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CHOLERA STUDIES *

10. Epidemiology

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SYNOPSIS

The first section of this study deals with areas where cholera is endemic and with the conditions normally favouring endemicity. Turning next to epidemics, the author discusses their origin and types, climatic influences on them, their periodicity and the possibility of forecasting them, the role played in them by different serological races of *V. cholerae*, and the causes of their decline. In a section on the factors governing the local spread of cholera, he considers contact and water-borne infection; the role of contaminated food and drink, of fomites, of flies, and of carriers; and the incidence according to sex, age, race, and occupation. The last part deals with factors governing the spread of cholera over longer distances, and includes discussion of the effect of movements of individuals and groups and of assemblies of the population on pilgrimages or at religious festivals.

Endemic Areas and Endemicity

Statistical evidence of the endemicity of cholera in the Bengal Presidency (now Bengal State) and adjacent areas, long suspected of being the principal endemic centre, if not the home, of the disease, seems to have been furnished first by Bryden, statistical officer with the Sanitary Commissioner of India, in a series of four publications which began to appear in 1869 and were issued in collected form in 1874. As quoted by Yacob (1944), in Bryden's opinion the endemic area comprised " the western part of Assam, all the regions of lower Bengal and Orissa up to the low Rajnahal and Cuttack hills to the west of this basin as well as eastern Bihar ". At the same time Bryden denied that cholera was endemic in any other part of India. However, according to Swaroop (1951), he " failed to give detailed data with regard to the Bombay and Madras Presidencies, hence his statements do not present a complete picture ". The same may be said to hold true of the statement made by Koch at the 1885 cholera conference in Berlin that Bengal alone was the home of cholera.

* This is the tenth of a series of studies which will be published as a monograph on cholera in separate editions in English and French.—ED.

In contrast to these observers, Bellew (1884), judging from a study of the records of the cholera incidence in India from 1862 to 1881, maintained that a state of endemicity existed, not only in Bengal and the adjacent areas, but also in the interfluvial tracts of the Godavari, Kistna and Cauvery rivers in Madras, in the southern coastal districts of the then Bombay Presidency, in Oudh and the southern Gangetic districts of the north-western provinces, and possibly even in part of the Punjab. As pointed out by Bellew, generally speaking the endemic areas appeared to be

" characterized by a low-lying alluvial soil, which is more or less supersaturated with ground water in a state of stagnation or but comparatively very slight motion, and which is subject to periodical inundations or water-logging by the seasonal floodings of the great rivers by which those areas are traversed in deltaic formation. These physical characteristics of the endemic areas are coupled with equally striking features characteristic of their climatic conditions, *viz.* with those of a moist and hot tropical climate, and they are among the most densely populated parts of the country."

The almost invariable validity of these general statements by Bellew has been conceded by all subsequent observers.

Before continuing with a consideration of further studies on cholera endemicity in India attention has to be paid to contentions made by a considerable number of writers that endemic areas existed in other countries besides India. Rogers (1921) summarized that

" Apart from India, cholera is endemic in parts of the East Indies, Java having suffered as far back as 1629. It also occurs yearly in Southern China and the Philippine Islands. To the west the disease is so frequently carried to Persia and Arabia that it is difficult to say if it has become endemic in those countries or not. From 1851 to 1861 it was certainly present every year in Persia, but appears to have been frequently absent in subsequent years, so that it is probably not permanently located in that country. The same remark applies to parts of South-East Russia."

Rogers' opinions were not shared by Bernard (1936), who denied that cholera was endemic in Indochina, Indonesia or the Philippine Islands and doubted that the infection was permanently entrenched in China.

That such divergent opinions were expressed by different authors regarding the status of the various cholera-affected areas is easy to understand, because there can be no doubt that in place of a permanent entrenchment of the infection in a given locality a state of temporary endemicity may exist. Attention to the latter was drawn by Gill & Lal (1931), for instance, who, pointing out that cholera sometimes persisted throughout the winter in the Himalayas and the northern part of the Punjab, to become epidemic in the following spring, stated that

" There would thus appear a temporary form of endemicity which although lasting for one winter only, is capable of causing a widespread epidemic mainly in the northern half of the Punjab in the following summer."

The existence of " secondary foci " (" foyers secondaires ") of cholera, where the infection persisted for three or four years, ultimately to disappear,

was postulated by Bernard (1936). In his opinion certain cholera foci considered as permanent, like those in Indochina and China, were actually due to such a temporary entrenchment of the infection.

The validity of Bernard's contention was proved through observations made at Changteh, situated on an affluent of the Yangtze river in Hunan Province, central China. Robertson & Pollitzer (1939) were able to confirm the diagnosis of cholera in several patients seen in January 1938 as well as to isolate *V. cholerae* from some samples of the Yuan river water, and learnt from the local doctors that similar outbreaks of varying extent had taken place practically every winter throughout a number of years. It seemed likely that these manifestations stood in causal connexion with the frequent summer epidemics occurring in that area as well as in the adjacent Yangtze valley and that thus the problem of cholera endemicity in the latter, postulated by many authors, had been solved. However, when a few years later the present writer again stayed at Changteh to combat a plague outbreak, he was unable to find any evidence of the continued existence of cholera and, as far as is known, the region continued to remain free from the infection.

Again turning attention to the problem of cholera endemicity in India, reference has to be made first to large-scale investigations undertaken by Russell & Sundararajan (1926, 1927). As the former summarized at the 1927 Conference of the Far Eastern Association of Tropical Medicine in Calcutta (Russell, 1929), a study of the cholera mortality over a long period of years had made it possible to divide the provinces of India into three groups:

" I. The first group includes the provinces of Assam, Bengal, Bihar and Orissa and the United Provinces, where more or less uniform figures are registered annually and where the average incidence is high. These areas are very likely to be endemic in nature.

" II. In the second group are included the Central Provinces, Bombay Presidency and the Punjab and North-West Frontier Province, where sudden peaks in cholera incidence occur at irregular intervals. These areas are normally free from cholera epidemics and infection is probably always brought in from outside.

" III. The Northern and Central Districts Groups of Madras Presidency are epidemic areas; whilst the Southern Districts Group, which presents a more uniform incidence, might almost be included in Group I as an endemic area."

Russell added that the differentiation of the areas of India into epidemic and endemic groups had been confirmed by various other statistical methods. His and Sundararajan's investigations had established that cholera tended to recur repeatedly in river deltaic tracts, the main endemic areas of India including the delta areas of the Ganges, Brahmaputra and Cauvery rivers. The outbreaks commenced in the towns or villages lying on the banks of these and other rivers, the infection then rapidly and systematically spreading down the waterways. " Moreover ", Russell added,

" there is no question that, in endemic areas, cholera spontaneously appears, year after year, in the same villages and towns. In other areas, *per contra*, it is necessary for other favourable conditions to be present before cholera becomes diffused, e.g. overcrowded and insanitary conditions associated with religious fairs and festivals."

Rogers (1926, 1928), studying the incidence and spread of cholera in India, claimed that a state of endemicity existed not only in Lower Bengal, Orissa and Assam, but also (*a*) in the extra-deltaic western divisions of Bengal and the north-easternly sub-Himalayan divisions of the United Provinces; (*b*) the extensive low-lying districts of South-East Madras (suspected by Russell & Sundararajan) and (*c*) a small low alluvial district of the North Konkan districts of Bombay Presidency (now Bombay State) lying in an area already incriminated by Bellew (1884). Rogers admitted, however, that the last three endemic areas differed from the " hyper-endemic " area of lower Bengal, Orissa and Assam " in that, although cholera is never absent for a whole year, yet the rate per mille not very rarely falls to less than one-tenth of the average rate ".

It is also interesting to note that according to an account published in the 1941 report of the Indian Research Fund Association a spurious form of endemicity existed in the Tanjore district of Madras State, which was due to differences in the seasonal incidence of cholera in the various parts of this region with the result that outbreaks were apt to commence in some of its parts at the time when they terminated in others.

In the first of a series of three most important articles devoted to a statistical study of the cholera incidence in south-west Bengal, Lal, Raja & Swaroop (1941) pointed out that the various districts of this area " present considerable heterogeneity in regard to their cholera experience and that there are also evidences of heterogeneity within the districts themselves ". For a closer study of the endemiology and epidemiology of the disease it was necessary, therefore, to divide up south-west Bengal into what the authors called " homogeneous cholera districts ".

As discussed by Lal et al. (1941) in their second publication, the following steps were adopted for this purpose:

" 1. Taking the thana [local district] as a unit the total variability of cholera incidence has been split up into three variables, viz., seasonal, yearly and residual, by the method of analysis of variance.

" 2. Contiguous thanas showing similarity in respect of different types of variation, mean cholera incidence and type of seasonal curve have been combined and the districts so obtained tested for homogeneity by trivariate analysis of variance. Those satisfying tests of homogeneity have been constituted into cholera districts."

It was found that the size of the 19 cholera districts thus created varied considerably from single thanas to combinations of up to 20 such local administrative units.

As stated in the third article of the series of publications presently under review, Swaroop et al. (1941) found that in 9 of these 19 cholera districts a state of endemicity existed, whereas 10 experienced only epidemic cholera

manifestations. Classifying the endemic as well as the epidemic homogeneous districts according to the degree of their epidemicity, Swaroop and his co-workers obtained the following results:

Category of districts	*Degree of epidemicity*					
	very high	*high*	*moderate*	*low*	*very low*	*total*
Endemic	3	—	—	1	5	9
Non-endemic	2	1	1	1	5	10

The interesting fact may thus be noted that cholera was but little epidemic in a majority of the endemic districts. It is an intriguing question to what extent this absence of major cholera outbreaks was the result not of extrinsic conditions but of a herd immunity acquired during preceding epidemics. Reference has to be made in this connexion to the following statement made long ago by Hart and his collaborators (1910):

" Within certain areas in India cholera is endemic, especially in the country of the Lower Ganges. If, however, we examine carefully the incidence of cholera within the endemic area, it becomes obvious that, although in every district deaths from it may be reported every year and every month in every year, still the incidence of this mortality is by no means evenly distributed; even within the endemic area cholera wanders about, one village after another being attacked and then left at peace for a time. It seems as if there were the same tendency for these outbursts to die down within the area as there is outside it . . . "

For a further study of the problem of cholera endemicity in India, Swaroop (1951) computed for each cholera-affected district the average mortality rate from the disease for the 10 years with the lowest mortality during the period from 1901 to 1945, and used the magnitude of this average as a rough measure of endemicity. He emphasized, however, that he applied this term in a purely relative sense, postulating the existence of highly endemic or moderately endemic districts and of districts without or with a low or doubtful endemicity only as far as the mutual relations of the various areas were concerned. Still, Swaroop maintained that though his analysis did not demarcate the endemic areas in any absolute sense, it served to exclude tracts of the country in which endemic zones of cholera could not be expected to lie.

According to these observations the principal regions in which cholera showed a tendency to persist at high level were the following:

" (1) In Bengal at the lower reaches of the Hooghly river, i.e. in the delta formed by the Hooghly, the Damodar and the Rupnarayan.

" (2) At or around the confluence of the Ganges and the Brahmaputra and the Meghna and in the lower reaches of their delta including that of the Madhumati . . .

" (3) In Orissa in the deltaic zone of the four rivers, viz. the Mahanadi, the Brahmani, the Baitarani and the Subnarekha.

" (4) In northern Assam in the Brahmaputra valley.

" (5) In Bihar in and around the Patna and Gaya districts, where the Sone, the Gandak and the Gogra join the Ganges.

" (6) In the U.P. in the low-lying river basins beginning from the junction of the Ganges and the Jamuna at Allahabad and extending through Banares to the points where the Gumti and the Sone join them.

" (7) In South Madras in the Cauvery delta.

" (8) Possibly also in North Madras in the delta formed by the Kistna and the Godavari rivers and around the Colair lake."

Swaroop also determined the average periods in months during which the various affected districts had been free from cholera during the past 30 years. The results obtained in this manner tallied well with those arrived at with the aid of the determinations mentioned above.

To guard against the possibility that the above-mentioned observations, extending over periods of 45 and of 30 years respectively, might fail to give a correct picture of the cholera endemicity then prevailing in India, determinations were also made of the length of the weekly periods during which the various districts had remained free from cholera during the past 10 years of the observation period. It is of interest that in this manner it became possible to establish the probable formation of a new endemic focus in the Bombay Presidency (now Bombay State). Otherwise, however, the results of these observations were in agreement with those referred to above.

Comparing the various regions in which endemic areas were expected to be present, Swaroop noted that

" 1. All endemic areas are located generally around rivers.

" 2. All these tracts lie in areas of high population density.

" 3. All of them lie in low-lying lands, i.e. none is more than 500 feet [150 m] above sea level.

" 4. All these tracts lie in areas of high absolute humidity."

There can be no doubt that these and other conditions usually prevailing in the endemic areas are rather favourable for the persistence of cholera and at the same time render the implementation of measures to control the infection most difficult. This is true in the first place of the water supplies, because it is an almost or even quite impossible task to dig satisfactory wells in the low-lying and water-logged endemic areas, while owing to technical and financial reasons it is as a rule not within the realm of practical politics to provide pipe-water throughout the often extended districts. Therefore the people densely populating them, even if they possess a knowledge of the elementary rules of hygiene—which is an exception rather than the rule—must usually draw their drinking-water supplies from the surface water courses which, because they also serve for all other household purposes and even as sewers, are grossly contaminated and thus apt to convey any cholera infection present. Facilities for the rapid recognition and the isolation of cholera patients are likewise poor, particularly during the frequent periods of high water-level or of floods.

These and most other conditions prevailing in the endemic areas appear to be so favourable for the spread of cholera that the question to be asked is not why the infection persists but why it does not constantly cause widespread epidemics. As has been alluded to above, it is probable that the development of a herd immunity in the endemic areas is apt to lead to a kind of equilibrium between the causative organisms and the host population. Factors like food shortages, which lower the resistance of the people to cholera, and, more still, a seasonal influx of labourers or the arrival of other susceptible persons presumably play an important role in the recrudescence of epidemic manifestations.

Epidemics and Epidemicity

Origin of epidemics

Cholera epidemics may arise either in endemic areas, where, as has been discussed above, the infection, fostered by particularly favourable conditions, is constantly present, or in localities ordinarily free from the disease. Most modern observers agree that such invasions of hitherto cholera-free areas are the result of importations of the infection by means which will be discussed later in this study. It is, however, curious to note that the idea of an autochthonous origin of cholera outbreaks, which had been amply discussed before the detection of *V. cholerae*, was afterwards supported by claims made by a few workers in regard to the possibility of a transmutation of cholera-like vibrios into the true type, the thus " regenerated " vibrios becoming capable of starting cholera manifestations in man. However, when exhaustively discussing the evidence brought forward in this respect in the fourth of these studies,[1] Pollitzer & Burrows came to the conclusion that

" there is no convincing evidence to show that such transmutations take place under natural conditions and that consequently cholera-like vibrios or cholera vibrios which had lost their agglutinability with the usual specific sera form a reservoir from which epidemics may be produced *de novo*."

Types of epidemic

As first exhaustively described by Koch (1893), two main types of cholera epidemic may be distinguished—namely, (1) an explosive type, apt to end as abruptly as it started; and (2) a protracted type of outbreaks which commence slowly and afterwards follow an insidiously prolonged course. Koch was, however, careful to point that often a combination of these two types of cholera outbreak became manifest. " Thus," he said,

[1] See *Bull. Wld Hlth Org.*, 1955, **12**, 1008 et seq.

" particularly the first type, which appears mostly at first in pure form, later becomes combined with the second type and finally altogether passes over into the latter. It also occurs that a local epidemic begins with the second type until accidentally the infectious material finds its way into the water and then, according to the system of water supply, produces small circumscribed explosions or suddenly produces infection in a whole precinct, occasionally even in the whole community." [Trans.]

Dealing in a detailed manner with the two types of cholera outbreak, Koch stated that in the first type of epidemic the instances of infection were distributed in a fairly even manner over the whole community affected without any immediate connexion between the individual manifestations. Such a uniform distribution of the infection, he continued, could be produced only through a vehicle capable of acting simultaneously upon all or at least most of the inhabitants of the locality concerned, for instance, the air, water, soil or foodstuffs. Koch maintained, however, that

" so far it has not been possible to demonstrate a role of the air, soil or foodstuffs in the explosive cholera outbreaks. Likewise insects, which have been suspected with reason, cannot come into question, because cholera explosions occur not rarely during the cold season when a transmission through insects is out of the question . . . Thus only the water remains; and that this can be the actual vehicle of the cholera germ not only for single groups of the population of a community, but for whole communities and even large cities, has been proved by past epidemics and quite specially by the present cholera outbreaks in Hamburg, Altona and Nietleben." [Trans.]

In the second type of outbreak, the single manifestations did not show a uniform distribution; on the contrary, the infection became entrenched in foci. In these not many fell ill simultaneously, but infections occurred, as it were, in chains, often standing in causal connexion with one another. Secondary foci could form not only in other precincts, but also in adjacent communities.

Koch admitted that it was not invariably possible to demonstrate how the chains of infection, characteristic of the second type of cholera epidemic, were formed. For, besides severe, slight and often unrecognized cholera attacks developed, and the patients were infectious not only during the manifest stage of the disease but also before and after this. Moreover,

" infection was by no means always derived directly from the cholera patients but still far more frequently was produced indirectly through linen, clothes, beds, foodstuffs, insects, etc." [Trans.]

It was possible nevertheless to study the spread of the infection in sparsely populated rural areas, but this was most difficult in larger towns, particularly because there cholera spread mainly among " the lowest, closely herded together and constantly fluctuating strata of the population, reaching but rarely the better situated ".

The lucid statements of Koch quoted above, which indeed one might reprint with but little modification in a modern textbook on cholera, have been accepted by most subsequent observers.

Owing to the great differences in the conditions prevailing in the various cholera-affected areas, the comparative frequency with which outbreaks of either the first or the second type take place shows marked variations, explosive epidemics due to a contamination of major water supplies preponderating in some areas, and protracted outbreaks in others. It is important to note in this connexion that explosive cholera manifestations may take place in villages as in urban communities. Thus Gill & Lal (1931), who were able to collect on this point large-scale statistics in the Punjab, found that out of a total of 298 outbreaks in towns eight exhibited definite explosive characters, whereas there were 60 epidemics of this type among a total of 2917 village outbreaks.

Climatic influences

Early observations

Dealing in his classical work on cholera with " the bearing of meteorological influences upon the spread of cholera ", Macnamara (1876) took a determined stand against the postulation of many of the early observers that the prevailing winds exerted a direct influence in propagating the infection. He quoted in this respect a statement made by the Bengal Medical Board on the 1817-18 epidemic, saying that the members of the Board, while

" they hesitated to express an opinion as to the nature of the apparent connexion between cholera and the easterly wind . . . expressly stated their belief, that of all the predisposing causes to cholera, the one most frequently and unmistakably in operation was ' alternations of heat and cold combined with rain, or a very humid state of the atmosphere '."

Evidently being in accord with the latter contentions, Macnamara laid down the general rule

" that cholera will not extend during the cold of a European winter, or even of our Punjab cold season ".

It was true that cholera outbreaks during the winter had occasionally been observed in Europe, e.g., in the Polish army in 1830-31. But these exceptions did not invalidate the rule that a drop of the atmospheric temperature to 50°F (10°C) impeded the spread of cholera, especially in dark and gloomy weather, whereas the progress of the epidemics was stopped when the temperature fell below 40°F (4.4°C).

At the same time Macnamara emphasized the important role of atmospheric moisture as " a necessary element for the development of the disease ".

Regarding the role played in this connexion by the rain, he pointed out that in Bengal

" cholera is at its height . . . every year in March and April and again in September and October, and these are the very months in which we get heavy downpours of rain, washing the surface soil and its contents into the wells and tanks from which we procure our drinking water; these storms are generally followed by intensely hot days. As soon as the regular rains set in, and we get a more or less continuous downpour for some three months, cholera ceases for the time, and in fact until the close of the year, when it breaks out again in the stormy weather which, with intervals of intensely hot days, succeeds the rain."

In the north-western parts of India, the moisture-laden south-west monsoon promoted the spread of cholera brought in from Bengal through boats sailing up the Ganges. But as soon as the rains ceased and the dry west winds of the upper provinces set in, cholera began to decline and, as a rule, remained in abeyance during the entirely dry period lasting from the end of September until the onset of the monsoon in the following June.

The rarity of cholera outbreaks in winter is well illustrated by statistics of Hirsch (1883), according to which out of 920 epidemics occurring outside of India only 42 started or became prevalent during the cold season as against 261 in spring, 496 in summer and 121 in autumn.

Sticker (1910) laid stress on the fact also noted by subsequent observers that cholera epidemics were particularly apt to occur in years during which an unusual drought prevailed. Thus it had been shown by Wolter (1898) that the cholera periods in Hamburg from 1831 to 1873 invariably fell in dry years. The 1892-93 outbreaks in that city also occurred during a dry period between the wet years 1888-91 and 1894-98, the year 1892 being quite exceptionally dry.

Later observations

Subsequent observations on the influence exerted on cholera manifestations by climatic factors may be discussed under the following headings.

Temperature. The contention of the early writers that as a rule cholera becomes epidemic during the warm season of the year and declines when cold weather sets in, has been fully confirmed through further observations. To quote an example, Bernard (1936) recorded that in Indochina the disease was most rampant during the hot and dry season (April-June). The situation again became aggravated after the cessation of the rains during September to November, but a marked decline of cholera occurred during the period from December to February, the coolest season in Indochina. The frequency of cholera cases thus stood in inverse proportion to the temperature as well as the precipitations (" chutes d'eau ").

Similarly to these and other observations, Takano and colleagues (1926) summarized that August and September, when importations of cholera into Japan were most likely to take place,

" are the months in which epidemics reach their peaks. The epidemic begins to subside gradually in October and November and practically ceases in December."

Limited winter epidemics were reported in Japan upon two occasions only (1886 and 1917) but, if the warm-weather outbreaks started late, sporadic cholera manifestations were frequently observed during the first two months of the following year. Thus, as Takano and his associates admitted,

" The existence of winter cholera in Japan indicates that the cholera vibrio is able to survive the winter. But it is very rare that the cholera vibrio becomes the source of another epidemic after surviving the coldest weather of February. No such instance has occurred since 1900."

Generally speaking, however, a recrudescence of epidemics after the sporadic subsistence of cholera throughout the winter not only occurs in the endemic areas but has also been observed by no means rarely in localities subject solely to epidemic inroads of the disease. An interesting example of the latter kind was met with by the present writer during the period 1939-40 in the northern part of Szechwan Province in China, where the winters are quite severe. The region in question had been quite heavily involved in 1939, which was a bad cholera year for China in general. As confirmed by a survey made in 1940, sporadic cholera cases continued to occur during the winter 1939-40. The spring of the latter year being unusually dry, the infection soon flared up and a further serious outbreak resulted. However, though cholera thus temporarily persisted, it did not become permanently entrenched in the area.

The question why cholera usually shows a markedly greater tendency to spread during the warm seasons of the year than during cooler or cold periods is rather involved. At first glance it would be tempting to ascribe this difference to a more prolonged survival or even a multiplication of *V. cholerae* in the surface water supplies or on substrates like fruits and vegetables at a high temperature. Actually, however, as will be gathered from the relevant data quoted in the third of these studies,[1] the length of survival of this organism under these circumstances was apt to show a decrease rather than an increase *pari passu* with an increase of the ambient temperature. Other factors must be therefore responsible for the increased frequency of cholera during the warm season. As recognized by Flügge in 1893, of great importance among these is that prevailing hot weather leads to an increased consumption of raw water and other cold drinks as well as of cold foods like fruits, salads and jellies, apt to be contaminated with *V. cholerae*. There can be no doubt that the frequent prevalence of flies during the summer greatly facilitates such contamination. Moreover, as pointed out with great reason by Flügge, the consumption of the raw drinks and foods is bound to lead to the frequent appearance of gastro-intestinal affections caused by other species of micro-organisms, which in their turn lower the resistance to cholera infection.

[1] See *Bull. Wld Hlth Orq.*, 1955, 12, 845 et seq.

A further postulation of Flügge was that the lowering of the level of rivers and other surface waters as well as of the ground-water in the wells, apt to occur in late summer and autumn, might be of importance because, if such waters became contaminated with cholera vibrios, their vibrio content would remain high and possibly also because the organisms could thrive better in concentrated waters containing a large amount of organic matter. As maintained by Jolly,[1] the survival of *V. cholerae* in surface waters is possibly also governed by changes in the reaction of the water, which might be favourable only at certain seasons of the year.

Humidity. Though many of the early observers had become convinced of the existence of a close relationship between a suitably high atmospheric humidity and the spread of cholera, it was only about thirty years ago that large-scale studies on this subject were made by Rogers (1926, 1928) and by Russell & Sundararajan, who embodied the final results of their exhaustive investigations in a research memoir published in 1928.

Rogers, studying the relation between climate and cholera incidence in India, found that rainfall alone did not account for the seasonal distribution of the disease, for the latter was at its minimum during the south-west monsoon in Assam and Lower Bengal, but at its maximum during the same season in other parts of the subcontinent, such as the Punjab.

The mean temperature showed a closer relationship to the cholera incidence, since the disease was at its minimum during the winter season in the Punjab, the United and Central Provinces and the Deccan area of Bombay, but the same was not true of Lower Bengal. Determinations of the relative humidity also failed to furnish universally valid clues, since in the Punjab, for instance, the latter was " nearly as high in the minimum cholera months of December to February as in the maximum ones of the rainy season in July ". Therefore, Rogers insisted, there remained only

" the absolute humidity or aqueous vapour pressure, which is measured as air pressure in terms of the length of a column of pure mercury at temperature 32°, and is obtained from observations of the wet and dry bulb thermometers by means of special tables . . ." [2]

Rogers admitted that no relationship existed between a high absolute humidity and cholera incidence but maintained that

" when we turn to the months of low absolute humidity we find that in every area in which this reading falls below 0.400 during the cold weather months, cholera at the same period falls to a very low rate, as in Bihar, the United Provinces, Central Provinces and North Deccan and altogether disappears in the Punjab."

" Still more significant ", Rogers continued, " is the fact that the winter decline of cholera in Assam and Lower Bengal in January and February immediately follows

[1] See *Bull. Wld Hlth Org.*, 1955, **12**, 858.

[2] As defined by Chun (1933), absolute humidity is " the weight of aqueous vapour in the air measured in terms of its mercury tension, so that low readings indicate both dryness and low temperature and vice versa. In order to find absolute humidity, one must first know temperature and relative humidity and then look up water vapour tension tables."

the lowest absolute humidity in December to February of from 0.425 to 0.475 and the mortality rises once more in these areas with the much increased absolute humidity in March . . . Equally close is the relationship to increasing cholera prevalence in North-western and Central India . . ."

As further postulated by Rogers, the autumn decline of cholera in the latter areas also coincided with a fall of the absolute humidity below 0.400, thus

" completing the evidence of the closest association with that degree of dryness and falling cholera mortality, and indicating that this condition is unfavourable to the continued survival of the infective agent outside the human body in sufficient quantity to keep up the epidemic prevalence of cholera over large tracts of country."

The validity of Rogers's conclusions was upheld by a number of subsequent observers, such as Chun (1933) in China, Khalil (1948b) in Egypt, Yu Wei (1949) in Shanghai and Banerjea (1951) in the United Provinces (now Uttar Pradesh) of India. However, disagreement with the findings of Rogers was recorded by some other workers, like Dunn & Khan (1929), Gill & Lal (1931) and particularly by Russell. Referring to Rogers's initial work in a paper read at the 1927 Congress of the Far Eastern Association of Tropical Medicine, Russell (1929) stated that

" The arguments brought forward in favour of an absolute humidity figure of 0.40 seem . . . to be based on broad generalizations. With all due deference, it is suggested that conclusions of this kind cannot possibly be reached without submitting the available data to detailed statistical analyses and it does not seem that such methods were employed."

Russell stressed that relative instead of absolute humidity was of epidemiological importance but maintained at the same time that the clue to the cholera problem was not to be found in any individual climatic factor. Analogously Russell and Sundararajan (1928), analysing the results of their exhaustive statistical studies on the epidemiology of cholera in India, concluded that

" The association of high relative humidity with high temperature, accompanied by intermittent rains, forms the most favourable atmosphere for the development of the disease. The presence of endemic centres from which epidemics spring at short intervals is also a fact which must be accepted. No single factor, however, can be held responsible for the periodic waves of the disease . . . and it must be recognized that these waves are preceded by conditions too complex to admit of complete solution with the aid of available data. Individual susceptibility, foci of infection, favourable atmospheric conditions, fairs and festivals, carriers, insanitary habits, all play their part in a manner which defies analysis."

As far as the present writer is entitled to judge, the great import of this statement cannot be overrated.

Rainfall. In agreement with observations by Macnamara (1876) quoted earlier in this study (see page 792), Koch stated at the 1885 cholera conference

that (*a*) even though the period from February to April, during which the incidence of cholera at Calcutta increased, was generally a dry season, occasional heavy downpours of rain were by no means rare; and (*b*) according to the local practitioners such rainfalls were invariably followed by a deterioration of the epidemic situation. This was not surprising, because the rains were apt to wash cholera-infected faeces and other contaminated materials into the " tanks " (or ponds) serving as sources of water supply in the foci. The danger of a heavy contamination of the tanks during the rainy seasons was considerably less because the infective materials then introduced were apt first to become diluted and finally to be carried away. If on account of the absence of rains the level of the tanks became low, a dangerously high concentration of cholera-contaminated materials introduced by bathers or through the washing of infected clothes or other objects might result.

The contention of Koch that, owing to variances in their duration rainfalls were apt to lead either to an exacerbation of cholera outbreaks or to a decline or even cessation of the epidemics, has been confirmed through numerous subsequent observations. It has to be noted, however, that prolonged rain does not invariably exert an inhibitory influence, in some areas the epidemics continuing or even starting during rainy seasons.

The early observations that cholera manifestations, if they arise after or during periods of exceptional drought, often become particularly dangerous has likewise been reliably confirmed. At such times the people are forced to make use of the scanty water supplies remaining available, however unsuitable or even repulsive they may be. If cholera becomes recrudescent or is imported, contaminations of these sources of water supply, at which the people concentrate, is easily possible, and once such infections have taken place, a rapid spread of the disease is well-nigh inevitable.

Long-term periodicity

Evidence indicating that cholera manifestations, in addition to showing a seasonal incidence, might also exhibit features of a long-term periodicity was adduced by some of the earlier observers. Thus, as summarized by Russell & Sundararajan (1928),

> " Bellew, in 1884, produced statistics from every province in India, relating to the period 1862-1881, in order to prove that the disease appeared in triennial waves. He attempted to show that cholera tends to run a definite course of revival, decline and subsidence in the successive years of each triennial cycle."

In agreement with Bellew's postulation, Koch stated at the 1885 cholera conference that, as shown by the mortality figures from 1870 to 1883, in Bombay Province the fatalities from cholera reached a peak every third year, while the mortality was remarkably low during the following two

years. The only exception to this rule occurred in the period immediately following the cholera year of 1875, when—owing no doubt to the influx of many half-starved people during the famine then prevailing—the mortality from the disease remained high. Koch felt convinced that this periodicity of cholera was due to the development of an immunity in the people who had survived attacks of the disease.

Attention to a periodical recrudescence of cholera in India was again drawn by Russell (1925), who, in a note published in the *Lancet*, stated that he had applied the method of periodogram analysis to study the incidence of cholera deaths in Madras Presidency for a period of 25 years and had found evidence of a six-year periodicity. He afterwards established that the cholera mortality figures also reached peak values at intervals of 72 months in many other parts of India suffering from epidemic invasions of the disease.

Rogers (1926) asserted, on the contrary, that

" from a study of the tables I have worked out during the last twelve months of the cholera rates per mille over 200 districts and forty-five divisions of India, for a period of forty-five years, I am unable to trace anything like a three-year, up to a six-year cycle if a long period of time is studied."

He felt certain, therefore, that Indian cholera outbreaks occurred at irregular intervals.

However, Russell & Sundararajan (1928) again claimed on account of their exhaustive statistical studies that cholera in India did show a long-term periodicity. Commenting upon these findings, Russell (1929) made the following statements at the 1927 conference of the Far Eastern Association of Tropical Medicine:

" Periodicities of longer duration, while not obvious, have been demonstrated by the application of the periodogram method used by Brownlee [1919]. By this means, it has been found that, in nearly all the areas where cholera is epidemic, waves of the disease recur once every five to six years, whilst in the endemic areas, a 4-5-years periodicity is most probable. In every case the periodograms show that cholera tends to run a more or less definite course of revival, decline and subsidence in each cycle of years. This phenomenon has been demonstrated further by the epidemic indices curves relating to the different areas of India [Russell & Sundararajan, 1927]."

Referring to Koch's postulation that an immunity developing in the survivors from cholera attacks accounted for the periodicity of the outbreaks, Russell stated:

" Probably other factors have equal significance, but whatever influences may be at work, it is certain that fore-knowledge of the probable advent of a periodic peak in the incidence of the disease would go far to prevent waste of effort in unnecessary directions and at unnecessary seasons. In Madras, we have for three years past made use of that knowledge with very considerable success."

Further statements made in regard to the cyclical incidence of cholera epidemics may be summarized thus:

Author	*Findings*
Chun (1935)	Stated at the 1934 conference of the Far Eastern Association of Tropical Medicine that " in order to ascertain if there was any periodicity in the cholera epidemics in Shanghai, a chart of the annual cholera deaths during the period of 49 years (from 1886-1934) was made. There was a suggestion that severe epidemics occurred at intervals of four years, that is to say, two mild or clear years might follow a severe epidemic. This condition of affairs was particularly noticeable within recent years."
Parthasarathy & Sundararajan (1937)	Found that the periodicity of cholera in Mysore State in India " is most likely to be of six years' duration."
Sen (1948)	Recorded in a study of the vital statistics in the United Provinces (now Uttar Pradesh) that: " As regards cholera, there was uniformly a 3-year epidemic cycle till 1930 and thereafter the pattern seems to have changed. The years 1930, 1938 and 1945 were high epidemic years for the province."
Benjamin (1949)	Stated in a report on cholera in Bombay State that: " Considering the province as a whole, widespread severe epidemics, i.e. involving more than half the districts of the province, have occurred roughly at intervals of 3 to 4 years during the first two decades after 1901, and at irregular and longer intervals since 1921."
Duggal (1949)	Found that in Bihar the peak of moderate epidemics was reached at an interval of about five years and that of severe outbreaks after 12 to 14 years.
Banerjea (1951)	Maintained that: " Seeing the mortality rates over a long period of 1877 to 1948 . . . one may say that cholera in U.P. [United Provinces] occurs at irregular intervals and . . . is largely influenced in its periodic explosiveness by big fairs and festivals held from time to time in the province. The three years periodic cycle noticed by other workers in this province may be due to the occurrence of Kumbh and Ardh-Kumbh [i.e., extraordinarily frequented] fairs at Hardwar and Allahabad which recur after every three years."

If consideration is given to the discrepant statements recorded above, it is impossible to support the earlier claims that cholera epidemics regularly show a long-term periodicity. At the same time, however, Russell (1929) was certainly right when insisting that observations on this point made in individual cholera-affected areas may be eminently useful for directing the main efforts of the workers to potential danger spots.

Forecasting of epidemics

As far as could be ascertained, a successful attempt to utilize observations on past cholera manifestations for forecasts of future outbreaks was first made by King. Commenting upon Russell's 1925 article in a letter to the

editor of the *Lancet*, King stated that the statistical method described in the article

" was already employed by one of his predecessors in office in 1894 and formed the subject of a paper read before the first Indian Medical Congress. This was illustrated by a diagram founded on the figures for a decade; it inhibited [sic] monthly periodicity so markedly as in after years to justify it being termed the ' Madras Cholera Clock '." [1]

Russell & Sundararajan (1928) paid a just tribute to King's pioneer work, stating

" that his ' cholera clock ' continued to ' keep good time ' and due acknowledgment must, therefore, be made to that distinguished sanitarian for the clue on which most of the work to be described has been based."

As already alluded to (see page 797), in the opinion of Russell (1925)

" the mere knowledge of this six-yearly periodicity of cholera has enabled the public health department in Madras Presidency to control its preventive work in connexion with the disease and has prevented waste of effort in unnecessary directions and at unnecessary times ".

Nevertheless Russell & Sundararajan (1927), in order to forecast cholera epidemics, resorted to the elaborate method for the early detection of epidemic trends devised by Bundesen & Hedrich (1925). These two workers had found that the expectancy or median incidence of an infectious disease during a period of from five to nine previous years was apt to serve as a reliable standard for gauging the present incidence of the disease and that the " epidemic index " or ratio of the current incidence to the expectancy was an approximate barometer of the fundamental epidemic trend, the index often rising a number of months before the approach of an epidemic could be detected with the aid of the ordinarily used methods.

Applying the new method in a somewhat modified form for a study of cholera incidence in India, Russell & Sundararajan established that by means of the epidemic index it was possible to forecast epidemics two or three months ahead of their actual occurrence. They maintained, therefore, that

" By the use of the epidemic index graphs a watch can be kept for the possible outbreaks of cholera; and, as a corollary, when an epidemic is forecast, preventive measures can be intensified; when a calm period is indicated, energy and expenditure can be conserved."

It is important to note, however, that, as stated by Taylor (1941), the above method failed to give satisfactory results in areas with homogeneous cholera experiences. Taylor added that

" Another method based on the regression equation connecting cholera mortality of the last few weeks of a year with the total cholera mortality of the succeeding year

[1] A reproduction of this diagram was appended to an article on applied hygiene in the tropics published by King in 1919.

gave more satisfactory results. This method gave good predictions for Calcutta for three successive years and was successful in 10 out of 19 of the homogeneous districts which have been designated for South-West Bengal."

Rogers (1928) claimed on the basis of his exhaustive investigations already referred to earlier in the present study that

" by watching the absolute humidity, at seasons when it is commonly low enough to lessen the incidence of cholera, for any rise to over 0.400 favouring the recrudescence or spread of the disease, and taking into account the deficiency or otherwise of the previous rainfall, and the prevalence of cholera in surrounding areas, it should be possible in future to form a good idea of the relative danger of any pilgrimage, fair or other large gathering in any given place in time to issue warnings and to take other precautions to avert or lessen the impending danger."

As Rogers summarized in a lecture delivered in 1933, his method of forecasting cholera epidemics had given fully satisfactory results on repeated occasions. Making a further study of the data available up to 1939, Rogers (1944) reiterated that

" 1. A close watch on the June to October South West monsoon rains enables high cholera incidence to be foreseen in the autumn months in the endemic areas with absolute humidities always over 0.400 and several months before the spread of epidemics of cholera in the next spring from the endemic to epidemic areas.

" 2. The danger of cholera spread by the return of pilgrims from any particular Fair can also be foreseen from the climatic data at the time and a knowledge that cholera is present in the areas through which the pilgrims have to travel."

Rogers's claims were not fully accepted by Taylor (1941), who reported that forecasts made according to the above method had proved successful in only 24% of areas with homogeneous cholera experiences. However, Napier (1946) was inclined to consider Rogers's method as the most successful as well as simplest of the various forecasting procedures.

Role of the serological races of V. cholerae

Observations on the incidence of the serological races of *V. cholerae* have been recorded in the following countries:

Japan

As noted in the fourth of these studies,[1] Kabeshima (1913), examining 195 strains isolated during the 1912 outbreaks in Japan and Formosa, found that the Japanese strains were of a " typical " serological character (or, to use the nomenclature now in use, were Inaba strains), whereas the Formosa strains were serologically " atypical ", thus belonging to the Ogawa race of *V. cholerae*. Kabeshima's findings were soon confirmed by other

[1] See *Bull. Wld Hlth Org.*, 1955, 12, 962.

Japanese observers who, as aptly summarized by Venkatraman & Pandit (1938), believed

" that the ' original ' or ' Inaba ' type is associated with epidemic outbreaks and severity of infection and the ' varied ' or ' Ogawa ' type with sporadic cases and mild outbreaks."

According to Nobechi (1923, 1933), from 1922 the " intermediary " or Hikojima type detected by him became predominant in Japan.

Evaluating this change of type, one must keep in mind that the successive waves of cholera epidemics in Japan were invariably the result of recent importations of the infection, which thus never became entrenched in the country.

Korea

According to Shiiba & Ushijima (1922), among 20 cholera strains examined by them those isolated during the 1921 outbreak in Korea showed the characteristics of the Inaba race.

Manchuria

As established by Manako (1933), almost all of 187 cholera strains which had been isolated during the 1932 epidemic in Manchuria were of the Ogawa type.

China

(a) Shanghai. Writing in 1933, Nobechi maintained that, as in Japan, so also in Shanghai the Hikojima race of *V. cholerae* was preponderant. However, Kuroya & Oho (1933) found that out of 53 strains isolated during the 1932 epidemic only five belonged to this race, while 48 showed reactions corresponding to those of the Ogawa type. According to Fournier (1939), the Hikojima type was once more preponderant in Shanghai in 1933. Nishimura (1938) found, on the other hand, that all 16 strains he had examined during the 1937 epidemic were Inaba strains.

As stated by Fournier (1939), 93% of the 100 strains examined during the 1938 Shanghai outbreak were again of the Hikojima type. However, according to Fournier & Lieou (1943), the sporadic cholera manifestations at Shanghai in 1939, 1940 and 1941 were caused by organisms of the Inaba race, whereas all 65 strains examined during the 1942 epidemic showed the reactions of the Ogawa type.

The great variance of the above findings lends support to the view that the cholera manifestations in Shanghai were the result of repeated importations of the infection, which thus never became permanently entrenched.

(b) Yunnan Province. Tang and colleagues (1944), typing 69 *V. cholerae* strains isolated during the 1942 outbreak at Kunming, found 64 to be of

the Inaba race, while five, met with at the end of the outbreak, were of the Ogawa type. Tang and his co-workers considered it possible that this appearance of another serological race might stand in causal connexion with a reimportation of the infection from an area different from that responsible for the initial outbreak.

(c) Szechwan Province. According to Reimann (1947), out of seven strains which had been isolated during the 1945 cholera outbreak at Chungking, two were of the Inaba type, three of the Ogawa type and two of the Hikojima type.

Indochina

Genevray and co-workers (1939) recorded that they had found exclusively strains of the Inaba type during a limited but rather virulent cholera outbreak in a Tonking village.

Burma

Out of 52 cholera strains examined by Maitra and colleagues (1938) at Rangoon, 51 were of the Inaba type, and only one of the Ogawa type.

India

(a) Bengal. As summarized by Taylor (1941), early investigations in Bengal had indicated the sole presence of cholera vibrios of the Inaba type, but it was afterwards established that side by side with this race Ogawa strains could also be isolated. Further observations proving the co-existence of these two serological types may be tabulated thus:

Author and locality	*Total number of strains*	*Inaba strains*	*Ogawa strains*	*Remarks*
Pasricha et al. (1939) Calcutta	379	266	113	
Read & Pandit (1941) Rural area	65	26	39	32 of these 65 strains had been isolated from patients, 23 from contacts and 14 from water samples.
Sen Gupta (1943) Calcutta	417	117	300	
Amberson (1945) Calcutta	159	5	154	
Sen Gupta (1951) Calcutta	415 (from 200 patients)	112	302	One strain showed the serological properties of the Hikojima type.

Author and locality	*Total number of strains*	*Inaba strains*	*Ogawa strains*	*Remarks*
Gilmour (1952) Calcutta	190	65	122	In three patients of Gilmour's series the presence of a mixed infection with both Inaba and Ogawa vibrios was noted. In five further instances he recorded "obvious reinfection in the wards", the convalescents in question first excreting vibrios of one race (usually of the Ogawa type) and then, after a vibrio-negative interval, of the other type.
Chakravarty (1954) Calcutta	1811	995	816	In agreement with the now generally accepted view, Chakravarty found no marked differences in the mortality percentages of the patients succumbing to infections with organisms of the Inaba or Ogawa type.

It is important to note that, as shown by the observations of Sen Gupta (1943) covering the period from 1941 to 1943, during that time the incidence of the Inaba strains steadily declined, the proportions being one Inaba-type strain to 154 Ogawa strains in 1943 as against 71 to 76 in 1941. Then, however, the incidence of the Inaba strains again increased to become slightly preponderant in 1954.

Another interesting statement made by Sen Gupta (1951) was as follows:

"In a majority of the cases repeated examinations of stools were done and in most of such cases no change in sub-type was found in repeated isolations. But in 24 cases changes in sub-types were observed. Thus 8 cases showed a change from 'Inaba' to 'Ogawa', 10 cases showed a change from 'Ogawa' to 'Inaba'; 4 cases showed a change from 'Inaba' to 'Ogawa' and back again to 'Inaba'; one case showed a change from 'Ogawa' to 'Inaba' and back again to 'Ogawa'; and one case showed a change from 'Inaba' to 'Ogawa', back to 'Inaba' and again to 'Hikojima' . . . Of the 8 cases showing 'Inaba' to 'Ogawa', one showed the presence of both 'Inaba' and 'Ogawa' types in the same specimen; so also did one of the 10 cases showing 'Ogawa' to 'Inaba'. Of the four cases showing 'Inaba' to 'Ogawa' to 'Inaba', one showed twice the presence of both 'Inaba' and 'Ogawa' types in the same specimen and one showed it once."

Remarkable as these observations are, one cannot share Sen Gupta's belief that he obtained proof of an in vivo transmutation of one subtype

of *V. cholerae* into another. As has been emphasized in this connexion in the fourth of these studies,[1] a transmutation of Inaba strains into Ogawa strains has not been effected even in vitro and appears to be improbable. There can be little doubt, therefore, that the simultaneous presence or successive appearance of the two subtypes of *V. cholerae* observed by Sen Gupta in but 24 of his 200 patients was the result of mixed or successive infections with both kinds of organisms.

(b) Assam. According to Taylor (1941), some strains of the Ogawa type had been isolated in Assam, but Inaba infections prevailed.

(c) Bihar. As stated by Duggal (1949), before the year 1942 the Inaba type of cholera vibrios had been predominant in Bihar, but since that time Ogawa strains had been found to predominate. However, in the Purnea district, Inaba strains were isolated—whether alone or side by side with Ogawa strains is not clear from the context.

(d) Punjab. Examining 55 strains agglutinable with cholera-diagnostic serum, Yacob (1944) found that they were all of the Inaba type.

(e) Bombay. Soman & Neil (1945), testing 164 strains which had been isolated during the 1943 and 1945 cholera epidemics in Bombay, found only three Inaba strains as against 161 Ogawa strains.

(f) Mysore. Testing 63 cholera strains isolated during the 1949-50 outbreaks in Mysore State, Rao and his co-workers (1952) confirmed the presence of 47 Inaba and 16 Ogawa strains.

(g) Madras. Dealing with early observations on the occurrence of Inaba and Ogawa strains in the Madras Presidency (now Madras State), Taylor made the following statement:

> " When a special investigation was carried out in the Madras Presidency in 1936 a series of 89 strains isolated in the previous years were re-examined and four of them were found to be Ogawa sub-type. In the same year it was found that strains isolated in Madras City and the Northern districts of the Madras Presidency were of Inaba sub-type, while the strains from areas in . . . the Southern part of the Presidency were all of Ogawa sub-type. In one area in Madras [? Madura] district 84 strains isolated from cases were studied in detail [2], and all were found to agglutinate to 75 per cent to 100 per cent of titre with a pure Ogawa O serum and to 10 per cent only with Inaba O serum . . . The observations made during the outbreak did not suggest that there was any difference in virulence or epidemiological features in the case of Ogawa sub-type infections as compared with the Inaba sub-type epidemics which are commoner in India.
>
> " Investigations carried out over a period of years in the Madras Presidency have shown extensions and recessions of the areas in which the respective sub-types may be the forms associated with outbreaks. A complete change occurred, for example, in 1940 in which the Ogawa sub-type was the form associated with an outbreak in the Northern Circars, all 252 strains isolated in the area being of this type."

Summarizing the results of further observations on this point in the Madras areas, Pandit (1948) stated that during the period from 1939 to

[1] See *Bull. Wld Hlth Org.*, 1955, 12, 972.

[2] See Venkatraman & Pandit (1938).

1945 (when major epidemics took place in 1942 and 1943), Ogawa strains were almost exclusively present. However, after a year of low cholera incidence a change once more took place, all strains isolated in 1947 and 1948 being of the Inaba type.

Commenting upon these and the previous findings in this region, Pandit pointed out that

" This extension and recession of the area with regard to the prevalence of the types and a complete change-over from one to the other are matters of considerable epidemiological interest and may have a bearing on the cyclical periodicity of epidemic cholera in India."

Egypt

As stated by Gohar & Makkawi (1947) and several other observers, the Inaba type of vibrio was exclusively met with during the 1947 cholera epidemic in Egypt.

As mentioned already, Taylor's above-quoted opinion that outbreaks caused by either the Inaba or the Ogawa type of *V. cholerae* do not differ in seriousness or epidemiological features has now been generally accepted. Nevertheless, systematic observations on the occurrence of these types are of considerable value, being likely to furnish clues in regard to the place of origin of cholera invasions in non-endemic areas or localities. One cannot help noticing, however, that, with the exception of a few areas, no sufficient evidence is available for such enquiries.

Causes of decline of epidemics

Since it is indicated to deal separately with the involved problem of the spread of cholera later in this study, attention can now be devoted to the factors governing the decline and disappearance of epidemic manifestations of the disease.

Dealing with the latter subject in his classical epidemiological study, Flügge (1893) considered it puzzling that the cholera outbreaks did come to an end, even though the reported instances of infection involved only 3%-5% of the population, and consequently many susceptible individuals remained present, while the infection quantum ought to have become maximal after the outbreaks had lasted for some time.

" This paradox ", said Flügge, " is explainable in part by the fact that the season gradually becomes unfavourable for the spread of the epidemic . . . in particular, the individual susceptibility is lowered. Associated with this factor is the influence of the actual extent of the infection [' Durchseuchung ']. This really involves a far larger part of the population than is indicated by the number of reported cases. We may assume that, in addition to the latter, there are very many individuals with very slight infections, which are not recorded but nevertheless confer immunity. Finally, it must also be borne in mind that the people only gradually learn the proper prophylactic measures against

cholera, the necessary care in regard to food and drink, the correct treatment of patients, etc. Those who are careless are, for the most part, attacked by the infection after a few weeks; the careful individuals, who listen to good advice, have learned how to act, and thus there is a gradual falling off both in the careless dissemination of the causative organisms and in the number of susceptible persons." [Trans.]

It is interesting to compare this early statement with the following opinion given by Napier in 1946:

" *The natural subsidence of an epidemic.*—This may be due to the favourable development of the climate, to some change in the water-supply or the availability of another from any cause, or to exhaustion of the clinical material. It has also been claimed that it is effected by the development of bacteriophage in the water supply."

As will be noted, both these observers, though in part making different postulations, were agreed that the subsidence of cholera epidemics was the result not of one factor but of a combination of various factors. These will now be considered seriatim.

Climatic factors

Since, as has been discussed earlier in this study, a high temperature and a suitably high atmospheric humidity promote the epidemic spread of cholera, it is clear that the outbreaks are bound to subside *pari passu* with the appearance of increasingly temperate climatic conditions. The cooler the weather becomes, the less the people will be inclined to consume unsafe cold drinks and foods, which can be easily contaminated with *V. cholerae.* There will be also a decrease of flies, which play a most important role in producing such contamination as well as in conveying gastro-intestinal affections due to other micro-organisms which, as discussed before (see page 793), in their turn are apt to lower the resistance to cholera infection.

An increasing dryness of the atmosphere is apt to exert an adverse influence on the survival of the cholera vibrios on foodstuffs like vegetables and fruits, which play a dangerous role in the spread of the infection.

Recognition and abolition of sources of infection

As will be fully discussed in the last of these studies, indispensable prerequisites for an efficient control of cholera epidemics are: (1) a fully adequate intelligence service having at its disposal a well-equipped and well-staffed laboratory branch to ensure prompt investigation of suspicious patients and the rapid establishment of the diagnosis of cholera; (2) sufficient facilities for isolating the patients so as to cut short the spread of infection by them; (3) a sanitary engineering service to deal with, or, if necessary, to prevent the further use of, contaminated water supplies, furnishing safe water in their place; (4) implementation of measures to control or, if necessary, to prohibit the sale of potentially dangerous cold drinks and foods

and, hand in hand with this, adoption of the necessary methods of fly-control; (5) large-scale public health propaganda to ensure that the people themselves take all necessary precautionary measures.

Carrying out this complicated programme in unforeseen or unexpectedly extensive cholera outbreaks is fraught with great initial difficulties. However, *pari passu* with the creation of adequate facilities for such work the outbreaks are bound to subside. It is obvious that the results of such programmes will be most satisfactory if the period of their maximum efficiency coincides in time with climatic conditions unsuitable for the spread of cholera.

Loss of virulence

As already alluded to in the sixth of these studies,[1] there can be little doubt that the drop in mortality figures often though by no means invariably observed during the later phases of cholera epidemics is not the result of a hypothetical change of virulence but is due to extrinsic causes: owing to a gradual improvement of the case-reporting system more slightly affected patients are detected, while for the same reason those attacked with cholera gravis are more promptly hospitalized and thus become more amenable to treatment. This view has already been advocated by Nichols & Andrews (1908), who stated that during the major cholera outbreak taking place at Manila from August to October 1908,

" The mortality in the last part of the epidemic was only 5 per cent less than that of the first part. This decrease may be much more reasonably attributed to increased facilities for finding and treating cases than to reduced virulence . . . "

Acquired immunity

Observations speaking in favour of the now widely accepted postulation that cholera outbreaks lead to the production of a herd immunity in the populations concerned had been made long before the discovery of the *V. cholerae*. Thus Koch (1885) referred to early experiences in India which had taught that recently arrived European troops suffered much more severely from cholera than the native regiments. Analogously, it was found advantageous to employ Indian nurses in the cholera wards of the military hospitals because they were not likely to contract the infection. Koch further stated that during the Crimean War and the Austro-Prussian War of 1866 it had been noted that the arrival of new troops in places where cholera was on the wane gave new impetus to the infection. Taking account of such observations, the *Cholera-Regulativ* (cholera regulations) by Griesinger, Pettenkofer & Wunderlich (1866) stated that if

" a body of troops has experienced cholera, it acquires in this manner for some time a certain degree of insusceptibility or immunity against the infection." [Trans.]

[1] See *Bull. Wld Hlth Org.*, 1955, 13, 1140.

In Koch's own opinion widespread cholera outbreaks in India engendered a herd immunity lasting for three or four years; then the infection spread once more from the endemic areas along the main route of traffic towards the north-west. Like Flügge (1893—see page 805 above) he emphasized

" that in order to become immune, it is most likely not necessary to suffer from the disease in its most severe form but that a slight attack also confers protection against a second attack; and so I assume that slight cholera attacks, even hardly noted cholerines, which are very frequent at the time of cholera, can also engender an immunity. For this reason a considerably larger part of the population is to be considered as having passed through cholera attacks ['durchseucht'] than one could conclude from the reported case incidence or the mortality figures." [Trans.]

Sharing this opinion, Dunbar (1896) recorded that the examination of the stools of 111 contacts of a total of 15 cholera patients, met with in nine groups on two steamers and in seven houses, had proved positive for *V. cholerae* in 28 instances. Two of these 28 individuals afterwards developed cholera gravis; 11, having solid stools, were evidently carriers; however, 15 presumably had slight attacks of the disease, since they showed signs of diarrhoea.

Observations recorded by Barikine & Cazeneuve (1925) at Rostov-on-the-Don showed that refugees were more susceptible to cholera infection than the resident population. The latter fell ill only after the infection had gained impetus among the former, and the morbidity and mortality was higher in the refugee group than among the residents. However, one should not forget that the refugees, in addition to not being immune to cholera, were presumably also non-specifically less resistant to the infection, as a result of the vicissitudes they had endured during their flight and of continuing to live under unfavourable conditions.

Koch's belief that the herd immunity engendered by cholera outbreaks lasted for three to four years was not shared by other observers. Some of them, like Basil (1910), believed that the state of immunity lasted longer. Flu (1915), on the other hand, stated that it persisted for six months only, while Heiser (1908) spoke in this connexion of a period of two years. It would seem likely that the length of the period is not uniform under all circumstances but depends upon the interaction of variable factors such as the duration and the extent of the outbreaks. While an individual outbreak may fail to produce a solid herd immunity, that immunity may eventually be engendered through repeated cholera manifestations.[1]

[1] The influence exerted on cholera epidemics by bacteriophage action will be discussed in the last of these studies.

Factors Governing the Local Spread of Cholera

Introductory remarks

As soon as the appearance of cholera in Europe facilitated scientific investigation of this disease, attempts were made to classify it according to the ideas of the two long-established schools of epidemiologists—the " contagionists " and the " localists ". The former insisted that cholera was a highly contagious disease, to be guarded against by a rigid system of measures consisting of isolation of the patients as well as of elaborate methods of disinfection and quarantine. The " localists ", on the other hand, maintained that the manifestation of cholera as well as of other infectious diseases was dependent upon local " miasmatic " factors and, to the delight of those interested in traffic and commerce, considered all the above-mentioned control measures unnecessary. As Flügge—from whose masterly publication the statements just quoted have been culled—noted, the constant tug-of-war between the two schools is well shown by the following quotation from a 1831 summary by Lichtenstädt:

" The subject of contagion has been much debated. The non-contagionists have found many adherents among the medical men and gained the support of the public, which sees in quarantine work only what is irksome and disadvantageous. Though one must unhesitatingly admit that the system of cleansing measures led to exaggerations, an unbiased observer cannot consider this as great an evil as the possibility of a spread of this disease . . . May there be finally an end to the often reiterated postulation of the non-infectivity of cholera! It belongs in the category of the most dangerous errors of our time." [Trans.]

As summarized by Flügge, Pettenkofer—who became the protagonist of the " localists " through a voluminous series of publications which began to appear in 1855—conceived the following hypothesis:

The still unknown causative organism of cholera (x) was spread through human traffic not only by patients but also by healthy persons. However, in order to become capable of causing infection (" infectionstüchtig "), it had to reach a soil possessing suitable characteristics (y), the favourable condition of the soil depending mainly upon a low ground-water level, which leads to some desiccation of the superficial layers.

The pathogenic agent resulting from the union of x and y (called z) was supposed to produce air-borne infection in man. If the factor z was imported by patients or healthy people into a hitherto cholera-free locality, sporadic infections might result. However, epidemics could develop only when the soil of the locality contained the y factor and when the time was ripe for an outbreak (" zeitliche Disposition ").

Though this hypothesis obviously became obsolete as soon as Koch had discovered the cholera vibrios and had proved that the faeces of the patients, since they teemed with the organisms in a virulent form, served as the immediate vehicle of the infection, Pettenkofer and his school tenaciously

adhered to their ideas. However, their attempts to adapt the facts to the Procrustean bed of their surmises were convincingly opposed by Koch at the 1885 cholera conference and Pettenkofer's views were also refuted by unbiased observers like Virchow (1885) and Liebermeister (1896). Nowadays Pettenkofer's theory is merely an historical curiosity even though his concept of an epidemic spread of cholera promoted by suitable local and seasonal influences remains basically sound.

As is now generally accepted, the local spread of cholera infection is governed by the following factors.

Contact infection

Though invariably contracted in the immediate vicinity of cholera patients, infection by " contact " may be effected in various ways. The causative organisms may be introduced into the mouth of those near the sufferers by fingers soiled with the faeces of the latter, particularly if those attending the patients partake of meals without having properly washed or disinfected their hands. But in other instances the infection is not quite so direct, things like eating-utensils or foodstuffs, which had become contaminated with *V. cholerae* while kept in the vicinity of the patients, serving as vehicles of the infection.

Assessing the importance of contact infection in the spread of cholera, one must first of all fully agree with the statement of Koch (1893) that this disease is by no means as highly contagious as, for instance, smallpox and measles, " in which simple contact or even a passing sojourn in the sickroom suffices to produce infection."

This difference is easily explainable when it is considered that oral introduction of the causative organisms is the only means of effecting infection.

Opinions of different authors regarding the frequency with which contact infection as defined above takes place in cholera outbreaks vary widely. Some consider it a frequent or even the common means of the spread of epidemics, while others assert the rarity of this mode of infection, pointing out in particular that the disease does not usually show a tendency to familial spread, infection in one inhabitant of a house as a rule remaining confined to that person.

Scrutinizing this discrepant evidence more closely, one can easily perceive that these marked differences depend upon the conditions under which the population groups concerned live. It is true that, generally speaking, cholera shows no tendency to spread in ordinary households. However, contact infection may become rampant in premises where people live crowded together under particularly insanitary conditions, as, for instance, in camps for pilgrims or seasonal labourers or in badly managed billets for soldiers. This state of affairs was recognized at an early date by Snow (1855), who spoke of the " communication " of cholera

" in the crowded habitations of the poor, in coal-mines and other places, by the hands getting soiled with the evacuations of the patients, and by small quantities of these evacuations being swallowed with the food, as paint is swallowed by house painters of uncleanly habits, who contract lead-colic in this way."

The cholera epidemics arising through contact under such particularly unfavourable living conditions may be of an explosive nature. Generally speaking, however, the curve of contact epidemics, as distinct from major water-borne outbreaks, tends to be flat, the infection creeping from one group of people to another (Koch, 1893).

While contact infection does not seem to play a generally important role in the epidemic spread of cholera in India, it is of great importance for the perpetuation of the infection in sporadic form in the endemic foci (Napier, 1946, 1951).

Water-borne infection

Introductory remarks

As alluded to in the first of these studies,[1] most convincing epidemiological evidence of the role of contaminated water in the spread of cholera was furnished by Snow (1855). His inquiries left no room for doubt that a most violent local outburst of the disease in 1854, during which in the immediate vicinity " of the spot where Cambridge Street joins Broad Street there were upwards of five hundred fatal attacks of cholera in 10 days", stood in causal connexion with the consumption of the water from the Broad Street pump. Equally illuminating was the difference of cholera incidence in the houses supplied respectively by the Southwark and Vauxhall Company, obtaining their water from a polluted part of the Thames river, and by the Lambeth Company, which had recently removed their intake to Thames Ditton, " thus obtaining a supply of water quite free from the sewage of London ". The figures in question were thus tabulated by Snow:

Water supplied by	*Number of houses*	*Deaths from cholera*	*Deaths in each 10 000 houses*
Southwark and Vauxhall Company	40 046	1 263	315
Lambeth Company	26 107	98	37
Rest of London	256 423	1 422	59

Thus, Snow commented,

" the mortality in the houses supplied by the Southwark and Vauxhall Company was therefore between eight and nine times as great as in the houses supplied by the Lambeth Company; and it will be remarked that the customers of the Lambeth Company continued to enjoy an immunity from cholera greater than the rest of London which is not mixed up as they are with houses supplied by the Southwark and Vauxhall Company."

[1] See *Bull. Wld Hlth Org.*, 1954, 10, 441.

In contrast to the opinion usually held that polluted water merely favoured cholera infection " by predisposing or preparing the system to be acted on by some unknown cause of the disease existing in the atmosphere or elsewhere ", Snow asserted

" that, if the effect of contaminated water be admitted, it must lead to the conclusion that it acts by containing the true and specific cause of the malady."

It was presumably due to the influence of Pettenkofer's theories that the clear-cut evidence of Snow was not, or at least not fully, accepted. Thus Griesinger (1857), while admitting the possibility of water-borne infection, considered it to be exceptional. A subsequent claim by Farr (1866) that defects in the filter plant of the East London waterworks, which had become grossly manifest by the presence of small fish in the tap-water, were responsible for a cholera outbreak was also received with quite unwarranted scepticism.

No notice seems to have been taken either of the following interesting observations recorded afterwards by Dehio (1892). In Reval (Russia) there was a cholera outbreak in 1871, which terminated on 21 November. In the course of December the cesspools of the city were voided and their contents were deposited outside the city on snow-covered ground, through which ran a canal servicing a city waterworks. Warm weather setting in at Christmas led to an inflow of the melted snow into the canal and cholera immediately became recrudescent in Reval, remaining, however, restricted to the precincts in which water from the canal was utilized.

However, irrefutable proof of the occurrence of *V. cholerae* in the water supplies used for human consumption and of the epidemiological importance of such contamination was furnished through observations of Koch, who reported thus on his initial findings in India at the 1884 cholera conference:

" I succeeded . . . in finding the comma bacilli with all their characteristic properties in a tank which supplies water for drinking and household purposes for all persons living round it and in the immediate vicinity of which a number of fatal cholera cases had occurred. As was established later, the linen of the first cholera victim succumbing nearby had been washed in the tank . . . On its shore there were 30-40 huts inhabited by about 200-300 persons and 17 of these had died of cholera. How many had been ill with cholera could not be established with certainty." [Trans.]

As further stated by Koch, a corollary to these observations was that the opening of the Calcutta waterworks in 1870 had been followed by a two-thirds reduction of the cholera incidence in the city, while the incidence of the disease in the suburbs remained unaltered. That the city did not become quite free from cholera was due to the fact that a considerable part of the population continued to use river or tank water. Fort Williams could be kept entirely free from cholera through the exclusive use of tap-water. Similarly Pondicherry ceased to suffer from the infection after the installation of artesian wells.

Koch's findings were confirmed by a number of other early observers (see summaries by Kolle, 1904, and by Greig, 1929). One must agree with the statement of Greig that owing to the unavailability of serological methods these early observations cannot be considered fully authentic, but there is no reason to deny the validity of most of them, the less so because on numerous occasions the presence of *V. cholerae* in water supplies has been confirmed by modern workers using exact methods of identifying the organisms. Referring to recent exhaustive investigations made in this respect in the course of a field enquiry in Bengal, Taylor (1941) made the important statement that

" *V. cholerae* was not isolated out of contact with the cholera case except on one occasion, although it was frequently isolated in the immediate vicinity of cases."

Kinds of water supply involved

(1) Waterworks water. Though, as described above, a strong case for a role of waterworks water in the spread of cholera had already been made through some early observations, particularly those of Snow and Farr, there is much reason to consider the 1892 cholera outbreak at Hamburg as the classical example of this mode of infection.

As can be gathered from an excellent summary by Kolle (1904), Hamburg at that time quite unbelievably possessed no facilities for filtering the water of the river Elbe from which the city waterworks drew their supplies. Thus with the aid of an intake the unfiltered river water was led into conduits (" Kanäle ") and then pumped into the pipe system. True, the intake was situated above the city but, owing to the tides, it was regularly reached by waves from the port. How the initial infection of the latter took place could not be definitely established, but a role of Russian emigrants, who had come from cholera areas existing in the east and had been housed in sheds near the port, was suspected with much reason.

For several weeks after the commencement of the outbreak only sporadic cases, involving almost exclusively the port area, were recorded. However, on 20 August, when, no doubt, the infection had reached the pipe system, an explosive spread of cholera commenced, the number of daily admissions reaching about one thousand by the end of the month. As stated in the first of the present studies,[1] the total number of patients recorded during the outbreak was 19 891, with 7582 deaths; at the same time, as shown by a tabulation in that study, the two adjacent communities of Altona and Wandsbeck, which derived their water supplies from other sources, suffered markedly less. Thus, as justly emphasized by Kolle, these observations furnished convincing proof of Koch's postulation that the spread of cholera was mainly due to contaminated water supplies and at the same time showed

[1] See *Bull. Wld Hlth Org.*, 1954, **10**, 450.

the untenability of Pettenkofer's theory. Kolle stated in this connexion the following:

"As is known, the political borderline between Hamburg and Altona is actually completely inapparent. It exists only on the map; the transition from one of the cities to the other is so imperceptible that usually one cannot recognize the borderline, for instance, whether in a given street one is in the territory of Hamburg or Altona. In the maps, on which all cholera cases had been shown, one found—not surprisingly for the adherent of Koch's theory—that the distribution of the cholera cases coincided strictly with the territory of the water supply and was limited within the political border which also formed that of the water supply system. It was observed that on one side of a street numerous cholera cases occurred, whereas the other side remained completely free. Both sides of the street were on the same ground, had the same subsoil, the same sewage system; over the street was the same heaven, shone the same sun; and nevertheless one side remained free from cholera, whereas on the other side numerous cholera cases occurred. Thus the houses and the inhabitants of this street had everything in common except one thing—the water supply system." [Trans.]

Further noteworthy observations on the role of water derived from faultily functioning filter-plants of waterworks in the causation of cholera outbreaks have been recorded by Koch (1893) at Nietleben in Germany (see below), at Astrakhan and in St. Petersburg by Kraus (1909), and at Shanghai, China, in 1926.[1] As noted below, full bacteriological proof of the role of the water was obtained at Nietleben and the same was true of the epidemics at St. Petersburg and at Shanghai. No details are available in regard to the Astrakhan outbreak.

The epidemic in the asylum for the insane in Nietleben lasting from 14 January to 13 February 1893 and causing 122 cholera attacks with 52 deaths has been described in great detail by Koch (1893). How the infection was imported into the asylum could not be definitely established, but the fact that the disease quickly became apparent in different parts of the institution and also involved persons not receiving their food in the establishment attracted attention; moreover, there was no cholera in the clinical institutions of Halle receiving the same food supplies. A water-borne infection seemed probable, therefore, and proof of its existence could be obtained. Evidently the faeces of the initial cholera patients had contaminated the sewage water, which, owing to the cold weather prevailing, could not be disposed of properly by the irrigation fields. As a consequence unpurified sewage water found its way into the river, which also served as source of water supply for the waterworks and which, as was found, was not properly filtered in the latter. Soon after the cholera outbreak had commenced, the presence of cholera vibrios could be demonstrated in the supposedly filtered water as well as in samples of the sewage water taken (*a*) before it reached the irrigation fields, (*b*) on the latter, and (*c*) from the effluent.

As Koch commented with great reason, the uneven distribution of the cholera attacks caused by contaminated waterworks water, which rendered

[1] See *Bull. Wld Hlth Org.*, 1955, **12**, 331.

many observers sceptical regarding the importance or even the existence of this mode of infection, was conditioned by various factors. It was well established that the individual susceptibility to cholera infection varied greatly. Moreover, Koch continued,

" the possibility of water-borne infection for different people was bound to be markedly different according to their relation to the water. The one does not drink it at all and comes only into indirect contact with it owing to its use for household purposes and is, therefore, correspondingly less exposed to the danger of infection than another who drinks the water. But even in regard to the latter it is of importance whether much or little water is drunk; and at what time it is taken, when the stomach is empty or full; whether the stomach and the intestines function properly; whether excesses have been committed, etc." [Trans.]

On the other hand, Koch emphasized, the distribution of the cholera vibrios was evidently not so uniform that each drop or each gulp of water contained the causative organisms. Moreover, even if the vibrios were at first evenly suspended, their distribution could afterwards become uneven. For, Koch said,

" One could easily imagine that they, like other bacteria, occasionally adhere to solid objects, e.g., to the inside of a pipe system, which will be the case particularly if the movement of the water is slowed down temporarily or permanently. They [the vibrios] can then perish at the place where they have settled down or they may be torn off again by a stronger current. Generally speaking, the unequal movement of the water in a pipe system must exert a considerable influence on the transport of the cholera bacteria and for this reason alone one pipeline may bring many and another few of the organisms to the corresponding houses. If the houses of the latter kind happen to be tenanted by well-to-do people, whose habits of life render them little vulnerable to cholera infection, it may happen that whole rows of houses or even streets remain free from the disease." [Trans.]

Regardless of such inequalities of distribution, cholera outbreaks caused by a contamination of central water supplies are invariably of an explosive nature, their extent depending upon the size of the area supplied by the waterworks in question.

(2) " *Riverine* " *cholera.* An excellent explanation why " riverine " cholera, i.e., infection contracted by the consumption of the raw water of rivers, plays a most ominous role in the spread of the infection not only in India but also in some other countries, particularly in central and south China, was given by Benjamin (1949) thus:

" (*a*) Rivers, their tributaries and even nullahs form the main, or often the only source of water supply on their bank.

" (*b*) Even where an alternative water supply, e.g. wells, exists, there is a tendency on the part of the inhabitants to take the river water for drinking and domestic purposes, specially when the well water is slightly brackish or hard. [1]

[1] In the Szechwan Province of China the inhabitants of some communities indignantly refused the proposal to utilize, instead of the heavily polluted river water, rain water collected in cisterns, because the latter, while running over the roofs, could become defiled by the excrements of birds!

" (*c*) In certain towns which are places of pilgrimage, even though a piped water supply is provided, there is a tendency for pilgrims to drink water direct from the river as the rivers are considered to be sacred . . .

" (*d*) Gross pollution of rivers by utilisation of banks, or dry portions of beds of rivers for purpose of nature, washing and bathing and washing of utensils in the river, and what is more dangerous, the washing of infected clothes and utensils during cholera outbreaks; entry of sullage from the town or village into the river, and washing of dirt and filth from the town surface into the river by heavy showers. The continuous pollution of a river or stream, specially due to the washing of infected clothes and materials in it, tends to prolong the duration of the outbreak."

Earlier observations in Europe (see, for instance, Flügge, 1893) as well as recent experiences in south China have shown that riverine cholera is particularly apt to affect people constantly dwelling on boats, who, because they have to rely almost invariably upon the consumption of the raw river water, can easily contract the disease, and whose faeces, voided almost or quite directly into the rivers, form a most dangerous means of maintaining and spreading the infection. Early and considerable cholera outbreaks among boat-dwelling populations in southern Chinese cities were therefore quite common (see, for instance, Turnbull, 1938).

As exemplified by the isolation of *V. cholerae* from the water of the Yuan river (Hunan Province, China) in the immediate vicinity of boats filled with night-soil from a cholera-infected city (Robertson & Pollitzer, 1939), such transport, which is usually made without any proper precautions, may also lead to a contamination of rivers.

The character of cholera manifestations caused by a contamination of rivers varies considerably. If they develop in a pilgrimage centre at a time when hosts of pilgrims bathe together in the rivers and simultaneously drink the sacred water, explosive epidemics may result and—as will be discussed later—may lead to a wide spread of the infection. Often, however, continued contamination of the river water by the faeces of cholera patients may lead to the setting up of a vicious circle, the perpetuated interchange of the causative organisms to and from the water resulting in much less violent but very protracted outbreaks.

(3) Irrigation channels. In some areas irrigation channels play a dangerous role in the spread of cholera. This is particularly true of a large part of Madras Province (Madras State), where according to Mathew (1949),

" smaller irrigation channels pass through every village in the area. Almost each house has direct access to the channel. The channel water is grossly contaminated by personal ablution, washing of clothes and vessels, washing of animals, etc. Even the clothing and bedding soiled with cholera excreta are washed in them and yet the people drink this water as such as they find it to be more tasty than well water."

(4) Tanks and ponds. The early claim made by Koch (1884—see page 812 above) regarding the dangerous role played by village tanks (ponds)

in the spread of cholera, recorded at a time when no fully reliable methods were available for the identification of *V. cholerae*, has been fully supported through adequate modern investigations in both the endemic and the non-endemic areas of India. Discussing the role of these sources of drinking-water supply in the non-irrigated areas of Madras State, Mathew (1949) recorded that the ponds and tanks

" are seldom properly protected or conserved. Their water level will be very low during the period March to June, and many of them may even dry up. This is also the festival season and the period of maximum atmospheric temperature. Any cholera infection of the water source at that time will give rise to an explosive outbreak of the epidemic as the dose of infection is more concentrated."

It would seem, however, that explosive outbreaks of this nature are exceptional rather than the rule. Usually a repeated contamination of tanks or ponds by means identical with those responsible for cholera infection of rivers and irrigation channels tends to lead to protracted outbreaks.

(5) Pumps. Observations in the role of the water of pumps (" Brunnen ") in the causation of cholera outbreaks were made after discovery of *V. cholerae* by Koch (1893) and by Zirolia (1913) who recorded the following interesting findings.

The outbreak described by Koch (1893), which, occurring towards the end of January 1893, was responsible for nine attacks with seven deaths, took place in a group of houses of Altona not provided with waterworks water, so that a pump served as source of water supply. A sewage system had been installed in the locality but this failed to function in the winter of 1893, when owing to the cold weather both the gullies and the ground round them became solidly frozen. As a consequence waste water and sewage were bound to run into the inadequately protected pump shaft and it was obviously in this way that the water of the pump became contaminated with the dejecta of one of the few earlier cholera patients observed in the immediate vicinity of the pump. An examination of the pump water at the end of the outbreak (31 January) revealed the abundant presence of cholera vibrios. While specimens subsequently taken from the pump proved negative, the organisms were found to persist for 18 days in one litre of the originally examined water kept in the laboratory at a temperature of 3°-5°C.

The pump-water-caused epidemic at Sori near Genoa, Italy, observed by Zirolia took place in the summer of 1911, when cholera was prevalent in the country. Evidently the small river flowing through Sori became contaminated with *V. cholerae* through the washing of the clothes of a cholera-suspect patient, and water from the river seeped through into an adjacent spring, which fed the pump serving almost exclusively at that time as a source of water supply for the town. A total of 31 cholera attacks was recorded subsequent to the contamination of the pump water, but only 27, occurring from 14 to 20 August, seem the direct result of this contamina-

tion, while the last four patients were apparently infected by contact. Laboratory examination proved the presence of cholera vibrios in the water of the pump. In a specimen of this bacteriologically positive water kept in the laboratory at a temperature of 16°-22°C the organisms were found to survive for 62 days without impairment of their agglutinability, but showed a decrease in their virulence for intraperitoneally infected guinea-pigs.

(6) Springs. As recorded by Lara (1927), an explosive cholera outbreak, involving 25 patients and causing 23 deaths, on the island of Romblon in the Philippines could be traced to the consumption of water from a spring found to have been contaminated with *V. cholerae*—presumably through the dejecta of an earlier unidentified sufferer from the disease. The level of this spring had become so low before the outbreak that, due to the disturbance of the bottom by the dipper, its water was usually turbid. This low water level must have led to a high concentration of the causative organisms, which no doubt accounts for the violence of the outburst. *V. cholerae* could also be demonstrated in two other springs, probably because they had been used for washing the clothing of cholera patients. Since, however, the water of these two springs was not used for drinking purposes, their contamination does not seem to have proved dangerous for man.

(7) Wells. As shown by observations in several cholera-affected areas, particularly in parts of India (e.g., in Bombay State) and in south and central China, wells may, for various reasons, play a dangerous role in the local dissemination of the infection. If the wells are not or inadequately protected or faultily constructed in general, they may be contaminated with materials containing *V. cholerae*, which had been deposited nearby and may either be washed into the mouth of the well from the surface or may reach the interior of the well through seepage, such entries of the polluted materials being particularly likely to occur during showers. Faulty methods of collecting the water may render even properly installed wells dangerous. Thus in China the wells were as a rule not provided with permanent fixtures to draw their water, so that the people had to bring buckets and ropes of their own, which, since they were ordinarily kept in the houses in a rather careless manner, could quite easily become contaminated during outbreaks with the dejecta of cholera patients or otherwise come in touch with materials containing the causative organisms. Repeated contaminations of the well water could thus be effected if cholera became manifest successively in various households obtaining their water supplies from the wells in question.

While shallow wells are generally involved in the local dissemination of cholera rather than the deep wells which are almost invariably far more solidly constructed, this rule is not without exceptions. For instance, as recorded by Sian (1931), during an outbreak taking place in 1930 in a

badly sanitated area in the Philippines, three out of 10 artesian wells examined for the presence of *V. cholerae* yielded positive results.

Successive contamination of the often numerous wells used in cholera-affected localities may contribute to the occurrence of outbreaks of a protracted type. At the same time, however, the manifestations due to the consumption of water from individual cholera-contaminated wells, though as a rule limited in extent, may be of an explosive nature. The occurrence of such localized outbursts is well illustrated by an observation recorded by Takano and colleagues (1926), according to which consumption of the water of a cholera-contaminated well led to the infection of five individuals within less than 24 hours.

(8) Water supplies on ships. As exemplified by observations of Brau (1905) and of Defressine and co-workers (1912), cholera contaminations of the water supplies stored on board ship may be the cause of outbreaks of the disease among the crews of the vessels. In marked contrast to these experiences, the cholera manifestations observed during the 1892 epidemic aboard two ships moored in Hamburg appeared to be due to the consumption of water currently taken from the Elbe river (see Koch, 1893).

(9) Sea-water. Claims that cholera-contaminated sea-water plays a most dangerous role in the causation of outbreaks of the disease have been made by Japanese observers. As Takano and his colleagues stated in this connexion,

> " Cholera in Japan has always been thought to be due to the pollution of sea-water by ships from abroad and by carriers and cholera patients landed in Japan. The improvements in preventive measures in recent years facilitate the early detection, diagnosis, isolation and disinfection of cholera patients. Therefore, only occasionally do patients on land infect persons with whom they come in contact. The quarantine on land is almost complete. When cholera breaks out in a port it spreads along the coast, and it is very rare to see the disease carried into provinces far from the coast. Thus cholera in Japan is spread directly or indirectly by polluted sea-water."

General considerations

Origin of the cholera contamination of water supplies. Attention will be drawn in the following section of this study to claims made to the effect that, if contamination of rivers or other water courses with *V. cholerae* has taken place at one point, the organisms could be carried by the current to communities lying downstream from the localities originally affected and that in the communities thus reached water-borne cholera outbreaks could follow. As will be discussed, the evidence adduced in this respect cannot be considered fully convincing. Moreover, even if such statements could be accepted at face value, it is clear that such " primary " contamination of the water supplies in individual localities could occur only because previously in another locality of the area in question there had been an invasion of the water supplies by cholera vibrios derived from human sources. That

the presence of cholera in man is thus an indispensable prerequisite for the contamination of water supplies, which then secondarily become the vehicle of the infection, has been proved by many observations showing that the water samples invariably yielded positive results for *V. cholerae* only after the disease had become manifest in man. Results of large-scale studies proving this point have been recorded for instance by Dunn (1929) and Read & Pandit (1941) in India, and similar findings were made by the present writer in the course of a prolonged investigation of the vibrio fauna of the Shanghai surface waters. Dealing with this problem in a general manner, Greig (1929) quoted the following interesting statement by Houston (1913):

" If the immediate cause of what are recognized as ' water epidemics ' in the past could be precisely ascertained, I believe in most cases it would be found that accidental infection of the supply by what is known as a ' porter ' or ' carrier ' of disease had occurred."

In Greig's own opinion,

" The human reservoir is in a position to supply an adequate dose of poison to various distributing channels—water, milk, flies, etc., and so initiate epidemics of cholera. It will be seen that the problem of the prevention of cholera is, shortly stated, the protection of mankind from man."

Mechanism of infection. Discussing the means by which cholera-contaminated water was apt to produce the disease in man, Flügge (1893) made the following important statement: [1]

" The infection by means of water which contains comma bacilli can be due to its mere use for cleaning the eating and drinking utensils, rinsing the beer glasses, etc. Certainly by far the most frequently it is produced through *drinking* of the water in question. The more the heat of the summer increases the urge to drink water, the more frequently does this mode of infection come into question. It is all the more dangerous because the comma bacilli in a drink of cold water probably pass the stomach most easily without becoming damaged. If water is introduced into the stomach . . . a small amount immediately passes into the small intestine; after about an hour there is a rapid passage of the remainder; but even this does not show an acid reaction, so that the comma bacilli have had no damaging influence and could enter the small intestine unharmed. Hence water is the substrate in which the comma bacilli are best preserved and in which they can reach the small intestine most easily. The numerous epidemiological observations, the repeated demonstration of the comma bacilli in water, and finally the special ability of water to lead the bacilli without harm through the stomach, render it quite certain that drinking water plays a *prominent* role in the spread of cholera." [Trans.]

Persistence of V. cholerae *in contaminated waters.* Claims that cholera vibrios may survive for prolonged periods in contaminated waters have been made by some workers. Thus Defressine & Cazeneuve (1913) maintained that (*a*) running waters may remain infected with *V. cholerae* for one month without reinfection; (*b*) the organisms were able to survive in

[1] Compare a similar statement by Koch (1893), quoted above on page 815.

the mud and slime of the river bed in Toulon, France, for as long as six months, and (*c*) this prolonged persistence of the cholera vibrios in surface waters was of great epidemiological importance. It is not possible, however, to place much reliance upon these findings, made at a time when no reliable methods were available for a differentiation of the true cholera vibrios from cholera-like water vibrios. In the experience of most workers, particularly during warm seasons, when cholera epidemics usually occur, the period of survival of *V. cholerae* in natural sources of water supply has appeared to be much shorter than the two French workers assumed.[1] Read & Pandit (1941), who examined numerous water samples collected from various sources in areas of Bengal where cholera was present, noted in this connexion that only about half (9) of 17 initially cholera-positive specimens showed evidence of the persistence of the organisms for more than five days and that the maximum period of their survival did not exceed 16 days.

As far as the present writer feels entitled to judge, however, he is led to believe that the question of the length of survival of *V. cholerae* in natural sources of water supply possesses less practical importance than is sometimes assumed, because at the time of an epidemic prevalence of cholera the role of the contaminated water supplies appears to depend upon their repeated reinfection rather than upon a single invasion of the causative organisms. This idea seems to have been advocated at an early date by Hart and colleagues (1910) who stated that

> " The persistence of cholera in a district is indicative of more than a single pollution of the water supply, and generally points to a persistence of some insanitary conditions which favour repeated infection."

Similarly Robertson & Pollitzer (1939), judging from ample observations on water-borne cholera outbreaks in China, came to the conclusion that

> " Though there can be no doubt that river water contaminated with cholera vibrios formed the most important vehicle of infection, we are far from asserting that it served as a permanent reservoir of cholera. We believe that a kind of vicious circle existed; infected faeces contaminated the river water which in its turn produced human cases."

It is possible, however, that the persistence of the cholera vibrios in contaminated water supplies is of some importance in helping to carry over the infection in the endemic areas.

Role of contaminated food and drink

General considerations

Food and drink destined for human consumption may become dangerous vehicles of cholera infection in various ways, most important among which are the following:

[1] See *Bull. Wld Hlth Org.*, 1955, 12, 856.

(*a*) A dangerous practice adopted in some countries is to use fresh night-soil as manure for lettuce and other vegetables destined to be consumed raw or after inadequate preparation, such as short pickling. As maintained by Takano and colleagues (1926), for instance, the infection may be spread by this means at the time of cholera epidemics in Japan.

(*b*) Foods like salads and jellies, to be consumed cold, as well as lemonades or other cold drinks, may become dangerous if water polluted with *V. cholerae* is used in their preparation. It is obvious that such polluted water is likewise apt to prove dangerous if it is used for cleaning articles like vegetables and fruits to be consumed without cooking.

(*c*) It has also been claimed that the handling of foodstuffs by cholera patients may lead to a spread of the infection. Thus Seligmann (1918) maintained that a small cholera outbreak at Berlin in 1918, ascribed to the consumption of raw or improperly cooked horsemeat by most of those affected, stood in causal connexion with the handling of the meat by a butcher who on the following day succumbed under circumstances suspicious of cholera. It is also noteworthy that Eugling (1938) claimed to have observed cholera infections among soldiers who, acting against orders, had eaten uncooked corned beef from open tins they had found in the possession of cholera victims. However, the validity of these observations, especially that of Eugling, is doubtful, and it is certain that instances of infections produced in the manner described by him or by Seligmann, if they really occur, must be rare.

(*d*) Cholera contamination of food or drink to be consumed cold may occur while the articles in question are stored or offered for sale. They may be put in containers which have become contaminated with *V. cholerae*—for instance, by having been cleaned with cholera-polluted water—or they may become dangerous because they have been kept in the vicinity of cholera patients. A more common source of contamination is that articles like fruits or vegetables for sale are freshened up by the merchants or hawkers by sprinkling them with cholera-polluted water. Another most dangerous practice is the frequent one of offering cold food or drinks for sale without protecting them against flies.[1]

As alluded to above (see page 806), an adequate degree of moisture of foods like vegetables and fruits, which depends in its turn upon a sufficiently high atmospheric humidity, is an essential prerequisite for the survival of the cholera vibrios on these articles. This was clearly recognized by Flügge (1893) who stated that

" Foodstuffs, if kept in a moist condition, can long preserve the comma bacilli deposited on them by contact or through flies . . . On many of these foodstuffs a multiplication of the comma bacilli occasionally seems to take place. However, the temperature, the

[1] The role of flies in the spread of cholera, already dealt with in the sixth study (*Bull. Wld Hlth Org.*, 1955, 13, 1134), will be further discussed in a later section of this study.

degree of moisture of the substrates and the competition of saprophytes exert a very great influence in this respect, and usually little more seems to result than a preservation of the germs which, however, fully suffices for infection." [Trans.]

Fish and shellfish

The reasons for the important role played in the spread of cholera by fish and shellfish are that (*a*) in view of the frequent cholera pollution of the waters in which they live, they can easily become infected, or at least contaminated, with *V. cholerae*; (*b*) once the organisms have gained an entry, they are apt to survive for a considerable length of time or even to multiply; [1] and (*c*) in certain countries, particularly in Japan and in the Philippines, raw fish form a staple product of the diet of the population, while shellfish, especially oysters, are consumed in the raw state on a fairly universal scale.

Early reference to the great epidemiological importance of fish in Japan was made by Dönitz (1886), who was able to establish that (*a*) during the 1877 cholera outbreaks involving Tokyo and its environs the disease raged with quite particular intensity in the coastal village of Haneda, through which most of the sea-fish destined for the Tokyo market passed; and (*b*) cholera repeatedly became manifest en route among the fishermen bringing cargoes of fish by boat to the capital.

" It is unnecessary to state in detail," Dönitz remarked,

" how much the merchandise became defiled, since it would have been difficult for the boatmen to avoid contamination of the fish, even had they been fairly competent bacteriologists. Once the fish had become infected, they could spread the disease the more easily, since in Japan the meat of some fish is sometimes consumed raw, being considered to be more tasty in this state." [Trans.]

Dönitz mentioned the possibility that people in Tokyo coming in touch with the diseased fishermen might have contracted the infection by contact, but there can be little doubt that the contamination of the fish played a preponderant, if not an exclusive role, because outbreaks due to the latter cause continued to be common in Japan (see Kabeshima, 1918, and Takano and co-authors, 1926, quoted in the sixth study [2]). In fact, one of these epidemics, which took place in 1922 and was described in the 1937 report of the Eastern Bureau of the League of Nations Health Organisation, had exactly the same history as the outbreaks observed by Dönitz :

" There had been some severe cases of gastro-enteritis in a sea-port about sixty miles from Tokio and, before a diagnosis of cholera was made, cargoes of fish had been sent to the Tokio fish market and distributed to various fish merchants. As a result, cholera cases were suddenly reported from all parts of the city to the number of nearly thirty cases a day for the short period the epidemic lasted. It was completely checked within two weeks by prohibiting fish from being brought into the city from the port in question."

[1] See *Bull. Wld Hlth Org.*, 1955, **12**, 850.
[2] See *Bull. Wld Hlth Org.*, 1955, **13**, 1132.

Some circumstantial evidence incriminating oysters in the spread of cholera was recorded by Netter (1907) thus :

" In the course of cholera epidemics, the disease on several occasions made its first appearance among persons who had consumed oysters.

" Thus in 1849 a cholera epidemic at Bridgewater and at Taunton became manifest among children who had eaten oysters which had been considered unwholesome.

" In 1893 the towns of Grimsby and Cleethorpes near the mouth of the Humber had cholera cases. A fairly considerable number of persons affected in other parts of England had partaken of oysters from the beds in this locality, either there or at a distance. With each tide the oyster and mussel beds of Cleethorpes received materials carried by the sewers of both towns.

" Thorne, reporting these facts in 1894 in the Local Government Report for that year, expressed the opinion that the ingestion of oysters contaminated in these towns could have been responsible for these cases." [Trans.]

Heiser (1908), discussing the problems of cholera epidemiology in the Philippines, drew attention to the particularly high prevalence of the disease there among the fishermen of Japanese nationality and also referred to observations which had shown that the early victims of the 1907 outbreak in northern Samar " all had eaten a poor quality of dried fish taken from waters in Manila that were presumably infected ". He added the following curious statement:

" The fishing industry in and about Manila is conducted on a most extensive scale. The low, marshy character of the section of the bay north of the Pasig River and the rise and fall of the tide produce ideal conditions for the growth and contamination of shell and other fish, and this is especially accentuated since the tidal currents are such that the contents of one of the city's largest sewers is extensively distributed throughout this area. Granting then, for the sake of argument, that fish, shellfish, and other sea products become infected with cholera and that through imperfect cooking the organisms are not all killed, and that one of the principal food substances of the masses in the city of Manila is gathered from the section just mentioned, it is evident that it would be possible to immunize a large portion of the city's inhabitants. Enormous quantities of sea products in a dry or partially dry state are also sent to various portions of the Islands from Manila and if it should happen that some of them have been shipped in a moist condition or are otherwise rendered a good culture medium of cholera vibrio, it is conceivable that such products might, in isolated instances, be the cause of appearances of cholera in places such as Samar, Leyte, etc. In view of the fact that this class of food products is the sustenance of the masses because of its cheapness, it is more probable that the underfed members of a community would be the first to be affected by the slight infection it might contain."

Further observations on a role of fish and shellfish in the causation of cholera outbreaks in the Philippines have been recorded by Pottevin & Abt (1925), according to whom the consumption of small fish and crayfish was responsible for the appearance of the disease in 1916; and by Fuentes (1932) who, referring to an epidemic at Barrio San Roque, Lingig Surigao, stated that raw sea-foods (fish, crustaceans, algae, etc.) served as important vehicles of the infection.

Dealing in the 1915 report to the Local Government Board in Great Britain with the subject of cholera, Johnstone stated that the sun-dried fish

largely consumed in the Noakhali district of Bengal, because it was most attractive to flies, evidently played a role in the spread of the infection.

As may be conveniently discussed at the present juncture, some observers postulated that, besides serving as vehicles for *V. cholerae* in the manner described above, fish played a still more important role in cholera by being apt to act as reservoirs of the infection. This possibility seems to have been considered first by d'Herelle and colleagues who briefly stated in their 1930 memoir that

" It is probable that aquatic animals peculiar to Bengal and Indochina may be concerned, annelid, crustacean or mollusc, in the intestine of which the cholera vibrio can live for a very long time and more especially in the intestine of which the avirulent cholera vibrio can become regenerated."

In an elaborate thesis published in 1951 Pandit & Hora postulated that a role in maintaining cholera endemicity in India was probably played by the hilsa fish (*Hilsa ilisha*) because, as summarized in a report on their work embodied in the proceedings of the first session of the WHO Expert Committee on Cholera (1952),

" (*a*) A striking similarity was found to exist between maps drawn to show the main foci of cholera endemicity in India and those showing the areas where the main hilsa fisheries were located.

" (*b*) Some correlation seemed to exist between the movements of hilsa during the various seasons of the year and the seasonal variations in cholera incidence.

" (*c*) Apparently there was also some correlation between the five-yearly peaks in hilsa fishery and the periodicity of cholera in Eastern Bengal.

" (*d*) The requirements suitable for a survival of the cholera vibrio in water, namely, (i) a high organic content of the latter ; (ii) a suitable concentration of salts, and (iii) an absence of the lethal effects of sunrays, seemed to be fulfilled in the normal environment of hilsa which was migrating near the bottom of the rivers.

" (*e*) The methods of handling hilsa fish to prepare them for consumption are compatible with the assumption that these fish may play a role in the spread of cholera, the procedure being not dissimilar to the dispersal of the vibrios in the environment through faecal matter."

In the course of the prolonged discussion of Pandit & Hora's hypothesis at the meetings of this expert committee, the necessity of further studying the possible role of hilsa fish in maintaining and causing cholera outbreaks was unanimously admitted. However, in the opinion of some of the speakers, part of the evidence adduced by Pandit & Hora in support of their thesis could be differently interpreted. In particular it was pointed out that

" (i) the cholera endemicity map was not only similar to the hilsa fishery map but that there was also a remarkably close correspondence between the former and the maps showing the density of population; (ii) the seasonal appearance of cholera in the inland focus situated along the Ganges in Bihar, ascribed by Pandit & Hora to hilsa migrations, might be actually due to the influx of seasonal labourers."

Still more important than these objections is that systematic studies by Krishnan (1953) and by Pillay and co-workers (1954) furnished no convincing proof of the validity of Pandit & Hora's hypothesis.

Krishnan failed to find cholera vibrios in 149 hilsa fish as well as in 317 common food fishes belonging to 32 other species caught in the Hooghly river at three points, the water of two of which yielded *V. cholerae* cultures upon several occasions. Cholera-like vibrios (which abounded in the river water) could be regularly isolated from the fish, but were found to be less abundant in their gall-bladder than in the intestinal and rectal contents. When five of the fishes were kept in the laboratory in vibrio-free water, which was changed daily, excretion of cholera-like vibrios was found to persist only for periods ranging from 2 to 14 days. One of these fishes was found to excrete first cholera-like vibrios for five days, then El Tor vibrios for one day and afterwards again cholera-like vibrios. However, in view of the frequent occurrence of El Tor vibrios in India, it is not possible to share Krishnan's belief that their appearance in this fish was the result of a mutation and not of a mixed infection with both El Tor and cholera-like vibrios.

Summarizing the results of the hilsa fish inquiry conducted under the auspices of the Indian Council of Medical Research, Pillay and colleagues stated that examination of (*a*) numerous specimens collected from the guts and gills of hilsa and other fish, (*b*) many water samples collected from the surface and the bottom of the Hooghly river at the three above-mentioned points, and (*c*) a fairly large number of samples of silt from the river, had given the following results:

Kind of specimen	*Results of bacteriological examination*	
	V. cholerae	*Cholera-like vibrios*
849 specimens from 349 hilsa fish	Negative	Found in 136 specimens
669 specimens from 422 fish of other species	Negative	Found in 267 specimens
935 Hooghly water samples (see text above)	Found 10 times at 2 of the sampling points	Found in 437 samples
102 river silt samples	Negative	Found in 44 samples

The viability of cholera vibrios in the alimentary tract of artificially infected fish was studied by Pillay and co-workers with the following results:

(*a*) " Specimens of the Climbing Perch (*Anabas testudineus*) and Murrel (*Opicephalus punctatus*), which were kept under observations and had been found to have ceased to excrete NAG [i.e., cholera-like] vibrios, were infected by feeding them on artificially infected pupae and larvae of house-flies bred in the laboratory. Each day the fish were removed to fresh sterilized aquaria and the water samples from the old aquaria were examined. But no vibrios were recovered for a period of more than 30 days. The fish were then dissected and their intestinal contents examined bacteriologically. Haemolytic non-agglutinable vibrios were recovered."

(*b*) " In 4 sets of subsequent experiments the Climbing Perch, *Anabas testudineus*, artificially infected with cholera vibrios, were found to excrete NAG vibrios of Heiberg's group II for a period ranging from 2-4 days."

That the cholera-like vibrios isolated during these experiments were *V. cholerae*, which had lost their specific agglutinability—as Pillay and co-workers suggested—is difficult to believe because (*a*) those obtained at autopsy in the first experiment were, in contrast to true cholera vibrios, haemolytic, and (*b*) those of the second experiment belonged to Heiberg's group II and not to group I, as true cholera vibrios almost invariably do.[1]

As will be gathered from the findings recorded above, (1) Krishnan as well as Pillay and his colleagues never succeeded in isolating cholera vibrios from fish caught in the Hooghly river, in the water of which *V. cholerae* had been found upon several occasions at two of the three sampling stations; (2) *V. cholerae* showing the typical properties of this species and agglutinating with specific serum could not be isolated from artificially infected fish either. It is noteworthy that Pillay and co-workers, evaluating the observations they had made in the course of their investigations in regard to the cholera-like vibrios, postulated

" that certain types of NAG vibrios may be mutant forms of the cholera vibrios and be responsible for maintaining cholera endemicity. It can be conceived that cholera vibrios brought into the water from human sources, when consumed by certain fishes, mutate into NAGs and are excreted into the water in that form, where they persist for long periods. These NAGs under certain conditions, as for example, through rapid serial passage through the human intestines, may change back into true cholera vibrios and lead to cholera outbreaks."

However, as has been stated in the fourth of these studies,[2] it is impossible to believe in such a transmutation of " non-agglutinable " cholera-like vibrios into true, specifically agglutinable cholera vibrios. It follows that thus far no convincing evidence has been obtained to support the belief that fish play a particularly important role in cholera epidemiology by acting as reservoirs of the infection.

Role of " fomites "

As alluded to earlier in this study (see page 809), in the past the "contagionists ", believing that cholera was a highly contagious disease, ascribed great importance to a spread of the infection through contaminated inanimate objects, the so-called fomites. During cholera epidemics they insisted, therefore, upon the rigid disinfection of all sorts of articles, including many which are now considered quite innocuous. Even the letters sent out from the affected localities were assiduously fumigated !

[1] See *Bull. Wld Hlth Org.*, 1955, **12**, 821.

[2] See *Bull. Wld Hlth Org.*, 1955, **12**, 1010 et seq.

Dealing with the role of fomites in the spread of cholera at the 1884 cholera conference, Koch refuted the idea that letters and goods in general could serve as vehicles of the infection, but quoted many observations showing that body-linen and bedclothes polluted by the faeces of cholera patients played a dangerous role in this respect—an opinion which was shared by other early workers, for instance, by Dönitz (1886) and by Flügge (1893).

Dönitz quoted in this connexion the following observation:

" During the summer of 1885 a French warship came from Tongking to Japan and landed at Nagasaki. A few hours after the anchor had been dropped, an officer succumbed on board to cholera. His linen was handed to a Japanese washerman, who fell ill with cholera and died . . . His wife also fell a victim to the disease at almost the same time. At once further cholera attacks followed and in a few weeks an epidemic was fully developed ". [Trans.]

Flügge (1893) referred to several other instances in which the contaminated linen of cholera patients apparently served as a vehicle of the infection. He maintained in this connexion that if such substrates became subject to desiccation, the cholera vibrios soon perished, but insisted that

" if the linen is closely rolled together, so that a desiccation of the inner layers is impeded, and if such a bundle is kept at a low temperature in moist air, e.g., in a cellar, one can still demonstrate live comma bacilli after 3-4 weeks, probably even after a longer time." [Trans.]

As pointed out by Flügge, other inanimate objects in the environment of cholera patients besides linen could also become contaminated, including various utensils, carpets and the clothes of those attending the sufferers. While cholera vibrios thus deposited on smooth surfaces succumbed within 24 hours owing to desiccation, the organisms could survive for two days in porous cloth.

The role of fomites in the spread of cholera was discussed at the international sanitary conference held at Dresden in 1893, when the conclusion was reached that, besides contaminated body- and bed-linen, only clothes and the wastage (" Abfälle ") of rags and cloth were apt to serve as vehicles of the infection (see Kobler, 1913). However, as will be discussed in the last of these studies, even this restricted list seems too long.

Role of flies

The results of laboratory investigations showing that flies play an important part in the spread of cholera [1] have been confirmed by many epidemiological observations. The following among the latter deserve special discussion.

Flügge (1893), referring to the problem presently under review, stated that

[1] See *Bull. Wld Hlth Org.*, 1955, 13, 1134 et seq.

" Various observers have shown that flies which have alighted on dejecta or on contaminated linen are capable of transmitting living comma bacilli even after some hours to articles of food. In small habitations without separation between the patient and the kitchen or the store-room, this mode of infection must be of serious importance in late summer and autumn, when masses of flies are present in such quarters." [Trans.]

It will be seen that Flügge was aware of two factors which facilitate the spread of cholera through flies—namely, (1) a seasonal prevalence of these insects, which is apt to give impetus to the propagation of the infection; and (2) nearness of the places where food is prepared or kept to the cholera patients or, as observations in non-European countries have taught, to the latrines.

Buchanan (1897) claimed that a cholera outbreak in an Indian jail was due to flies which had been carried by the wind from some nearby cholera-affected huts into the prison compound. The infection became manifest solely among those prisoners who partook of their food on the side of the jail near the affected huts.

Dealing with the importance of flies in the spread of cholera in the Philippine islands, Heiser (1908) stated that

" Many isolated observations which seemed in some way to be intimately connected with the spread of the disease were made since 1903. It has been noted that when flies are particularly active and persistent and refuse to be driven away, as for instance is the case in the United States before a thunderstorm . . . immediately a considerable increase in the number of cholera cases almost invariably follows."

Attention to the role of flies in the spread of cholera in Java was drawn by Flu (1915). Making collections from 20 houses in which cholera patients had been found, he was able to demonstrate the presence of *V. cholerae* in the flies obtained from 10 of these dwellings. According to Tull (1920), flies were responsible for the spread of a cholera outbreak arising in 1920 in a labourers' camp at Syriam in Burma. The insects were most abundant at the time and the cook-house and the latrines were only 10 feet (3 m) apart.

Ample experiences convinced the present writer that in China as well flies played a prominent role in the spread of cholera. Outbreaks arising or running their course during the seasons of fly-prevalence usually showed a wide spread and there was invariably a marked drop in the case-incidence *pari passu* with the diminution of the flies. As noted by Robertson & Pollitzer (1939), the cholera situation always became particularly dangerous during the time when cut water-melons were offered for sale on the streets or in open shops, and the severity of the outbreaks abated as soon as the melon season had come to an end or the sale of the fruits was prohibited. No doubt cholera contamination by flies played an important role in rendering the cut melons dangerous. However, an additional danger was constituted by the practice of the hawkers of freshening up the fruits offered for sale with rags dipped in raw and often even dirty water.

Referring to the subject at present under review in his valuable study on cholera in the United Provinces (now Uttar Pradesh) of India, Banerjea (1951) made the following statement:

" In this province the worst months for cholera are those in which house-flies abound, *i.e.* summer and autumn months. Another thing which is observed is that while epidemics in rural areas are mostly explosive and localized in character, those in towns and cities with protected water supplies but bad conservancy are protracted in nature with cases widely separated and are restricted to summer and autumn. These facts point to some connection which flies may have in the dissemination of the disease. According to Russell and Sundararajan (1928) the association of high relative humidity with high temperature, accompanied with intermittent rains, forms the most favourable atmosphere for the development of the disease. All these, as pointed out by Ross (1928), are favourable conditions for the rapid multiplication of the flies. Heavy rainfall is unfavourable to the breeding of flies and it is noticed that a temporary decrease in cholera also occurs whenever there is a good rainfall."

Role of carriers

Early observations

The possibility that healthy carriers of *V. cholerae* might play a role in the spread of cholera seems to have been considered long before the causative organism of this disease was detected. Thus Griesinger made the following statement in 1857:

" While the fact that patients suffering merely from diarrhoea can transmit and spread cholera has been indubitably established, it is not possible thus far to decide with full certainty that fully healthy persons coming from the place of an epidemic or generally speaking from a focus of the infection can bring the poison with them. Some observations render this most probable; but there remains the possibility that such apparently healthy individuals had suffered to a slight degree at least from specific diarrhoea." [Trans.]

It will be noted that, while suspecting the existence of healthy carriers of *V. cholerae*, Griesinger also hinted at the possibility that persons who had recovered from a cholera attack might continue to harbour the contagion in their intestinal tract, thus becoming convalescent carriers.

That both these categories of carriers actually exist was confirmed fairly soon after the detection of *V. cholerae*, first apparently during the 1892 cholera epidemic at Hamburg by Dunbar (1896), whose interesting findings may be summarized as follows.

Subjecting 142 stool specimens to smear examination, Dunbar obtained the following results:

Category of specimens	*Number examined*	*Found positive*
Patients with manifest signs of cholera	68	41
Patients with diarrhoea only	47	25
Persons without clinical signs	27	4
	142	70

In this connexion Dunbar made the important observation that the faeces of the carriers contained fewer cholera vibrios than those of the patients. A corollary to this observation is that, as shown by the table inserted below, the faeces of the carriers were not as a rule suitable for making a rapid diagnosis by cultural methods:

Diagnosis established	*Patients with*		*Healthy carriers*
	manifest cholera	*diarrhoea only*	
Within 15 hours	60%	40%	10%
Later	40%	60%	90%

Referring in a later part of his study to the frequency with which healthy carriers were met, Dunbar stated that during a recrudescence of the epidemic they had been found in " quite surprisingly " large numbers among immediate contacts of cholera patients. In his opinion,

" These findings furnish a new clue for the assumption that during cholera epidemics a far larger number of persons temporarily harbour the cholera vibrios than show manifest attacks of the disease. That this could lead to immunization cannot be excluded in the present stage of our knowledge." [Trans.]

As far as Dunbar could judge from a limited number of observations, the causative organisms did not as a rule persist in the stools of cholera patients for longer than five days; however, in two instances faeces of convalescents continued to give positive results up to eight and 10 days respectively. But the validity of these results was not confirmed by findings made by Simmonds (1892) when dissecting numerous victims of the 1892 Hamburg outbreak. For according to these observations *V. cholerae* was still found to be present in more than half of the individuals who had succumbed on the seventh to twelfth day after onset of the disease, though persistence later than that was exceptional, with a maximum of 18 days in a subject who after recovery from cholera had succumbed to pneumonia.

The pioneer observations of Dunbar were soon confirmed by some other workers, such as Metchnikoff (1893), Rumpel (1893, 1894) and Abel & Claussen (1895), who seem to have been the first definitively to establish that excretion of cholera vibrios by carriers was apt to be intermittent rather than continuous.

Investigations up to 1935

The numerous observations on cholera carriers recorded during the present century up to 1935 cannot be accorded full credence, because it was only in the latter year that satisfactory methods for the identification of *V. cholerae* became available. Hence it is impossible to decide whether the earlier workers, especially those few recording a prolonged persistence of the causative organisms in the stools of carriers (for periods of a year or even a little longer), actually were in the presence of true cholera vibrios.

Moreover, the possibility has to be seriously considered that the individuals in question, instead of continuing to harbour cholera vibrios, had actually become reinfected with these organisms. Since, finally, such long periods of harbourage have not been observed either by any of the modern workers or by most of those recording their experiences before 1935, it seems altogether unlikely that cholera carriers harbouring the causative organisms for periods longer than at most some months do exist.

As can be gathered from a study of the voluminous literature on the subject, which has been ably summarized by Khan (1929) and by Couvy (1933), the views held by different authors regarding the importance of carriers in the spread of cholera varied most considerably. Some maintained that even carriers of long standing played a dangerous role in this respect, while others reached the conclusion that the propagation of the infection depended solely upon what they called significantly, though somewhat incorrectly, " acute " carriers, i.e., persons incubating cholera, patients in the actual phase of the disease and those in the early stage of convalescence. The relative merits of these contending views will be considered in the concluding part of the present disquisition.

The statements made by the various authors regarding the frequency with which they had found healthy cholera carriers were also at great variance. Generally speaking, since such carriers were mostly met with among persons in close contact with cholera patients, their incidence was bound to be highest in groups of people living crowded together in underprivileged households, where no doubt unfavourable hygienic conditions favoured the direct or indirect passage of the cholera vibrios from the sufferers to their contacts. Some authors also drew attention to the frequency with which small children were found to be carriers of *V. cholerae*.

Recent observations

Bringing up to date a report rendered by Pollitzer in 1952, essential recent observations on the carrier state in cholera may be tabulated thus:

(a) Maximal periods of vibrio excretion in convalescent carriers

Author	*Number of cases*	*Maximal period of vibrio excretion (days)*
Tao, Woo & Loh (1948)	?	9
Read & Pandit (1941)	1	13
Peterson (1946)	1	17
Gohar & Makkawi (1948)	1	23
Gilmour (1952)	1	25
Ying (1940)	1	21-28
Wilkinson (1943)	1	31
Kordi (1948)	1	33
Shousha (1948)	2	42

(b) Average duration of vibrio excretion in convalescent carriers

Author	*Number of cases*	*Observations*
Ying (1940)	200	Positive up to 7 days: 76.5% Positive up to 10 days: 21.5%
Read & Pandit (1941)	10	Negative within 6 days: 70% Negative within 8-13 days: 30%
Peterson (1946)	1149	Average period of excretion: 5.4 $\pm$ 2.3 days
Reimann et al. (1946)	160	Excretion period usually not longer than 7 days
Tao, Woo & Loh (1948)	218	90% negative on or before 6th day; none positive beyond 9 days
El-Ramli (1948)	689	Negative by 15th day: 86.5% Negative by 20th day: 93.6%
Kamal, Messih & Kolta (1948)	1971	Positive not longer than 7 days: 83.5%
Kordi (1948)	250	111 (44.4%) of these carriers were free from vibrios by the 7th day, 208 by the 14th day
Shousha (1948)	463	Positive up to 10 days: 56.16% Positive up to 20 days: 29.80%
Hussein (1949)	250	Negative in 15 days: 86.0% Negative in 20 days: 99.6%
Cossery, Ashouk & Hilmi (1949)	60	Majority negative in about a week's time, others within 4 weeks
Gohar et al. (1952)	78	96% became vibrio-free within at most 12 days, 4% within 20 days
Gilmour (1952)	113	71.6% were negative after the 1st week, 89.3% after 2 weeks and 98.1% after 3 weeks

(c) Maximal periods of vibrio excretion in healthy ("contact") carriers

Author	*Maximal period of vibrio excretion (days)*
Wahid (1948)	1
Read & Pandit (1941)	9
Gohar & Makkawi (1948)	10
Kordi (1948)	14
El-Ramli (1948)	15
Hussein (1949)	15
Omar (1947) (Quoted by Khalil, 1948a)	16
Shousha (1948)	19
Kamal, Messih & Kolta (1948)	26

(d) Incidence and average duration of vibrio excretion in contact carriers

Author	*Number of persons examined*	*Percentage of carriers*	*Observations*
Smith (1938)	10 407	2.84	Ships' passengers arriving from the mainland in the Philippines
Read & Pandit (1941)	—	7.0	75% free after 5 days
King Institute (1941)	61	6.56	Examined during a cholera outbreak
El-Ramli (1948)	2 035	4.1	Free after 5 days: 50% Free after 10 days: 91.7%
Kamal, Messih & Kolta (1948)	14 473	3.43	Intimate contacts of cholera patients; usually became free from vibrios within 10 days
	2 411	1.9	" Stampeders " from cholera foci and boatmen examined at quarantine stations; 13 of the 47 individuals harbouring *V. cholerae* were afterwards found to be incubatory carriers
Kordi (1948)	2 037	4.1	Free after 5 days: 50% Free after 10 days: 92.9%
Shousha (1948)	13 702	2.1	Free after 5 days 65.96% Free after 10 days: 93.62%
Wahid (1948)	600	2.66	Free after 2 days: 75% Free after 7 days: 100%
Hussein (1949)	2 027	4.14	Free after 5 days: 50% Free after 10 days: 92.7%
Venkatraman (1949)	245	2.04	These carriers were found among the staff members of a cholera hospital. In each of the five carriers *V. cholerae* could be isolated but once
Gohar et al. (1952)	1 745	3.56	Average duration of the carrier state was 4.4 days

From these data it emerges that the average duration of the carrier state has been found to be appreciably shorter in healthy or, as many modern authors prefer to call them, contact carriers of *V. cholerae* than in convalescent carriers. While in at least 50% of the former the stools were found positive for not longer than five days and most of the individuals were free from vibrios after 10 days, some observers continued to obtain positive results in a considerable minority of convalescent carriers during the second week following onset of the illness. The maximal periods of excretion in contact carriers were definitely shorter than those found in the case of convalescents.

Confirming findings made in the past, some modern workers drew attention to the frequency of the carrier state in infants or young children. Abdou (1948), commenting upon these observations, postulated that the frequency of gastric achylia or of hypochlorhydria in children might be the cause of the high contact carrier rate in this age-group. Be this as it may, it deserves great attention that, as shown by Gohar and colleagues (1952), achlorhydria or hypochlorhydria may be significantly frequent in adult healthy cholera carriers.

Infectivity of cholera carriers

Though some authors postulated that cholera carriers, even those of long standing, were apt to play a dangerous role in the spread of the infection, many observers militated for various reasons against this view. Some early workers, like Jatta (1912) and Piras (1913), stated in this connexion that the likelihood of a spread of the disease by healthy carriers was limited both because their dejecta contained fewer of the causative organisms than the stools of patients and because the stools of the carriers were solid instead of being diarrhoeic and, therefore, much less easily diffusible. The markedly intermittent excretion of the causative organisms by cholera carriers no doubt also reduces the risks of spread of the infection.

Attempts have been made by a few workers to test the virulence of the cholera vibrios excreted by carriers with the aid of guinea-pig experiments. While Pane (1912) and apparently also Babes (1914) found no appreciable difference between the strains of cholera patients and carriers tested in this manner, van Loghem (1911) recorded that the *V. cholerae* cultures he had isolated from two healthy carriers proved to possess little virulence. Van Loghem pointed out, however, that these observations on guinea-pigs did not necessarily imply that the organisms in question were also innocuous for man.

A large-scale attempt to test the virulence of cholera strains obtained from both patients and healthy carriers with the aid of guinea-pig experiments (intraperitoneal inoculation of standard doses of half a loop) has been made by Piras (1913). He found that on the average the strains isolated from carriers possessed much less virulence than the cultures from patients. Whenever the growths initially isolated from carriers were virulent for the test animals, subsequently made cultures proved less virulent and, very often, finally quite avirulent. In Piras's opinion this diminution and eventual loss of virulence of the cholera strains isolated from healthy carriers accounted partly for the comparative harmlessness of the latter. It is of interest to add that tests with cultures successively isolated from cholera convalescents also indicated a gradual loss of virulence, part of the strains finally becoming avirulent.

Discussing the problem of a lessened virulence or avirulence of the cholera vibrios excreted by carriers, Pollitzer (1952) pointed to laboratory observations by Bruce White, the results of which were thus summarized in

the report on a meeting held in 1948 under the joint auspices of the Office international d'Hygiène publique and the World Health Organization:

" At the end of the disease and during convalescence an increasing proportion of the vibrios excreted by the patient are in the process of ' roughening ' or are entirely rough. Transformation from the smooth to the rough state corresponds to a loss of pathogenicity of the organism."

As stated by Kamal (1951), these postulations were supported by findings made by Rainsford at the end of the 1947 epidemic in Egypt, according to which cholera convalescents " excrete the ' R ' forms and none but the ' R ' forms ". Kamal felt convinced that the dissociated cholera vibrios were really non-infective because, even though it was necessary to discharge the convalescents not later than 15 days after onset of the disease—i.e., at a time when they still might excrete the organisms—no infections (" return cases ") resulted in their households.

Since, however, the observations of Gilmour (published in 1952) were not in agreement with these findings, the experts taking part in the meetings of the WHO Expert Committee on Cholera (first report, 1952) came to the conclusion that

" so far no conclusive evidence was available as to whether or not and to what extent the vibrios excreted by convalescent and contact carriers tend to be rough and to have an altered virulence ",

and added that

" It would be highly desirable to elucidate this point through systematic studies, advantage being taken of the recently recommended serological tests for the recognition of the R-type of *V. cholerae*."

It follows that up to the present the problem of the role of carriers in the spread of cholera has to be elucidated with the aid of epidemiological rather than of laboratory observations.

Epidemiological observations on cholera carriers

In contrast to the now generally accepted views, some of the earlier observers believed that, as with other gastro-intestinal affections, like typhoid and dysentery, in the case of cholera also carriers played an important role in the spread of the infection. However, the never convincingly documented claims made in this respect by Greig (1913a, 1913b) and a few other writers were refuted in an exhaustive report on the role of carriers in cholera by Khan (1929) and in a series of further large-scale studies on this problem carried out under the auspices of the Office international d'Hygiène publique and published in 1933.

The conclusion which Khan (1929) reached after a careful consideration of the experiences of previous workers and of observations on cholera convalescent and contacts he had made at Hardwar was that

" The reservoir of cholera is not the ' chronic carriers ' of *V. cholerae* because they do not exist; it is also not in the ' carriers ' of the ' inagglutinable ' vibrio because they

do not cause epidemic cholera. The real reservoir is in the presence in the endemic areas of patients suffering or recovering from cholera. The only source of the infection of epidemic cholera are patients suffering from the disease, in the acute stage for about 4 days; also some though to a much lesser extent in the convalescing stage for about 14 days; and perhaps a few in the incubation period for a few days."

The large-scale inquiries under the auspices of the Office international d'Hygiène publique (see Couvy, 1933; Stewart, 1933) led to similar conclusions, according to Taylor (1941) the evidence suggesting on the whole

" that with a very short persistence of *V. cholerae* in the intestinal tract of the convalescent or contact carrier, it was unlikely that the carrier was responsible for transmitting infection at any prolonged interval after the primary infection and consequently to places remote from cholera infected areas."

The results recorded above, obtained before fully specific O sera had become available for the laboratory diagnosis of cholera, were confirmed by further observations made with the aid of such sera by Read & Pandit (1941) who, as Taylor summarized, came to the conclusion that

" the detection of a carrier before the onset of a case in the vicinity was not accomplished and positive evidence was not obtained incriminating a carrier as the source of infection."

Seal (1945), discussing the problem of cholera endemicity in Bengal, came to identical conclusions. He noted that cholera vibrios could not be isolated from the stools of the general population or from water in the endemic areas except in direct relation to cholera patients, and considered contact carriers and water to be infective agents " for short periods and a short range " only.

The question whether or not and to what extent carriers play a role in the spread of cholera was once more the subject of considerable debate at the 1951 session of the WHO Expert Committee on Cholera (World Health Organization, 1952) and the meetings held in connexion with this.

Those experts with long experience in India or countries farther east maintained that cholera carriers were of no epidemiological importance. Attention was drawn in this connexion to the important observations recorded by Nicholls (1953), under the title " Carriers of *V. cholerae* who enter Ceylon from South India ".

As summarized by Pollitzer (1952), Nicholls calculated that during the period 1924-33 at least 200 cholera carriers must have arrived in Ceylon during a year of average immigration. It was known, on the other hand, that during the same period there were only ten occurrences of cholera in the areas to which the majority of carriers went. Nine of these cholera manifestations were due to the arrival of *incubatory* carriers; the origin of the tenth outbreak could not be elucidated. Nicholls concluded, therefore, that the great majority of the carriers must have been excreting avirulent vibrios.

It was also emphasized that the observations made in West Pakistan since the partition of India fully supported the experiences in Ceylon. Though hosts of immigrants or other travellers, using all available means of transport, arrived in West Pakistan from all parts of India without being subjected to quarantine measures no importation of cholera took place.

Claims were made by one member of the expert committee that cholera carriers, particularly contact carriers, had played a role in the 1947 Egyptian outbreak. Dealing with this contention the other experts stressed that

" The observations made in Ceylon, China, India, and Indochina during many years, on the other hand, do not point to contact carriers as playing a significant role. The trend of opinion in the committee was to the effect that contact carriers do not play a significant role in the spread of the infection."

It is important to add that, as admitted by Kamal (1951), cholera convalescents, even though they were usually discharged 12-14 days after onset of the disease, i.e., at a time when their stools were not necessarily free from *V. cholerae*, played no role in the spread of the 1947 Egyptian outbreak. Therefore, as concluded by Pollitzer (1952) there seems no reason

" to revise the opinion, held by most experts with experience in areas where cholera is endemic or frequent, that only ' acute ' carriers, that is, individuals late in the incubation stage, those actually ill, and possibly also those in early convalescence, are instrumental in spreading the infection."

Sex and age incidence

The task of evaluating the statements made by numerous writers regarding the comparative frequency with which cholera occurs in the two sexes and in the various age-groups is rather difficult in that (*a*) the size of the samples on which the various authors base their conclusions varies greatly, thus rendering their statistical significance different; and (*b*) worse still, most observers merely quoted statistics regarding the sex and age incidence in the cholera outbreaks observed by them without correlating these figures with those showing the comparative frequency of members of the two sexes and of the different age-groups in the general population.

A closer study shows, however, that these objections to the statistics available in regard to the sex and age incidence of cholera are of theoretical rather than practical importance, because the most marked variance of the data recorded in these respects by the different observers speaks strongly in favour of the assumption that extrinsic factors rather than intrinsic causes are at work to lead in some epidemics to a markedly more frequent incidence of the disease in males or in children, while other outbreaks are characterized by diametrically opposite features. While thus, in view of the markedly different extrinsic conditions under which the various cholera outbreaks evolve, no general rules can be laid down, it seems legitimate to state that

(*a*) in a majority of the cholera outbreaks both sexes are affected in a comparable manner, and (*b*) the opinion held by some workers that the infection is particularly rampant among young children has not been confirmed by many other observations. Particularly noteworthy in this respect is the following statement by Kamal (1951):

" I have studied the morbidity amongst the age groups 0-1 and 1-5 during the 1947 epidemic. While infants and children comprise 13.24% of the total population at risk, yet the number of cases that happened amongst them amounted only to 4.57% and while the general morbidity rate per 100 000 was 103, the same rate for the age group 0-5 was 34 only, and for those above five was 113.

" These figures show clearly that although children stand the same risk of infection, yet the disease incidence amongst them is low."

Similar views were expressed by several other authors and it is usually held that cholera is as a rule most rampant among adults within the age limits of about 20 years to 40 or 50 years. Commenting upon observations made in this respect in 1848-49 at London, Snow (1855) stated that

" The greater part of the female population remain almost constantly at home, and take their meals at home, whilst a considerable number of the men move about in following their occupations, and take both food and drink at a variety of places; consequently in the early part of an epidemic, when the disease only exists in a few spots, the male part of the population is most liable to come within the operation of the morbid poison; but at a later period of the epidemic, when the cholera is more generally diffused, it may reach those who stay at home as readily as those who move about; and in addition to the risk which the women share with the men, they have the additional one of being engaged in attending on the sick."

Racial incidence

As manifested by its history, cholera has been able to cause outbreaks of identically wide spread and equally great severity among practically all races inhabiting both the old and the new world. That no racial insusceptibility to this infection exists is likewise manifested by the severe toll formerly exacted by cholera among the British, especially among British troops, stationed in India. It is true that nowadays adequate standards of living and due attention to the precepts of hygiene are apt to go a long way in keeping foreigners resident in the eastern cholera areas free from the infection. However, a similar freedom from the disease may likewise be enjoyed by the strata of the local populations with standards of living and hygienic habits comparable to those in the western world. Thus, as has been discussed already, cholera has become mainly a disease affecting the underprivileged classes of the population, who, alas, are not rarely even unable to pay for the pure water supplied by the waterworks and have thus to rely on unsuitable and easily contaminable other sources of water supply.

Occupational incidence

As with the freedom from the disease at present enjoyed by the well-situated and reasonably careful classes of the population, so instances of infection among the staff of adequately managed cholera hospitals are rare exceptions nowadays, if they occur at all. Such an absence of infection among hospital staffs was frequently observed even long before the discovery of *V. cholerae*, but, as summarized by Griesinger (1857), in other early cholera outbreaks there was a high incidence of such infections. In Griesinger's opinion the reason for the difference was that the standards of cleanliness and sanitation adopted in the various hospitals varied considerably, and particularly that

" rapid removal and disinfection of the evacuations are sometimes resorted to and sometimes omitted; that the medical personnel is sometimes urged to watch over their health and to submit to rapid treatment of any diarrhoea and at other times neglects such precautions; that old and overworked individuals leading an immoderate life sometimes function as nurses; that, briefly, in some instances various auxiliary factors either exert an untoward influence or grant protection." [Trans.]

Similarly, Flügge (1893) pointed out that on the one hand untrained nurses were apt to fall a prey to the infection, while on the other hand

" Physicians, the trained nursing staff of hospitals, persons educated to cleanliness, who do not touch their mouth or their food with unclean fingers and do not keep their food in the sickroom, are not exposed to the infection." [Trans.]

Factors Governing the Spread of Cholera over a Distance

General considerations

While many authorities are in agreement with the dictum of Greig (1929) that " the spread of cholera is effected by man himself ", some observers have maintained that long-distance transport of the infection may be effected also by other means, among which the following deserve mention.

Infected water courses

It has been claimed or at least implied by a few writers that rivers or other water courses, if they become contaminated with *V. cholerae*, may serve as a transport vehicle for the organisms. This was upheld by Strong (1944), for instance, who stated that

" In countries which lie adjacent to endemic centres of infection, the disease . . . may spread considerable distances by an infected water supply. Thus in India the infection has been carried by the River Cauvery for approximately 18 miles [30 km] to the Madras Presidency. The infection was also said to be carried by Lake Five (the source of the water supply) which became infected through water pipes, for a distance of at least 11 miles [18 km]. Also in Mesopotamia cholera infection has apparently travelled long distances down the Tigris River."

Mathew (1949), dealing with the epidemiology of cholera in Madras Province (now Madras State), pointed to an ominous role played in the irrigated part of this area by the smaller irrigation channels. These, he explained,

" pass through every village in the area. Almost each house has direct access to the channel. The channel water is grossly contaminated by personal ablution, washing of clothes and vessels, washing of animals, etc. Even the clothing and bedding soiled with cholera excreta are washed in them and yet the people drink this water as such as they find it more tasty than the well water. It is a common experience that once any of these villages is infected with cholera, all villages situated lower down on the same canal or channel will be affected one after the other in quick succession. The rapid dissemination of cholera infection in canal-irrigated areas and its greater prevalence there are the natural consequence."

As stated by Benjamin (1949), a role similar to that of the Madras irrigation channels was played in the spread of cholera by the large number of rivers and their tributaries intersecting the districts on the Deccan Plateau of Bombay State. There also it was

" observed repeatedly that once a town or village on the bank of a river or stream is infected, other villages downstream of the river usually get infected rapidly; the distance to which infection spreads varies (*a*) with the size of the village or town first infected and (*b*) the duration of the infection in that village."

As Benjamin added, widespread outbreaks of " riverine " cholera produced in this manner were characterized by a distribution of the infection

" in the form of a band with the river in the centre of the band and an almost equal area consisting of villages on either side of the river but at a distance from it; the latter being infected by infection due to communication with the infected riverside villages. In non-riverine areas, the infection spreads radially round the initial infection, the radius varying with the size of the original town or village infected, its importance as a trade centre and the facilities for communication."

Interesting as these observations are, the opinion that contamination of water courses with *V. cholerae* plays an important *direct* role in the long-distance spread of the infection has not been universally accepted. Robertson & Pollitzer (1939) in particular, who had good opportunities for similar observations in China, maintained in this connexion:

" We have no evidence suggesting that the causative organism has been carried over any appreciable distance by the waterways themselves. In fact, infection, though often travelling along them, has frequently spread upstream rather than downstream. Certainly, however, water borne traffic was one of the principal means of broadcasting cholera. On the other hand one must not overlook the fact that motor roads and other routes of land traffic generally run parallel with the waterways, the principal settlements lying usually on both. It was thus sometimes difficult to decide by what particular route infection had reached a given settlement."

Notwithstanding these objections, one should not categorically rule out the possibility that contamination of water courses with *V. cholerae* may be responsible for a spread of the infection to riverine settlements lying a short distance downstream from the originally affected localities. At the same time, however, persons incubating cholera or developing the disease in transit are apt to carry the infection to more distant important towns.

Contaminated inanimate objects

That " fomites " like the body- and bed-linen soiled by patients, which may be of some importance in the local spread of cholera, also play a role in the long-distance propagation of the infection seems altogether unlikely. However, certain foodstuffs have been incriminated in this respect. Thus Heiser (1908), dealing with the cholera problem in the Philippines, related that

" The commencement of the disease in 1902 was ascribed to a shipment of presumably infected Canton cabbage arriving in Manila from Hongkong which, upon being refused landing, was thrown overboard in the harbor, with the result that the whole bay was literally covered with this vegetable. Much of the cabbage was gathered and consumed and within forty-eight hours from the time of its reaching the beach, cases of cholera commenced to appear."

Even if one accepts this story at face value, there can be no doubt that a long-distance spread of cholera by contaminated vegetables is exceptional. However, it deserves serious attention that, as has already been discussed earlier in this study (see page 823 et seq.), some convincing evidence exists to show that the transport of cholera-contaminated fish to distant markets may be a means of importing the infection. That shellfish may play a similar role is exemplified by observations of Ronchetti (1911-12) and Kundu & How (1938). The former worker found the fatal infection of a cholera patient in Milan to be due to the consumption of oysters which had been imported from Taranto, while Kundu & How succeeded in isolating cholera vibrios from two prawns brought from the delta region of Burma to the Rangoon market.

As discussed by Kamal (1951), during the 1947 outbreak in Egypt a controversy took place regarding the long-distance spread of the infection through date-pulp cakes exported from the cholera-affected areas. The bacteriologists asserted that *V. cholerae* remained viable in dates for not more than three to five days, but local health officers reported that

" new foci in certain regions began with the importation of dates from infected areas, especially Korein and neighbouring localities."

As described by Kamal, the date-pulp cakes were certainly prepared in a most unhygienic manner likely to facilitate greatly their contamination

with *V. cholerae*. Nevertheless one must wonder whether they or the persons who brought them were responsible for the importation of the infection into hitherto cholera-free localities. The results of the bacteriologists speak certainly in favour of an introduction of the infection by human agency.

Passive transport of flies

As claimed by a few observers, for instance by Bernard (1936), the long-distance spread of cholera may also be effected by the passive transport of contaminated flies, carried, for instance, by railway trains. Certainly, however, such a transfer of the infection, if it takes place at all, must be rather rare as compared with the usual mode of cholera propagation through persons who leave affected localities when already ill or when incubating the disease to fall a prey to it either in transit or after they have arrived at their destination. The factors responsible for the speed and the intensity with which such a long-distance spread of cholera through human agency takes place will now be given attention.

Spread by infected individuals using various methods of transport

In the course of an interesting discussion of cholera epidemiology, Hart and his colleagues (1910) pointed out that man

"can but carry the disease so far as he is able to travel between receiving the infection and being laid low. What we find then, on comparing the march of the earlier epidemics of cholera with those that have occurred in more recent years, is that whereas when travel was slow the disease swept steadily forward, occupying the land as it advanced; in later times it has bounded forward with long strides, occupying outposts far ahead of infected areas by means of railway and steamboat communication, and then from these outlying foci of infection, has spread in both directions, coalescing perhaps at a much later date with the main body of the epidemic which has slowly advanced across country from the earlier centres."

While this statement is of great value in so far as it does justice to the ability of cholera to make long-distance sprints if modern means of communication are available, it might give the wrong impression that the slow contiguous spread of the infection is a quite uniform process. Actually, as will be discussed below, in this mode of spread as well initial foci may be created in places particularly suitable for inroads of the infection and from these centres the disease may be carried to the surrounding localities. One might thus say that a spread of the infection by shorter or longer relay stages is typical of the propagation of cholera.

Though, as pointed out by some authors, first perhaps by Greig (1919), a rapid spread of cholera might be effected by air traffic, as far as the present writer is aware this fear has not been substantiated thus far. It is permissible, therefore, to focus attention upon other means of communication.

(1) Caravans

Dealing with the problem of the spread of cholera by caravans, Dugue (1931) stated that

" The sterilizing effect of caravans has always been stressed. Indeed, upon seven occasions, the caravan of Syria, starting from Medina, left with cholera and the malady became extinct en route without ever being imported into Damascus." [Trans.]

However, in a subsequent publication, Duguet (1932), while admitting that as a rule cholera persisted in the Medina caravans only for 10-15 days (corresponding to a trip of 300-400 km), the infection could be carried over wider distances by caravans passing through well populated areas.

(2) Railways

The great importance of railways for the spread of cholera was well illustrated by observations recorded at the 1885 cholera conference by Koch, according to which

" The Punjab belonged to those parts of India which formerly suffered least from cholera. From the year 1820, in which the first reliably authenticated cholera epidemic came into the Punjab, up to the sixties, i.e., in a period of about 40 years, the province had only 5 epidemics: 1820, 1827, 1845, 1852 and 1855. Then railway traffic was opened. From then onwards a comparatively large number of epidemics followed regularly: 1861, 1862, 1865, 1867, 1869, 1872, 1875, 1879, 1881. Thus suddenly from 1861 onwards the behaviour of cholera in the Punjab became changed. The population remained the same, and the meteorological conditions did not change; only the traffic with the cholera focus in Bengal was accelerated." [Trans.]

Agreement with the opinion of Koch that railway traffic played a most important role in the long-distance spread of cholera was expressed by several other early observers, some of whom pointed in this respect particularly to the causation of a quite considerable epidemic at Altenburg in Saxony in 1865 through the arrival of a woman with a cholera-affected child from Odessa in Russia.[1]

It is reassuring to note that some of the modern observers are inclined to lay less stress on the role played by railway traffic in the spread of cholera. Thus Napier (1946), dealing with the conditions met with in India, maintained

" that normal railway travel on business or pleasure does not tend to spread the disease to any great extent on account of the control that can be exercised over passengers, and that, though the sanitary arrangements are far from perfect, especially at the small stations, there are latrines and a safe water supply. Such travellers of course come from all grades of society, but even the poorest are seldom destitute, and the fact that they are travelling usually indicates that they can afford the ordinary necessities of life."

[1] See *Bull. Wld Hlth Org.*, 1954, 10, 443.

(3) Ships

While it was always agreed that the local traffic by small craft played an important role in the propagation of cholera, the question to what extent ships undertaking long voyages were of importance for the spread of the infection was in the past the subject of much debate. The anti-contagionists tried to prove that cholera manifestations on sea-going ships were rare and, therefore, of no epidemiological significance. However, as pointed out by Koch (1885), it was wrong in this respect to take into account all cholera-infected ships, including those leaving ports which were only occasionally affected by the disease, instead of focussing attention upon ports like that of Calcutta where the infection was constantly present. More important still, as stated with great reason by Flügge (1893),

" on well-found ships there were not so many opportunities for the direct transmission of the infection as in the habitations of the less affluent classes of the population. Strict supervision and obligatory cleanliness led on board such ships to a much more thorough abolition of most sources of the infection than was the case in small urban habitations. Secondly, the drinking water taken for the voyage was either free from comma bacilli from the first or the organisms perished within a few days and there was no occasion for further contamination. Finally, it is far more easy to observe the initial cases and to pay early attention to them, than is possible in urban settlements." [Trans.]

However, Flügge emphasized,

" if cholera cases occur in the overcrowded space between decks, kept without adequate control in an unclean state, direct transmission is apt to take place repeatedly, especially among groups relying on common food and living closely together. Moreover, even on well-kept ships protracted epidemics may be caused by accidents, such as incautious treatment of the patients' linen or contamination of certain foodstuffs with comma bacilli." [Trans.]

Under these circumstances it is not surprising to find that a by no means inconsiderable number of sometimes quite intensive and protracted cholera outbreaks has been recorded on sea-going ships. As summarized by Flügge,

" Most long lasting were the epidemics on the *Apollo*, which in 1849 had 18 cholera deaths among 593 persons, with the last case on the 56th day after departure; on the *Franklin*, on which out of 611 between-decks passengers more than 200 showed choleraic symptoms and 43 (i.e., 7%) died, and on which the last case occurred on the 33rd day after departure; and thirdly on the *Matteo Bruzzo*, which left Genoa for Montevideo in 1884 and had, among 1333 persons, 40 cholera cases (with 20 deaths), the last on the 52nd day after departure. Severe epidemics also occurred on the *Leibnitz*, which went from Hamburg to New York in 1867 and had 165 cholera attacks with 105 deaths; and on the steamer *England*, which left Liverpool for New York in 1866 and had until her arrival at Halifax 150 cholera attacks and 46 deaths; 200 further cases were recorded during quarantine at Halifax." [Trans.]

While under these circumstances sea-going ships in the past played an ominous role in the transport of cholera infection, it is consoling to note that, as will be discussed in the following of these studies, this danger was

afterwards practically abolished through adequate measures of sanitation on board the vessels [1] in combination with implementation of quarantine measures ashore.

Group movements and assemblies of the population

General considerations

Ample experiences have shown that (*a*) the arrival of groups of people who have come from, or passed through, cholera-infected localities is apt to lead to outbreaks of the disease at the places of their destination; and (*b*) if the infection is already established in the latter, its spread may receive impetus through the influx of groups of travellers both because, having undergone vicissitudes during their journey, they may fall an easy prey to the disease and because the newly arrived persons are apt to crowd together in unsanitarily kept quarters and, generally speaking, to lead an unhygienic life.

The purpose, and consequently the scope and epidemiological importance, of such movements of the people may vary considerably. Relatives and friends may assemble to celebrate marriage feasts or—what is far more dangerous—to attend the funeral of cholera victims. Groups of people may proceed at regular intervals from surrounding districts to communities where markets are held, the latter thus often becoming the distributing centres of cholera epidemics. As discussed above, in the past insanitarily kept ships on which emigrants or seasonal labourers were herded together served as a means of carrying the infection to distant parts, for instance, from European to American ports. While, as has been noted, this danger has been practically abolished, migrations of seasonal labourers on land routes still continue to be responsible for the dissemination of cholera. Thus Mathew (1949), aptly discussing the observations made in this respect in Madras State, recorded that

> " Labour families leave their villages in quest of work and proceed to other localities either in the same district or in the adjoining districts according to the working season. There is absolutely no control over their movements and they live in the fields or by the roadside. They drink any water which is readily available near the workspots and eat any food that they get. Cholera outbreaks are common amongst them and when infected they take fright and disperse in various directions leaving behind the dead and the suffering. Groundnut picking in the central districts and transplantation and harvesting of paddy in the river irrigated areas attract considerable labour from less fertile regions."

However, even the role played in the spread of cholera by the movements of seasonal labourers is overshadowed by far by the influence exerted on

[1] As reported by Koch (1885), the introduction of sanitary improvements, particularly the provision of pure waterworks water in place of Hooghly river water in 1874 led to a marked decrease of the cholera incidence on ships leaving Calcutta with coolies (i.e., seasonal labourers) for Assam and other ports.

the spread of the infection by wars, usually involving the movements not only of large bodies of troops but also of hosts of refugees, and by pilgrimages. While sufficient evidence of the epidemiological importance of wars has already been furnished in the first of these studies, the following additional comments on the role of pilgrimages have to be made.

Pilgrimages

(1) Mecca pilgrimages. Apart from pilgrimages in various parts of India, to be dealt with soon, a most ominous role has been played in the long-distance spread of cholera by the perennial pilgrimages to Mecca where, as summarized in the first of these studies,[1] a violent epidemic broke out first in 1831 and was evidently responsible for importation of the infection into Syria, Palestine and Egypt. As can be gathered from the literature summarized in the study just mentioned, outbreaks continued to occur among the pilgrims at Mecca at frequent intervals—according to Duguet (1931), thus:

Period	*Number of epidemics*
1855-1866	9
1881-1883	3
1890-1893	4
1907-1912	4

Due invariably to the early arrival of some pilgrims from the eastern cholera foci (mostly by the sea-route), the Mecca epidemics gained impetus when large numbers of pilgrims had crowded together during the festivals, reaching their maximum, Duguet maintained, either in spring and early summer (May-July) or in autumn and winter (October-January). They continued to be a serious menace for the countries to the west until the progress of the disease was effectively barred through the establishment of a quarantine camp at El Tor.

(2) Pilgrimages and religious festivals in India. As aptly stated by Banerjea (1951),

"The association of cholera with pilgrimages, fairs and festivals is well-known. These have acquired notoriety and are considered by health authorities as starting points of wide-spread epidemics. If one of these fairs of some respectable size happens to take place in the cholera season, then, but for the most stringent sanitary precautions, a severe outbreak of cholera is certain and is spread widely . . . by the dispersing crowds. Smaller fairs and festivals can also be dangerous because of the less satisfactory arrangements and control. As pointed out by Lal (1937), not only is the place of congregation the danger spot, but the nodal points along the pilgrim routes are sources of equal anxiety to the health administration."

According to the statements of authors like Lal (1937) and Banerjea (1951), the festival centres in India fall into two classes—namely, (1) places

[1] See *Bull. Wld Hlth Org.*, 1954, **10**, 435.

of perennial pilgrimage, and (2) temporary camps specially erected for periodical fairs and festivals.

As explained by Lal,

" The former possess some special religious sanctity apart from the occurrence of holy days. They continually attract pious people from different places. Kalighat, Benares, Puri, Rameswaram and many others may be cited as examples of such centres. The latter type of centre comes into prominence only on certain days in the year and, more particularly, periodically after a number of years. The attractions of these places are as much secular as religious. Kumbhs and Adha-Kumbhs at Hardwar and Allahabad are the best examples."

Pilgrimages of a peculiar kind, possessing an epidemiological importance of their own and therefore requiring special methods for their control, were the so-called " moving religious festivals " or " palkies ", a term derived, according to Rao (1947), from the word " palanquin ", because these

" were in this particular case used or intended for carrying the wooden sandals of some well-known saint to a central place of pilgrimage . . . These *palkies* start from a place which is either the birthplace of a saint or where his ashes lie buried. They sometimes travel a distance of more than three or four hundred miles . . . "

As pointed out by Rao, when dealing with the special case of the " palki " proceeding to Pandharpur (in Bombay State), such processions formed not only a safe but also a cheap means of making a pilgrimage, especially since the participants used to be fed en route by the devotees of the saint in whose honour the pilgrimage was made. Therefore they continued to be popular even though much more rapid means of transport had become available.

Among the numerous important pilgrim festivals and fairs held—mostly every year—in various parts of India (see list by Lal, 1937), the following deserve special mention.

(*a*) As summarized by Banerjea (1951), about 400 fairs, attracting a total of over 12 million people, were held annually in the *United Provinces* (now *Uttar Pradesh*). Of these, Banerjea continued,

" 116 fairs with a total gathering of 287,000 are held mostly in eastern districts in the months of March and April (when meteorological conditions become favourable for the spread of the disease). Besides these, Kumbh and Ardh-Kumbh fairs at Hardwar and Allahabad, the two largest fairs in India, alternate every sixth year at each place. A large pilgrimage, therefore, occurs at one or the other every fourth year. Kumbh and Ardh-Kumbh fairs attract a gathering of nearly three and two million respectively at Allahabad and about one million and half million respectively at Hardwar."

The dismal influence which these large gatherings exerted on the incidence of cholera in the United Provinces is well illustrated by the following data furnished by Banerjea, showing the effect of Kumbh (K) and Ardh-Kumbh (A) fairs on cholera mortality:

Hardwar			Allahabad		
Year	*Category*	*Number of deaths*	*Year*	*Category*	*Number of deaths*
1879	K	35 892	1882	K	89 372
1885	A	63 457	1888	A	18 704
1891	K	169 013	1894	K	178 079
1897	A	44 208	1900	A	84 960
1903	K	47 159	1906	K	149 549
1909	A	21 823	1912	A	18 894
1915	K	90 508	1918	K	119 746
1921	A	149 667	1924	A	67 000
1927	K	28 285	1930	K	61 334
1933	A	1 915	1936	A	6 793
1938	K	70 622	1942	K	7 662
1945	A	77 345*	1948	A	52 604

* This epidemic was said not to have been connected with the Ardh-Kumbh.

As Banerjea added, the average death-rate from cholera in the years during which no Kumbhs or Ardh-Kumbhs were held was 1.6 times lower than that during the festival years.

As proved by statistics of Rogers (1928) and by other observations, the prevalence of cholera in the United Provinces during the Kumbh and Ardh-Kumbh years inevitably led to an exacerbation of the cholera incidence in India in general. Particularly important was that, owing to its geographical position in the north-west of the United Provinces, a high incidence of the disease in Hardwar almost invariably led to a serious cholera situation in the Punjab and that the prevalence of the infection in the latter area was apt to be responsible for a further westward progress of the scourge resulting in its pandemic spread. Thus, as noted in the first of these studies,[1] the Hardwar pilgrimage of 1826 and the subsequent invasion of the Punjab were responsible for the second cholera pandemic gaining impetus in 1829. Banerjea (1951) asserted on the authority of Wu Lien-teh (1934) that the pandemics of 1866-70 and of 1892-95 respectively likewise

" spread from Hardwar pilgrims to the northwesterly province of Punjab in India and by the overland route spread to Afghanistan, Persia, Southern Russia and finally invaded both Europe and America."

(*b*) Duggal (1949), discussing the cholera situation in *Bihar*, stated that, besides religious festivals and cattle fairs of solely local importance, some festivals of interprovincial importance were also held in Bihar, the pilgrim centres at Deoghar and Gaya, for instance, attracting visitors from all parts of India. Unfortunately most of the religious festivals in Bihar fell in the cholera season, thus being likely to serve as starting points or distributing centres of widespread epidemics.

(*c*) Dealing with the cholera situation in *Orissa*, situated south of Bihar, Hajra (1949) declared that

[1] See *Bull. Wld Hlth Org.*, 1954, 10, 432.

" The large number of perennial festival centres, for which the province is most popular, forms a strong foothold for cholera. As an instance I may cite the case of the car festival at Puri. This festival attracts the largest number of pilgrims varying from 1½ to two lakhs [i.e., 150 000-200 000] not only from the province but also from the whole of India . . . The festival occurs during June and July just as the south-west monsoon sets in and fly breeding is at its maximum. All these factors contribute to the introduction and spread of cholera infection in the province . . . This might as well have been responsible for some of the epidemics in other provinces in certain years."

(*d*) As described by Mathew (1949), *Madras State* had over a thousand festival centres. Though many of them were merely of local importance, nevertheless they could play a dangerous role if they became infected with cholera, because, as Mathew maintained with much reason,

" Although it is possible to make elaborate sanitary arrangements at the festival centre itself, the innumerable routes by which the pilgrims travel to the festival centre and back are a difficult problem especially in regard to water supply and conservancy."

Thus, Mathew found,

" It is a common experience to find that although the festival itself has passed off without any cholera outbreak, the returning pilgrims have conveyed the infection to several localities. This was the case with the cholera invasion of 1947. Not a single case of cholera was recorded at the Adi Ammavasi festival at Rameswaram which took place in July 1947 and yet several districts in the south were infected by returning pilgrims and a severe epidemic was started . . . The source of the infection was traced to a party of pilgrims from outside the province."

(*e*) Dealing with the cholera situation in *Bombay State*, Benjamin (1949) recorded that among the numerous places of pilgrimage in this area that at Pandharpur was the largest. Fairs were held there almost every quarter, but the two most important ones, which were visited by a large concourse of people from almost all the Marathi-speaking regions of Bombay State as well as the Central Provinces and Hyderabad State were the Ashadi and the Kartiki fairs, the former being held in June or July, the latter in November. As has been noted before (see page 848), a peculiar feature of the Ashadi fair was that it was the goal of some organized processions ("palkies") starting from localities outside as well as inside Bombay State and attended, at the time they reached Pandharpur, by up to twenty thousand people. It is obvious that these processions, which repeatedly had to camp en route, were in the past a dangerous means of spreading cholera. However, as will be discussed in the following of these studies, most gratifying progress has been made within recent years in coping with these situations and controlling the pilgrimages in general.

RÉSUMÉ

Dans une première section, l'auteur traite des régions où le choléra existe à l'état endémique et des conditions qui, normalement, favorisent l'endémicité. Passant aux épidémies, il discute leur origine et leurs types, les effets que le climat exerce sur elles, leur

périodicité, la possibilité de les prévoir, le rôle joué dans leur apparition par diverses races sérologiques de *V. cholerae*, les causes de leur déclin. Une section est consacrée aux facteurs qui conditionnent la propagation locale du choléra : l'auteur étudie la transmission par contact et la transmission par l'eau ; le rôle des aliments et des boissons contaminés, des contages, des mouches et des porteurs de germes ; la fréquence de l'infection suivant le sexe, l'âge, la race et le métier. Dans une dernière section, l'auteur passe en revue les facteurs qui déterminent la propagation du choléra à de grandes distances et il discute les conséquences des déplacements individuels ou collectifs, ainsi que des rassemblements de population à l'occasion de pèlerinages ou de fêtes religieuses.

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