TABLE II.

Showing the treatment of malaria cases and the by-effects noted.

Treatment.	Number of cases.	Cerebral symptoms. (Fullness in head, headache, dizziness.)	Deafness.	Diminished vision.	Gastric symptoms. (Vomiting, etc.)	Intestinal symptoms. (Diarrhœa, etc.)	Urticarial skin eruptions.	Epistaxis.	Hæmoptysis.	Cardiac and respiratory depression.
Quinine mixture. (Containing gr. v. quinine sulph. thrice a day.)	1,000	781	698	2	218	112	4	3	1	1
Cinchona mixture. (Containing gr. v. cinchona febrifuge thrice a day.)	1,000	972	799	10	694	763	1	2	..	6
Powders containing gr. v. cinchona febrifuge with gr. v. sodii bicarb. thrice a day.	1,000	866	693	..	310	684	1	..	..	3

September—and not October, as in 1927. The lowest point was in January.

The curve for 1929 is the most interesting of the three. From February to May there is an abrupt increase, followed by a drop in June. In September malaria cases constituted 62.5 per cent. of all cases seen, and even in December remained as high as 35.4 per cent. of the total attendance. There is no doubt that Lucknow and its environs are becoming more and more malarious, and epidemic conditions may become established unless proper measures are taken.

Table II shows the general symptoms noted and the methods of treatment adopted. There were a few additional cases whose temperature came down only after intravenous injections of quinine hydrochloride. In the early part of the epidemic in 1929 quinine mixture alone was used; later, when the supply of quinine ran short, cinchona febrifuge mixture was issued; this however was badly tolerated by the patients, and was positively harmful to many. Until supplies of quinine were renewed, therefore, I replaced the cinchona febrifuge mixture by powders containing equal amounts of cinchona febrifuge and sodium bicarbonate. This was not only better tolerated than the cinchona febrifuge mixture, but was especially appreciated by the poorer classes who had not enough money to buy the bottle of medicine which they required. A study of Table II will show the merits and demerits of the three methods of treatment adopted, but it is to be noted that the quinine mixture brought about a more rapid recovery than the cinchona mixture or the cinchona powders.

A characteristic feature of the 1929 outbreak was that villagers as a rule took longer to get rid of their malaria than the more educated patients from the city. This may be explained by two facts: (*i*) villagers come for medical aid only when they have been ill for some time, and (*ii*) they are naturally more exposed to re-infection from mosquito bites.

THE DIFFERENTIAL DIAGNOSIS OF CHOLERA AND FOOD POISONING.

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As fatal cases of cholera occurring amongst Europeans in India are often euphemistically attributed to "ptomaine poisoning," notwithstanding the fact that no bacteriologically attested outbreak of food infection, so far as the writer is aware, has ever been recorded in India, it will be profitable to consider the differential diagnosis of these two apparently similar conditions.

The term "ptomaine poisoning" is a misnomer and should therefore be discarded. The word *ptomaine* which is derived from the Greek word *ptoma*, a corpse, was originally coined in 1870 to describe the products of protein decomposition. Some of these products, though by no means all of them, when injected parenterally into laboratory animals were found to be poisonous. Subsequent research however showed that although this was the case, none of these products were actively poisonous when taken by the mouth, and that even those that were active did not produce intestinal colic, vomiting and diarrhœa—the common syndrome of food poisoning. The term "food poisoning" or preferably "food infection" should therefore be substituted for the popular and wholly inaccurate term "ptomaine poisoning," since it is not decomposed food but food infected with certain pathogenic organisms which gives rise to outbreaks of so-called food poisoning. Infected food does not differ from uninfected food in either appearance, taste or smell.

The organisms responsible for the outbreaks of food poisoning which have been bacteriologically verified in Europe, belong to the

Salmonella group of bacteria and include the *B. enteritidis* of Gærtner and *B. ærtrycke*.

B. ærtrycke is very closely related to *B. paratyphosus B* and can only be differentiated from it by the absorption test.

These organisms in cases of food poisoning are found chiefly in beef and pork, particularly in "made-up" dishes such as brawn, sausages and pies. They are also found in the milk of diseased cattle. Mutton and fish are seldom infected. Other foods such as fruit and vegetables become infected indirectly. Rats and mice are natural carriers of the Salmonella group of bacteria and food may be infected by their fæces.

Owing to the very short latent period in many outbreaks of food poisoning, it has been concluded that the causative organisms may form toxins in the infected food. Such toxins however have never been isolated. Filtrates of these organisms are toxic when injected parenterally into laboratory animals but cause no symptoms when given by the mouth. Infected food can be boiled for an hour or more without impairment of its toxicity. The exact way in which the Salmonella group of bacteria produce their irritant effects on the human intestine is at present unknown.

The differential diagnosis of cholera and food poisoning by bacteriological investigation being out of the question in most cases in India, we shall consider the clinical symptoms of these two conditions in detail.

In the great majority of instances, an attack of cholera begins as a painless diarrhœa, without nausea or physical discomfort of any kind, in fact one of the outstanding characteristics of cholera is the general absence of pain, with the exception of that associated in the later stage of some cases with muscular cramps. The textbooks fail to lay adequate stress on this very striking phenomenon. The stools which are at first fæcal, soon become watery and copious in character, white shreds of denuded intestinal epithelium being passed from the bowel along with the colourless fluid. The stools at this stage are described as "rice-watery" from their resemblance to water in which rice has been boiled—water in which rice has been boiled containing numerous white flakes of rice in suspension. After a certain interval corresponding with the onset of the watery evacuations from the bowel, vomiting supervenes. As soon as the stomach has emptied itself of its original contents, the vomiting becomes watery, copious and projectile, considerable quantities of fluid being frequently evacuated per os. The vomiting in cholera is unaccompanied by nausea, retching or distress. Intestinal pain and discomfort are usually absent, and where present are generally mild in character. Acute abdominal pain is extremely rare and is associated in a considerable percentage of cases with pink blood-stained stools, which, contrary to what might be anticipated, are of very favourable prognostic import. Tenesmus is absent, the stools as a rule being passed involuntarily. Owing to the dehydration of the body through the copious watery evacuations, muscular cramps, collapse and suppression of urine, all in time supervene. The voice becomes husky and whispering and the features shrivelled. The axillary temperature falls below normal. The breath is cold. The mortality in cholera is very high, being 90 per cent. in untreated cases.

The clinical picture in food poisoning is strikingly different. An attack of food poisoning generally begins with acute abdominal pain, accompanied by headache and a rise of temperature (99—102°F.). In severe cases a sensation of chilliness may also be present. After some time violent vomiting and retching set in, followed, or in some cases accompanied by, diarrhœa and tenesmus. This syndrome of symptoms in food poisoning—acute abdominal pain, violent vomiting and retching with diarrhœa and tenesmus—generally in the order named, is very constant and characteristic. The stools are usually offensive and though fluid, remain fæcal or bilious in character, never becoming colourless or "rice-watery." Prostration and muscular weakness are common, but there is no collapse from loss or fluid, though in severe cases, owing to toxæmia, there may be faintness or syncope with temporary disappearance of the pulse at the wrist.

Suppression of urine never occurs nor does the voice become whispering or the features shrunken. Muscular cramps of a mild nature are sometimes found in very severe cases together with tingling or numbness of the extremities. The mortality is low, ranging from one to two per cent. Death when it occurs is due to toxæmia and exhaustion from intractable retching, which is constantly present in fatal cases.

For convenience sake the differential diagnosis of these two conditions is given in tabular form below:—

Symptoms.	Cholera.	Food poisoning.
Diarrhœa ..	Painless. Precedes vomiting.	Associated with severe intestinal pain. Generally follows vomiting.
Vomiting ..	Causes no distress. Watery, copious and projectile. Follows diarrhœa.	Often violent and distressing. Vomit consists of food, and is never watery, copious or projectile. Generally precedes diarrhœa.
Nausea ..	Absent ..	Constant.
Retching ..	Rare ..	Constant, often severe.

Symptoms.	Cholera.	Food poisoning.
Acute abdominal pain.	Rare ..	Constant.
Tenesmus ..	Absent ..	Common.
Stools ..	"Rice-watery" and copious.	Liquid but fæcal and offensive. Never colourless or copious.
Urine ..	Complete suppression.	Never suppressed.
Muscular cramps.	Constant. The severity depending on the amount of fluid lost from the tissues.	Present only in very severe cases. Often associated with tingling and numbness. Mild and confined to the extremities.
Collapse ..	Frequent. Chiefly from loss of fluid.	Never from loss of fluid. In severe cases faintness or syncope may occur from toxæmia.
Fever ..	Surface temperature below normal.	Axillary temperature 99—102°F. Accompanied by shivering in severe cases.
Headache ..	Absent ..	Frequent.

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AN EXPERIMENTAL STUDY OF HOST SUSCEPTIBILITY TO CHOLERA.

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SEVERAL workers in this laboratory have been investigating the general problem of host susceptibility to disease. The body is enclosed in an epithelial covering layer. The anatomical structure of this layer varies a great deal. The skin, the upper and lower respiratory tract, the alimentary tube, and the urinary tract are continuous body-covering layers. The causative agent of disease must be able to maintain itself upon and in contact with a body surface-covering layer; penetrate or invade the host and then colonize or live as a parasite within the host. The resulting abnormal physiological and anatomical changes characterize the particular disease process. We have devoted most of our attention for a number of years to the beginning of this chain of events, namely, to the reason why foreign or exogenous bacteria are able to survive upon the body-covering layer of the host. Most investigators have devoted their attention to the actual disease process itself. Bacteria have been injected into animals in varying amounts and the results have been recorded. The anatomical changes have been studied even more than the physiological alterations. It occurred to us that this method of attack omitted the most important link in the epidemiological chain of evidence. When two or more people come in contact with a disease-producing microbe in the same concentration and one person becomes ill, we ordinarily explain this by assuming an increased susceptibility of that particular person. The literature is full of vague references to "decreased resistance," "lowered resistance," "increased susceptibility," etc.

Some of our work has been reviewed in relation to the problem mentioned above (Arnold, 1929). A note was published upon the possibility of certain aspects of this problem in relation to cholera (Arnold, 1927). The mucosa of the alimentary tract has the power of regulating the bacterial life that exists within the lumen and in contact with this body surface in the normal individual. Foreign bacteria injected are destroyed. The bacterial population inhabiting the incubated semi-fluid or solid contents of the digestive apparatus are under the control of the mucosa. Certain environmental changes will allow ingested bacteria to remain viable within the lumen of the intestinal tract for hours or days. The loss of the self-disinfecting power of the alimentary canal is closely associated with the entrance of bacteria into the body of the host. The gastro-intestinal tract is not the only body surface that possesses the power of destroying foreign strains of bacteria. Arnold, Ostrum and Singer (1928) have shown that the mucosa of the nose has the same self-disinfective power. Arnold, Gustafson, Hull, Montgomery and Singer (1930) have studied the self-disinfecting power of the skin. The body surfaces so far investigated by us show that they possess the power to regulate the bacterial flora in contact with the epithelial covering.

The contents of the lumen of the upper half of the small intestine are slightly acid in reaction. This segment of the small intestine is almost free from bacteria in the normal animal. Bacteria placed in this part of the small intestine, either by oral ingestion or by injecting directly into the lumen, are rapidly destroyed. The slightly acid reaction of itself does not play a