

Cholera in the Manicaland Province of Rhodesia February to May, 1974

BY

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IN ANTICIPATION

Cholera had been creeping down Africa for at least two years (*Wkly. Epidemi. Rec.*, 1973) and rumours of its penetration into neighbouring territories had become increasingly persistent in the later months of 1973. Angola had in November 1972 between 200 and 300 cases and with the large volume of traffic between that country and Mozambique, there was a certain inevitability that cholera would reach the east side in due course. Since the Manicaland Rhodesian—Mocambique border is some 820 km long and virtually uncontrolled, provisional measures were instituted early in 1973. These consisted of:

1. Sanitary and Health education teams engaged on the improvement of sanitation and water supplies concentrated their efforts on those areas particularly at risk.
2. A geographical census of safe water supplies, a census of available water bowsers and other means of transferring water was taken.
3. Stocks of vaccine, prophylactic and therapeutic drugs and parenteral fluids were built up and maintained in readiness for rapid distribution to affected areas. Laboratories were alerted to expect diagnostic specimens.
4. Five mobile teams were trained to deal with the disease both prophylactically and therapeutically. Health Inspectors were instructed to maintain a constant watch for gastro-enteritis cases.
5. Arrangements were made with the Civil Authorities (Internal Affairs, Police and Immigration) to limit and control movement of the population both nationally and internationally.
6. As a precautionary measure all food handlers in the area deemed at risk were placed under regular surveillance.

On September 18th, 1973 reports that cholera had occurred in Mozambique adjacent to the remote Nyamaropa T.T.L. some 200 km. north of Umtali were received. Immediately supplies were taken to the area, the frontier closed and patrolled as well as possible, and a school straddling the border temporarily shut down.

In the event no clinical cholera was reported from the Rhodesian side of that area during the Manicaland epidemic, all cases occurring over 300 km. further south or 100 or more km. to the north.

INTRODUCTION

Cholera entered Rhodesia during the latter half of 1973 in the north eastern part of the country near the border with Mozambique. Over the next few months outbreaks and sporadic cases occurred over a wide area but all were confined to the north of Salisbury. Several hundred cases were identified in all, the last towards the end of May, 1974.

A separate outbreak began at the end of January, 1974 in the south east of Rhodesia, some 300 km. away from where the first outbreak was, at that time, reaching its maximum level. This epidemic extended along the Sabi river for about 100 km. and over 200 cases were diagnosed by the time it too died out in May.

The two areas involved are very different from each other. The terrain in the north is harsh, dry, scrubland while that to the south is to a considerable extent irrigated farming land both European and African. To the north the population density is low and there is little mass migration; to the south the density is high and seasonal recruiting for harvesting brings in thousands of migrant workers of all ages both from other parts of Rhodesia and from Mozambique. The tribal groups are quite distinct in each area and while the African tribesman is an impressive long-distance walker there is little traffic between the two groups. This unlikely interchange would have been even further reduced by an unusually heavy rainy season causing river crossings to become impassable for weeks at a time.

It was thus considered that the two outbreaks were in no way related in terms of origin or in the exchange of infectious persons. This communication describes only the problems in the south east with particular reference to the investigations carried out and methods of control.

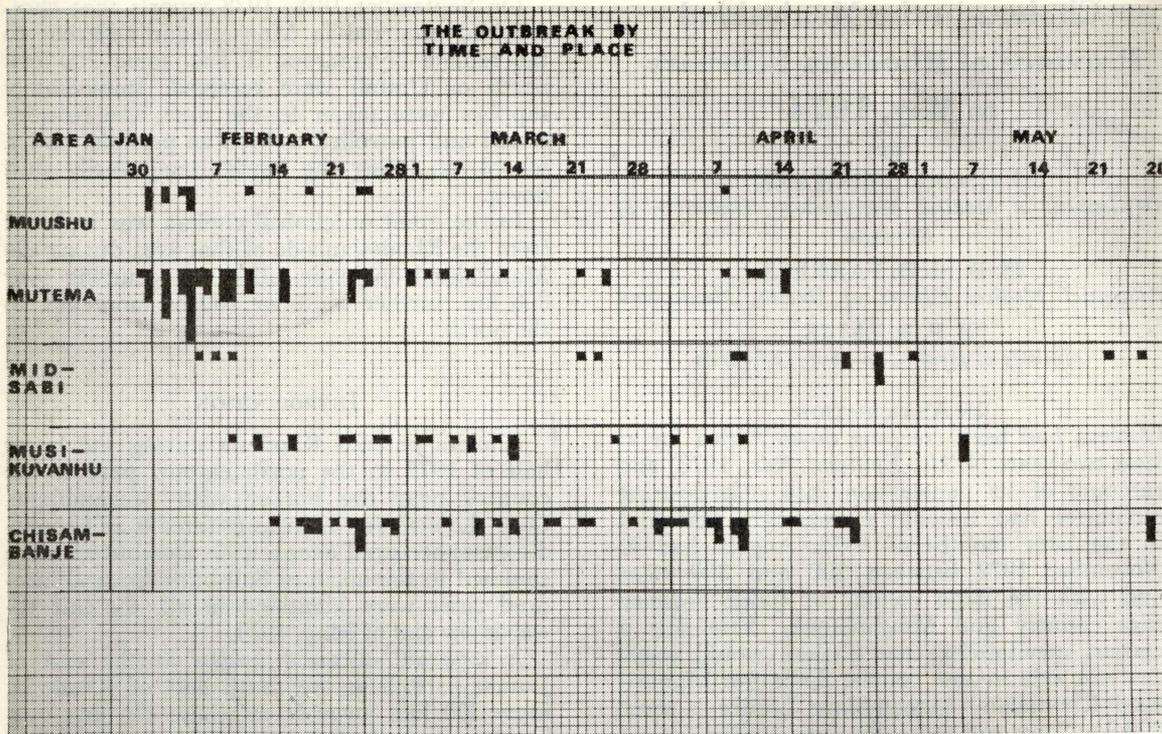


Fig. 1.—The table shows the time and locality of each case recorded in the outbreak.

GEOGRAPHY

The area involved is shown in Figs. 1 & 2. All cases occurred to the east of the Odzi river in the north and the Sabi in the south. Throughout the whole length of the epidemic area irrigation schemes extend from the river for distances varying from 200-300 metres to 8 km. or so. The cases came from, starting at the north, Muushu tribal area to the north of Birchenough Bridge, the Mutema Tribal Trust Land which is separated by the Tanganda river from the European Mid-Sabi irrigation scheme (Sabi-Limpopo Authority (S.L.A.)), the large Musikuvanhu Tribal Trust Land and the Tilcor Development Corporation Chisambanje European administered irrigation scheme in the Ndowoyo Tribal Trust Land. One isolated case came from Mutandahwe further south, 40 km. from the junction of the Sabi and Lundi rivers. To the east from the edge of irrigated areas the land rises steeply, though not impassably, between 600 and 800 metres as far south as the lower end of Musikuvanhu. Major roads run through the whole area as shown. There are bridges at Birchenough Bridge and

100 km. further south near Mutandahwe. Throughout the period of the outbreak, the Sabi river was too high to be forded or swum in safety. On the far side of the mountains lies the long Mozambique border across which there is a regular traffic of work seekers.

THE PEOPLE

The indigenous population in all areas except the Mid-Sabi scheme, live in kraals scattered haphazardly over the countryside. These are clumped mainly in family groups of up to six or seven huts with occasional larger settlements near stores, clinics, markets and other natural meeting points. Water is taken generally from where it is most conveniently available — sometimes a river but most usually from standing rainwater and pans. There are few boreholes and wells normally available. Food is either grown locally or purchased, and the level of nutrition is high. Cattle and goats abound throughout the whole area. The main commercial crops are cotton, maize, wheat and bananas.

There are few latrines and faecal contamination of water holes is always possible.

Mid-Sabi is divided into two sections: 23 — 200 acre farms each under a European owner, and five farms worked directly under the S.L.A. Each has a permanent labour force of 100 or more workers, who live together with their wives and families, in compounds either adjacent to the Sabi river or in the centre of the estate. Water is either from boreholes or piped direct from the irrigation canal, though washing and ablutions are done either in the river or one of the balancing dams. Each year at cotton picking time the permanent force is supplemented by up to 300 migrant workers per farm who are housed either in the same or separate compounds. Pit latrines are provided but the majority prefer to use the traditional bush methods. Water supplies however, are not readily contaminated. The European household takes water from boreholes fed from a large underground aquifer at a depth of 90 metres.

There is a constant free and easy movement of persons between all areas.

GENERAL DESCRIPTION OF THE OUTBREAK

When cholera was first reported from Manicaland at the end of January, 1974 it had already been epidemic in the north-eastern part of the country for nearly two months. The Manicaland outbreak reached its peak during February and March, waned during April and died out by mid-May. The rains were unusually heavy during the 1974 season, the rainfall figures being 190, 50, 28 and 14 mm. respectively for February, March, April and May. The mean local ambient temperature was just under 30°C and the relative humidity varied from 65 to 96 per cent.

An isolated case occurred at the Mutandahwe mine compound in December, 1973 in a local African from whom no relative epidemiological information could be obtained. The interval between this case and the next ones was over six weeks which suggests little likelihood of there being much relationship between them.

The first cases proper were reported simultaneously on the 31st January from the Muushu and from the Mutema Tribal Trust Lands. In Muushu five cases occurred within three kraals a few miles apart and no apparent connection between any could be traced. In Mutema the first six cases were all from Manese kraal but from a number of different families. Within the next week there were four new cases involving two further kraals in Muushu and 14 more in Mutema involving 11 kraals over an 8 km. radius. Thereafter while only four sporadic cases were reported from the

Muushu area, in Mutema the infection spread to the Sabi river in the west and the Tanganda in the south in a constant but sporadic fashion and a total of 45 cases were finally recorded.

On February 11th the first cases were diagnosed in Chibuwe kraal in the Musikuvanhu Tribal Trust Land and on the 15th Chisambanje reported three cases from a kraal in the Ndowoyo area. In Chibuwe 25 further cases occurred from 10 different kraals over a widely scattered area up to early May. In Ndowoyo there were a total of 46 cases again over 10 kraals but here there were 11 cases from each of two kraals. The disease persisted longest in this area, the last cases being a batch of three from the same village on May 29th.

The only cases reported from Mid-Sabi occurred during the March and April on two farms, one privately owned and in the centre of the scheme, and the other belonging to the S.L.A. situated at the northern end.

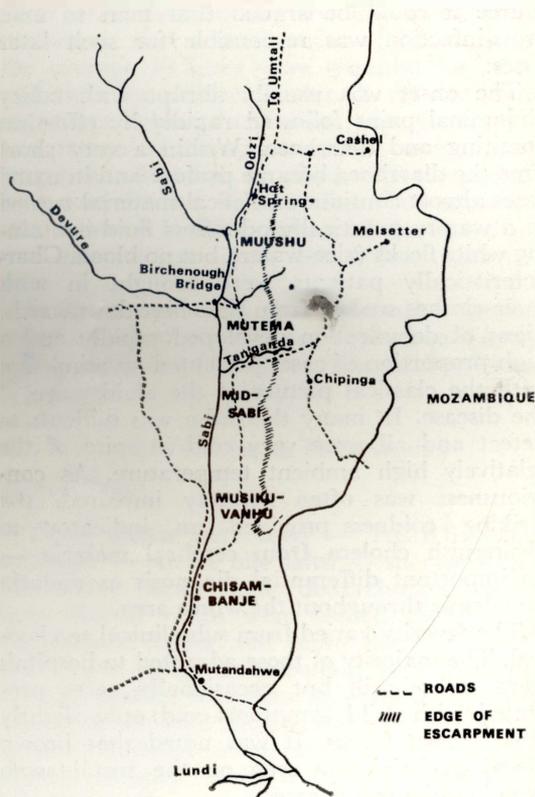


Fig. 2.—Map of the area involved.

Figs 1 & 2 give a graphical representation of the outbreak case by case in time and by locality.

In addition to those described above a few sporadic cases were reported from Umtali, Chipinga and Tamandayi which were some

distance from the main centres of activity. There were no incidents in which large numbers of persons were infected from a common source causing an explosive type of epidemic such as has occurred in many other countries (e.g. Coulanger and Coulanger, 1972). In spite of the severity of the clinical disease in those persons who were infected, the pattern was throughout of an endemic smouldering type with little or no apparent relationship between one case and the next.

CLINICAL PRESENTATION

Because of the endemic nature of the outbreak it was difficult to assess the incubation period. In the few cases in which families were involved, it appeared to be about three days. Where there was clustering in a particular area cases followed each other often by as short a period as 24 hours. In the absence of a common source it could be argued that man to man cross-infection was responsible for such later cases.

The onset was usually abrupt with colicky abdominal pains followed rapidly by effortless vomiting and diarrhoea. Within a very short time the diarrhoea became profuse and in many cases almost continuous. Faecal material turned to a watery practically odourless fluid containing white flecks (rice-water) but no blood. Characteristically patients were brought in with their clothes soaked from the chest downwards. Signs of dehydration developed rapidly and a high proportion of cases presented on admission with the classical picture of the algid stage of the disease. In many the pulse was difficult to detect and all were very cold in spite of the relatively high ambient temperature. As consciousness was often severely impaired, the striking coldness provided an indicator to distinguish cholera from cerebral malaria — an important differential diagnosis as malaria is endemic throughout the whole area.

The severity varied from sub-clinical to classical. The majority of those admitted to hospitals were severely ill but occasionally cases presented with mild symptoms and only slightly loose brown faeces. It was noted that brown faecal material was seen on the rectal swab even in quite severe cases.

58 per cent. of the cases were women. The youngest patient was 18 months old and the eldest well over 80; the majority were between 25 and 45 but 27 were under 14 years old. The most clinically severely affected were the young adults.

Following treatment (see below) clinical recovery was as dramatic as the onset. Generally, even in the most severe cases, within six hours patients both looked and felt much better and many wanted to smoke. By the time rehydration was complete — usually about 12 hours, the patients asked for and took solid foods.

Vomiting and abdominal pain usually stopped once rehydration had been started, and the diarrhoea ceased within 12 hours of the completion of rehydration. One or two patients had minor relapses 24 to 48 hours after apparent recovery from the initial episode. All four patients in the first trimester of pregnancy aborted but recovered themselves, in one patient in the second trimester the pregnancy continued. No late sequelae were reported. There were nine deaths, all but one in the early days of the epidemic, and all but one from Muushu and Mutema. Seven were women and two men. The later death was in Ndwoyo (Chisambanje) in an already frail old lady of at least 80. All deaths were apparently within 24 to 48 hours of the onset and were attributed to the effects of dehydration. In one case an autopsy was carried out (Dr. Carshalton, Chipinga) but only non-specific inflammatory changes in the small bowel were found. It is probable that other cases and deaths occurred in the Tribal Trust Lands and were not reported to the authorities, but as the disease and its consequences and the help available were well known, it is unlikely that there were very many.

Convalescent carriage of the organism is discussed in a later section but suffice it to say here that in a small number of cases that were followed up for several weeks most had ceased to excrete the vibrio within 48 hours of recovery, and none carried for longer than three days.

DIAGNOSIS

It was decided that while most cases with the symptoms described above were probably cholera, each one should be confirmed bacteriologically and that any relatives or contacts available should also be swabbed. The information thus obtained would include:— the proven incidence of the disease, the type or types of *Vibrio* involved, the incidence of other diseases mimicking cholera, the patterns of sensitivity to antibiotics and any changes thereto, the duration of convalescent carriage and the incidence of sub-clinical infection. The laboratory methods are described below. It should be noted that both *Vibrio* isolation and its serological

confirmation as *V. cholera* can be and was done efficiently in the field (Craig, Cruickshank, Ellis and Farrell, 1974).

In all cases diagnosed clearly clinically as cholera, confirmation was obtained culturally in the field within 18 hours at most and often within six hours. Of the more clinically doubtful cases, five or six grew no *Vibrio cholera* and no other bacterial pathogen. Parallel laboratory cultures of cases, suspects and contacts failed to reveal any missed by the field technique. All organisms tested were of the El Tor biotype Inaba serotype and were sensitive to chloramphenicol, tetracycline, ampicillin, cephalothin, streptomycin, kanamycin and sulphadimethoxine. No changes to this pattern were observed during the outbreak.

CARRIAGE RATE AND SUBCLINICAL INFECTION

In patients treated with tetracycline as well as by rehydration the vibrio was cleared from the gut within three days at the most. A few cases were treated by rehydration but with no antibiotic and followed up daily thereafter. Generally one to two days longer carriage was noticed, and one patient cleared only seven days after the establishment of treatment. There was, however, no difference in the rate of clinical recovery between the antibiotic and no antibiotic groups.

Surprisingly only a few close contacts or relatives of florid cases either developed the disease or were shown, at least over a period of a few days, to be carrying the organism. Therefore from the one farm in the Mid-Sabi area in which cholera had been found, 160 field workers had rectal swabs taken and plated immediately. Three totally symptomless carriers were found and one of those proved negative at that time developed the disease 12 hours later. It should be recognised that carriers generally excrete 10^6 organisms less per gram of faeces than do clinical cases and that rectal swabs are less satisfactory than stool specimens for tracing such people but they are the only practical method possible under the circumstances. Therefore it can be assumed that there were a significantly higher number than were actually demonstrated by this technique.

More striking evidence was obtained from serological studies. Out of 268 sera taken from apparently healthy people in three separate affected areas, 36 (13.4 per cent.) had titres of 1 in 15 or more against *Vibrio cholera*.

Clearly the subclinical infection rate was very high — several times that of the rate of clinical disease.

TREATMENT

Rehydration and the re-establishment of electrolyte balance was the primary objective. Initially some patients were treated with Darrows solution and others with physiological saline but as there was no apparent difference in response between the two groups, saline was adopted because of its lower cost. Physiological saline supplemented with two grams of potassium chloride per litre was administered as rapidly as possible either intravenously or, if no veins could be found, intraperitoneally. Cut down facilities were not available in the emergency field hospitals, but as soon as intravenous therapy became possible other routes were abandoned. In some cases six or seven litres were given in the first few hours and a further six over a total period of 12 hours. On the average 10 litres were required for adults and eight for children under 14. As the vomiting stopped glucose and saline were given orally and parenteral therapy was rarely necessary for longer than 12 hours by which time the patients were convalescent. Tetracycline (300 mgm IM. stat. followed by 250 mgm. q.i.d. for five days) was given to minimise the *Vibrio* excretion time.

In those few cases that relapsed a further course of rehydration brought the disease under control within a few hours. The antibiotic was not changed.

INVESTIGATIONAL TECHNIQUES

Bacteriology

Three media were used — T.C.B.S. (thio-sulphate. — citrate bile salts. — sucrose agar), McConkey agar, and deoxycholate citrate agar. Rectal swabs were seeded thickly onto the cholera media and more thinly onto the D.C. agar using a half-plate per specimen. Usually plating was done directly at the bedside or in the field. Incubation was carried out at local ambient temperature as the circumstances dictated — on a window ledge, in a Landrover glove compartment, etc. within a sealed plastic bag to prevent drying out. Temperature varied between 20° and 33°C. In positive cholera cases characteristic bright yellow to orange growth was detectable between six to 18 hours. Occasionally certain coliforms grew yellow-white colonies which were quite

easily distinguishable from *V. cholera*. Confirmation was made in the field by slide agglutination using polyvalent antiserum according to the method of Craig et al, 1974. Recovery rates were tested at 37° and at room temperature and no significant difference was found.

Swabs from index cases and from cases picked at random during the outbreak were placed in modified Stuarts transport medium or into alkaline peptone water and transported to the University laboratory generally within 24 hours. All strains isolated were motile and fermented with acid but no gas production, glucose, maltose, sucrose and manitol, and after three days lactose. They were indole positive, V-P positive, M-R negative and cholera red positive. All were resistant to polymyxin B and were haemolytic on sheep blood agar. The organisms agglutinated with polyvalent and with Inaba anti-serum but not with Ogawa. The vibrios were clearly of the El Tor variety and no other variants were encountered during the outbreak.

Water samples were examined by incubating aliquots in alkaline peptone water and sub-culturing after 24 hours onto T.C.B.S. agar. Cotton and other fomites were soaked in alkaline peptone and the supernatant treated as for water.

Serology

Sera were tested for agglutinating activity against live suspensions of the El Tor vibrio (McIntyre and Feeley, 1964). Suspensions were made by scraping off the overnight growth of the vibrio on nutrient agar into phosphate buffered saline and adjusting to a concentration corresponding roughly to No. 2 Brown's tube. Equal volumes of the suspension and serum dilution were incubated overnight at 37°C. Formalinised suspensions gave lower and less reproducible titres.

Sulphonamide levels

Serum sulphamethoxine levels were measured using a standard diffusion cup method to the nearest 50 µl/ml. using a fully sensitive staphylococcus. During a course of administration serum levels between 100 and 200 µg/ml. were found.

Disposal of Infected Material

Excreta and disposable items were buried in a deep pit with chloride of lime. Bedding, clothing and other fomites were soaked for 12 hours in a phenolic disinfectant, rinsed and

sun dried. The dead were buried in chloride of lime.

Control Measures

The purposes of public health measures were to control the outbreak where it was active and to prevent the spread to neighbouring or more distant areas.

Local Control

Hospitals were available within the stricken area at Birchenough Bridge, Nyanyadzi and St. Peter's Mission, Chisambanje and an emergency hospital was set up in a school building in the Mutema Tribal Trust Land. Though some outside cases were treated in Umtali and Chipinga hospitals, no cases were transferred outside the epidemic area. Birchenough Bridge and Nyanyadzi hospitals were manned by a senior medical assistant and his staff, and the others by a nursing sister and a medical assistant with locally recruited orderlies where necessary. A senior health official took charge of the day to day execution of the control plan under the overall direction of the Provincial Medical Officer of Health and his senior staff.

Cases were admitted and treated and, if possible, not discharged until a negative stool culture had been obtained. Attending relatives and friends were also swabbed and if necessary treated. The villages and kraals from whence the cases came were kept under daily surveillance for further developments. Water samples and occasionally food samples were taken from possible sources of infection but uniformly without positive results.

Vaccination was not undertaken (a) because of doubts expressed in recent world literature as to its protective efficiency (Mosley et al, 1972), (b) because of the difficulty of giving a full course of injections, (c) because of the delay in establishment of what protection there might be and (d) because of indications that the carrier state is associated with high circulating antibody titres (Dizon et al, 1967) and therefore the danger of possible prolongation of carriage time following vaccination.

The contact population at risk was given sulphonamide prophylactically in the form of the long acting sulphadimethoxine (Depomide, Sulphribon, (Central African Pharmaceuticals)). A loading dose of 2 grams was followed by 500 mgm. per day. Initially four-day courses were given followed by three days rest and then a new course. This became difficult to administer and a regimen of one 500 mgm. tablet per day was adopted. The dosing was placed under the supervision generally of health

assistants, farmers and managers. Random blood samples were taken from time to time for serum sulphonamide levels to detect defaulters who would generally amount to 10-20 per cent. in any particular locality. The surprise on being found out was in most cases very salutary.

The attention of the population was drawn to the importance of clean water supplies and the possible routes of contamination. The dams, water holes, boreholes and rivers proved negative on sampling though low numbers of organisms could not be excluded. Arrangements were made to supply fresh water to villages where possible. Clean water bowsers were available in the fields on European farms for the use of the workers and the carriage of water from other sources was discouraged. No chemical disinfectants were distributed.

Movement between villages and kraals in the affected areas was discouraged but was quite impossible to control and obviously took place extensively.

All migrant workers brought in for the cotton harvest — men, women and children — were placed immediately on sulphonamides and maintained thereon throughout the epidemic.

General Control

The whole area involved and the neighbouring Chipinga district were gazetted after consultation with the Ministry of Internal Affairs, as restricted zones, which provided for the control of persons entering and leaving. Road blocks were set up on all major roads at the zone boundary and a certificate signed by the Health Officer was necessary to pass the barrier freely. The destination of all traffic was noted and all travellers were required to start a course of sulphonamide. Through traffic was discouraged and persons advised not to obtain food or water on the way.

Problems arose concerning the movement of produce, i.e. cotton and bananas, from farms known to have had cases, to processing plants or markets. Many cotton samples were heavily contaminated with faecal organisms but no vibrios were found. It was recommended that the cotton should be held for at least three days and if possible exposed to sunlight away from any likely source of infection before being moved out. Infection from bananas seemed less likely but they were passed through a hypochlorite rinse before being released.

It was recognised that these measures were in many ways token gestures as only move-

ments by road could be controlled. However, it was felt that the measures were salutary and served to indicate the seriousness with which the situation was regarded by the authorities. Further, those on the road were the ones most likely to travel long distances perhaps while they were incubating the infection. Indeed one patient in the early stages of the disease was removed from a bus at the Nyanyadzi road block controlling traffic to Umtali.

Epidemiology

Two types of cholera epidemic are recognised — the explosive and the protracted (Mosley, 1970). In the former a common vehicle of transmission is readily evident and a large number of cases appear in the affected area over a few days. In the protracted type cases appear sporadically in ones or twos over weeks or months; in many instances the mode of transmission is difficult to discern.

The Manicaland outbreak was clearly of the protracted type, the cases appearing over 3½ months, never more than four in one day and rarely more than five in total from any one kraal or village. Table I indicates the incidence in the 60 kraals involved. No direct evidence of water contamination was found but an attempt to demonstrate that water, probably infected,

Table I

| No. of patients | No. of kraals |
|-----------------|---------------|
| 1 | 35 = 58,2% |
| 2 | 10 = 16,7% |
| 3 | 6 = 10,0% |
| 4 | 1 = 1,7% |
| 5 | 3 = 5,0% |
| 8 | 1 = 1,7% |
| 9 | 2 = 3,3% |
| 12 | 1 = 1,7% |
| 14 | 1 = 1,7% |

Table showing the number of patients from each of the 60 kraals involved.

(the Tanganda river receiving sewage from the involved Mutema T.T.L.) flowed into the Sabi river and back into the irrigation system of the Mid-Sabi scheme, using 10 gallons of carbol fuschin as marker, was a failure. The dye was diluted to extinction too rapidly and one engine of the observer's aircraft stopped! Person to person contact type spread is regarded as very rare and could hardly account for the widely scattered sites from which cases came. It must therefore be assumed that the large bodies of water abounding in the area — rivers, flooded fields, balancing dams and small pans of standing water were contaminated at a low level

and during the period of the outbreak were fairly constantly reinfected. In view of the comparatively few clinical cases from any one area, the implication must be that a significant proportion of cholera cases were inapparent.

Rectal swabbing of 170 workers picked at random revealed three non-clinical carriers, but as the technique was recognised to be inefficient, a serological survey to demonstrate anti-Vibrio cholera agglutinating antibodies was undertaken in three specific affected areas. The incidence of antibody was nine out of 154 (5.9 per cent.), 18 out of 91 (20 per cent.) and nine out of 23 (39 per cent.). No one in any of the sampled groups had had clinical cholera. Since no vaccinations had been undertaken these results must reflect a very high non-clinical infection rate. It can, therefore, be suggested that the disease was maintained and spread largely by the carriers of the vibrio ($<10^2$ per gram of faeces) moving from place to place contaminating water supplies and food stuffs. Either the virulence of this strain of El Tor was low or the dose was generally too small to give rise to overt infection. A few received a high dose or were susceptible for other reasons, e.g. nutrition, and developed cholera proper. Where there were adequate supplies of clean fresh water there was no cholera.

The original source of the outbreak remains purely speculative. Cholera was present in north eastern Rhodesia and it is believed in Mozambique as well. The terrain between both possible sources and the affected area is "tough" and hilly but readily passable and there is a constant traffic to and fro. The disease was presumably introduced and reintroduced by carriers from an epidemic area contaminating, at low level, water or food stuffs or was possibly carried in infected food supplies.

DISCUSSION AND CONCLUSIONS

Cholera is a disease classically acquired by ingestion of the vibrio and during the disease vast numbers of the organism (10^7 - 10^9 per gram) are passed in the copious fluid stools (Barua, 1970). The environment becomes heavily contaminated and in relatively clean water the vibrio may remain for long periods (Pesigan, Plantilla and Rolda, 1967). Water is by far the commonest vehicle of transmission, and most authorities agree that contact spread is very uncommon.

Evidence shows quite clearly that this outbreak was of the endemic or protracted type

and, as is usual in such circumstances, no common service or vehicle was found. It is likely that less than one out of every five to 10 of those infected developed clinical disease of any sort, and, as man is the only known host of *Vibrio cholera*, these carriers were responsible for the maintenance of the infection in the population. It is curious, with a susceptible population such as this one, that explosive epidemics were not super-imposed upon the endemic disease through infection of water or food supplies, as this usually happens in other such outbreaks. Climatic conditions best suited to overt cholera have yet to be defined in this part of Africa but the abrupt cessation of the epidemic by mid-May suggests that both temperature and humidity may play a significant part in the epidemiology of the disease here. There is no rule — in places quite close to each other contrasts are found (Mosley, 1970). In Calcutta the peak incidence is in the hot dry season before the monsoon, while just across the Ganges delta in Dacca in Bangladesh the monsoon rains usher in the seasonal epidemics. Southern Africa may follow the latter pattern.

While clinical cholera cases excrete 10^9 or so organisms per gram of stool, carriers produce many less (10^2 per gram) (Barua et al 1967). If this strain is of low virulence it could well be maintained in the population for long periods particularly bearing in mind the short duration of immunity from clinical disease or immunisation. A saving grace is the relatively short survival of the organism outside the body and the generally accepted maximum of two weeks for carriage whether convalescent or non-clinical. However even if the infection should eliminate itself during the "off season", there continues the risk of reintroduction from outside — a factor which can only be controlled in those territories themselves. It must be borne in mind that as the dry season progresses water will become scarcer and greater therefore will be the chance of infection of a common source vehicle and a subsequent flare-up when environmental conditions, whatever they are, become advantageous.

Assessment of control measures is more difficult than may appear at first perusal. One feature that does seem totally satisfactory is the therapeutic regimen in established cases. The type and extent of fluid replacement and administration of tetracycline failed in very few cases indeed; all failures occurred early in the epidemic where experience was limited and when the population did not recognise the

necessity of obtaining help as soon as possible after the onset of symptoms.

A second cause for satisfaction is that the disease remained confined with very few exceptions to those areas afflicted within the first three weeks. The few cases which were reported from the west side of the Sabi river in Victoria province most likely were derived from people from the epidemic area, but those occurring to the north in Umtali and in the Cashel area were probably direct imports from the east. It must therefore be concluded that the movement restrictions are effective presumably in part due to physical restriction, the insistence of prophylactic chemotherapy for travellers, and an enforced awareness of the potential seriousness of the situation. Sulphonamide administration gave cause for some anxiety because of possible side-effects; in fact only one came to light — in a European water bailiff who developed methaemoglobinemia. He recovered rapidly and was placed on tetracycline instead.

The real question is still whether the local preventive measures had any real effect on the course or termination of the epidemic. The results of estimating serum sulphonamide levels indicated clearly that any assumption of total co-operation in taking these drugs was naive in the extreme. Indeed while co-operation with chiefs and headmen generally was satisfactory — even good, there were areas where it was rather negatively reluctant. As always rumours abounded and the story that the tablets were in fact sterilising agents was widespread. It was this gap in coverage that ostensibly permitted the maintenance and transmission of the infection. There must however have been large numbers of people in the Tribal Trust Lands also at risk and to whom no prophylaxis was given, and the thought that there might have been no greater risk had no such preventive steps been taken is a matter for some concern. In many parts of the world the expense of such drugs prohibits their prophylactic use and the provision of clean water supplies seems enough to bring epidemics under control and, indeed, here too it was apparent that where there were to be found reasonable standards of hygiene, cholera would not occur. The proof of the pudding is however in the eating. Conditions in terms of temperature, humidity (using Roger's criterion, see Manson-Bahr, 1966), living, water and food supplies all were ideal for a widespread and severe outbreak and in the end only some 200 clinical cases occurred and we believe the measures taken must be supported.

The development of field laboratory methods enabled each suspected case to be confirmed and while the majority of acute diarrhoeas proved to be cholera some did not. Other serious gastro-intestinal disorders are endemic throughout Rhodesia and delay in the institution of correct treatment or in recognition of the real nature of a disease may be disastrous. Further, the more sophisticated a community, the greater is the disruption when suspicion of dangerous infection is directed at it and many social strata were represented directly or indirectly in this outbreak. Search for carriers amongst relatives and contacts is also facilitated as results are rapidly available and action can consequently be instituted with minimal delay. Using a plate for four samples the cost, small as it is, is a low price to pay for accuracy in disease control.

The cost of exercises such as this is however considerable. There are rather more than 100 000 people in the area involved and the drugs bill alone, which included intravenous fluids, giving sets, tetracycline and sulphonamides, was R\$43 000. At approximately 50 cents per head of population with only 200 cases the figure may seem high but that there was probably up to 10 times as many subclinical cases puts the budget in a much more favourable light.

Will the disease return? We see no reason to suppose that it is not now passing gently through the population unnoticed ready to reappear clinically when circumstances are right. However epidemics tend to be self-limiting and the infection has disappeared from most affected areas as mysteriously as it came six times before so we can be reasonably confident it will do so again. However it is to be hoped that our experience of this season's epidemic will be of value should there be one next year.

ADDENDUM

Since this paper was completed, a number of sporadic cases of cholera have been reported from the same area. The first of these occurred seven weeks after the last case of the epidemic described.

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The control of this outbreak was accomplished only by the sterling and dedicated efforts of many medical and para-medical personnel and it would be invidious to name some and not others. The authors wish therefore to acknowledge their debt to all those who took part in the exercises described in this paper.

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ADDENDUM

Within two months of the last of the events described in this paper, a fresh outbreak was reported from the Musikavanhu Tribal Trust Land. The pattern of spread was until recently of the endemic type and over 60 cases with seven deaths occurred in Musikavanhu and Mid-Sabi, and in the adjacent Sangwe district of Victoria province. In October an explosive epidemic of 30 cases with five deaths was superimposed in a localised area of Musikavanhu in which it was possible to pinpoint a probable common water source.

These events are compatible with the theory proposed that the disease is largely maintained by widespread clinically inapparent infection.