas; in Europe, Iceland, the Hebrides, Shetland and the Orkneys; Russia north of the sixty-fourth parallel. No explanation altogether satisfying is found for these exemptions. We can only affirm that India was the home of cholera and that there are certain parts of the globe into which the virus has been

introduced, and more than once, by traffic, but which have, nevertheless, not become the sites of outbreaks. This would seem to point to local peculiarities favourable or inimical to endemicity, as Hergt formulated a hundred years ago (1838). Elevation was thought to play a part, but this per se does not protect. Though, generally speaking, there is a diminution of disease with altitude and high places may remain free when the plains below are suffering severely, nevertheless it may occur at all altitudes and present the same symptomatology. Again, saturation of soil with retention of decomposing organic matter has been held to favour the prevalence and spread, and that diffusion followed the course of rivers was noted in India in the epidemic of 1817 and was observed to progress up-stream as well as down, and so was not ascribable to infection of river water used for drinking. We know now that this is explained by traffic routes.

In this connection mention may be made of Radcliffe who in the middle of last century reported, relative to outbreaks of cholera in England, that

the state of the soil and the degree to which it was charged with moisture and decomposing organic matter, especially excrementitious, has been held to exercise an important influence over the localization of the disease, while over thirty years earlier Gendrin, speaking of Paris outbreaks, noted as "the chief general predisposing cause"... the crowding of the inhabitants along the river banks.

The influence of seasons and weather we cannot now consider in detail for their importance cannot be regarded from the historical aspect; attention in this respect may be directed to the investigations of Sir Leonard Rogers who compared outbreaks in various parts of India over a period of forty-five years with the rainfall in those areas. He found that all but one of forty-one epidemics had been preceded by failure of the rains; also that an unusually early rise of the absolute humidity favoured early recrudescence or spread, and on these lines correct forecasting of epidemics had been made. As regards rainfall, however, two points, apparently opposite, may be noted. In some places, for example Hindustan and at the junction of the Jumna and the Ganges and in Nepaul, epidemic outbreaks, if occurring at unusual seasons, have been found associated with sudden and heavy falls of rain; in others, as in Bangalore in 1874 and in other parts of the Deccan, unusually copious rains seemed to bring the outbreak to an end. Possibly in the former case the rain helped to disseminate the infection, in the latter to wash it away. The character of the soil is, therefore, probably a factor of importance.

The actual type of disease appears to be unaffected by latitude or longitude, climate and so forth, but the fatality rate differs in different epidemics; thus, in India in the first pandemic it varied between 20 and 40 per cent. among European troops according to the locality; in 1831–8 in the Bombay Presidency it ranged between 33 and 50 per cent. Like other infections diseases, cholera outbreaks may be at one time mild, at another severe, whether due to less concentration of the poison or to climatic or geographical influences cannot be stated.

Individual predisposition, however that may be explained, and peculiarities of race and nationality, food, habitations, mode of life, may each play a part. It has been widely, in fact generally, held that the negro race is very susceptible; this has been recorded by Christie in East Africa, by Dauban in Mauritius, Petit and Vonson in Réunion, Walther in Guadeloupe, and Aschenfeld in Brazil. On the other hand, again and again the relative immunity of the Sepoys in India has been observed, by Cunningham and Bryden among others, although in those who are attacked the type of disease differs in no way from that seen in Europeans. The different modes of living, social conditions, density of population doubtless have some influence, for in certain parts of India, Assam, Siam and Burma where the life of the poor is a hard one the natives suffer more than do the Europeans.

As we did in the cases of the first and second pandemics we may here state briefly the general view held at the time of the 1840-9 outbreak. It was the worst recorded up to that time; over a million deaths from it were said to have taken place in Russia between 1847 and 1849, and 53,293 in England. Snow's views were that there was a specific poison present in the excreta of patients which set up the disease when particles were ingested by others; that these particles multiplied in the second patient who similarly conveyed the infection to others, perhaps many if the "poison gained entrance to a drinking water supply." Budd of Bristol suggested that there might be a special living organism, fungal in nature, which multiplied in the intestine and gave rise to the symptoms, and was disseminated in food and water, and consequently prevention would be attained by disinfection of the evacuations of the patient and safeguarding the water-supply. At the same time Drs. Baily and Gull reported to the Royal College of Physicians that spread of cholera was independent of contact between sick and healthy and that in their opinion infection was probably wind-borne.

We are now in a position to sum up the views held in the

middle of the nineteenth century and the following decade (1850–60) as to the causes and mode of propagation of cholera; they were an attempt to effect a compromise between the two directly opposite opinions as to its contagiousness or non-contagiousness, and the outlook of the day was pessimistic. These views may be brought together under the following twelve propositions, based on Ranald Martin's observations.

1. Cholera is produced by a special poison of eastern or foreign origin, certain local conditions, and predisposition in the inhabi-

tants being necessary.

2. Diarrhea, dysentery and other forms of flux precede the true epidemic and prepare the people for its inception. [These might be early cases not very severe, or, as in the case of the Broad Street Pump outbreak in Westminster in 1854, a non-specific enteritis might be prevailing, true cholera developing when by some chance the vibrio enters upon the scene.]

3. After subsidence of an epidemic, mortality from fluxes never entirely recedes within its former limits. [That is, residual

cases would be seen and possibly carriers.]

4. People at home were more ready for attack by disease of a flux character, perhaps owing to extension of the great town system. [That is, there would be more crowding and bad sanitation.]

- 5. Thus, it might be smouldering in England but the flame causing conflagration might be brought from the East; "the local insalubrity amidst and around us requires the combined influence of a certain atmospheric condition to produce the pestilence." The 'terrene' and 'the atmosphere' are inert when apart, but when they meet they become the 'shears of Fate' and true cholera results.
- 6. Meteorological conditions such as favour chemical decomposition of organic substances and so lead to contamination of the air, assist the spread of cholera; thus high barometer, low temperature (60° F.), small rainfall, still air and overcast sky. [But, that this was rather of the arm-chair type of argument was indicated by the observation that under similar atmospheric conditions some localities would escape while neighbouring localities were suffering severely; even neighbouring streets might be attacked while others remained free. Further, in places abroad, meteorologically all the conditions spoken of as suitable might be present and an outbreak be expected, yet none would occur.]
- 7. The disease would show a tendency to linger in certain districts, or, after attacking a place at the beginning of its visi-

tation, it might again return before its close, after an interval during which the district might be free of it. Or, later outbreaks might start from the same place, an "additional proof that local circumstances have great influence in determining its seat." [Carriers were unheard of in those days, though, as we shall see later, the carrier question of cholera does not rest on such firm ground as the typhoid carrier.] Thus, Acland noted, when writing of cholera in Oxford, that "those yards and streets in St. Thomas's parish, with one exception, which had been attacked with cholera in 1832 and 1849, were visited by the disease in 1854."

8. Local causes of insalubrity were universally considered necessary to the evolution of an epidemic, by producing a low

state of general health.

9. Dr. Carpenter's zymotic theory was coming to the fore, that the special poisons of smallpox, scarlatina, typhus, cholera and so on were capable of exciting each its peculiar fermentation in the blood "already charged with organic compounds in a state of retrogressive change."

10. The "exciting cause was more likely to be simply toxical, because new arrivals from a pure atmosphere and in sound health are often prone to suffer after a brief exposure to the epidemic influence before any predisposing condition of the blood could

be set up."

11. Pettenkofer's view was that the nidus was not in the air or water, but in the soil itself. The subsoil, if damp and porous, was readily penetrable by decomposition products of human and animal excrement. In such a soil and in the presence of this peculiar organic matter the cholera poison is generated. The ferment setting up the decomposition of which the cholera poison is one of the products is the matter of the dejecta of cholera patients. The germ-bearing excrement becomes finely divided and cholera miasma is produced.

12. Farr, Acland, Sutherland, and Snow favoured the idea of impurity of water, though they seemed to feel that impure water alone would not constitute a necessary factor of cholera,

nor would mere odour, 'privy odour' as it was termed.

To recapitulate, we may say that cholera was probably not known in Europe before the nineteenth century; three times it spread from India to Russia and Europe overland, once by Mecca pilgrims to Egypt (see later), and thence to Marseilles, whence in 1865 a woman introduced the disease into Paris. The outbreaks of 1865, 1873 and 1884 came to France by way of the Mediterranean, and in the last the route was traced from Alexandria to Naples, Marseilles, Toulon, Nantes, Paris and Spain and in the following year reappeared in Toulon. Before we speak of more recent outbreaks we must tell of measures taken to cope with the disease by certain Commissions appointed from time to time to consider the question and give advice.

In 1866 a Conference was held at Constantinople but did not accomplish very much beyond stabilizing, as it were, the information already acquired. The members found that India was the permanent home of cholera, that the disease was propagated by man with the rapidity of his own movements, that water and food might serve as vehicles, and that the most potent of all causes was the movements of pilgrims who tended to develop and propagate it. There was no evidence that animals could spread infection, at the same time it was only rational to regard them in certain conditions as under suspicion. The Conference supported the conclusions of Baily and Gull (v.s.) and recommended measures of general cleanliness, fresh air, avoidance of overcrowding, disinfection of buildings where cases occurred on shore, and of merchandize or ships if cases occurred on board.

The reasons why more was not accomplished at this Conference were probably the differences of opinion at that time as regards the mode of infection, and in consequence the difference in quarantime measures proposed by different countries, arbitrarily made and independently carried out, which resulted in sudden interruption or stoppage of traffic, not only personal but also commercial.

A second International Sanitary Conference was held in Vienna in 1874 to draw up quarantine regulations which would be generally acceptable. The subject of cholera was debated under four heads: First, its origin, nature and infectious character; second, quarantine matters proper; third, the establishment of stations for the study of cholera, whether these should be temporary or permanent, where they should be placed, whether there should be an International Central Committee to consider the results of investigation. and, if so, whether this should be temporary or permanent; fourth, whether the quarantine rules should be such as would militate against cholera only or against other epidemic diseases also. The conclusions as to quarantine are too important to be cursorily mentioned and will be dealt with later when we consider historically the part played by quarantine measures in prophylaxis; regards the other points considered, the conclusions reached were that Asiatic cholera spontaneously developed in India [i.e. was its home, or endemic centre], and that it was not endemic except in India; that it can be transmitted by man coming from a place where the germ already exists and that transmission may be by the personal effects, as soiled linen, or be carried in these effects if they are shut away to prevent free contact with air. Further, the infection may be propagated [transmitted?] by drink, especially by water, and by merchandise under certain conditions. The following general preventive measures were recommended: Inspection of ships from infected ports, detention of infected persons, disinfection of the clothes and effects of the patients and passengers and of the cargo after the sick have been removed to hospital.

In 1875, a year after the Vienna Conference, another was held at Brussels to consider the Prevention of Cholera. The outcome of the deliberations was that the "cholerigenic miasma spontaneously developed in certain conditions" in India, notably in the Delta of the Ganges and the low-lying districts surrounding Madras and Bombay, and had migrated thence to Europe, Africa and America; that cholera was contagious and its poison could be dissolved in water and diffused in air. Limited outbreaks have occurred in Europe, it was acknowledged, and it was a question whether these were due to the spontaneous production of the cholerigenic miasma in European soil or to the slow development of miasma left by the preceding Asian epidemic. [Such verbose tautology is not very helpful.] They were on surer ground in concluding that the sources of contagion were the ejecta, the corpse, the linen and clothes, ships, rooms, carriages, latrines, contaminated water, the air (but at short distance only), animals and merchandise—no great advance on the records and opinions of Dr. Snow in the eighteen-fifties. This miasma, they averred, penetrated by way of the pulmonary and digestive passages.

Ten years later a Sanitary Conference was held at Rome and recommended disinfection to guard against the introduction and spread of cholera, referring more particularly to vessels, their reception and treatment at ports of arrival. Meanwhile, in 1883, the Local Government Board issued a circular to local sanitary authorities, enclosing copies of a memorandum on the precautions to be adopted in view of the suspected approach of cholera and stating that the trouble and the cost incurred would not in any event be wasted, since those conditions which favoured the spread of cholera favour that of other diseases also and that sanitary improvements which would confer security against the apprehended importation of cholera would, even if the invasion by