The cholera epidemic in South Africa, 1980 - 1987

Epidemiological features

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Summary

During the cholera epidemic in South Africa, 1980-1987, 25 251 cases of cholera were bacteriologically proven. The case-fatality rate was 1,4%. Outbreaks occurred in the summer rainfall season. Age-specific attack rates followed the pattern typically found during the 'epidemic phase' of the disease in most years. The vast majority of patients were black South Africans living in rural areas with an average annual rainfall in excess of 600 mm. The containment strategy employed is summarised.

Despite the apparent eradication of the disease, it is strongly recommended that vigilance should be maintained and investigations of all possible sources of infection and all human contacts of any new proven case should be carried out speedily and thoroughly.

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The dynamic spread of the seventh pandemic of cholera can conveniently be subdivided into three geochronological periods. The first stage involved the invasion of all the states of South-East Asia, the second the invasion of the states of mainland Asia and the third stage the Middle East-Afro-European invasion. Cholera in South Africa was clearly part of the third period. As early as 1971 South Africa was considered to be at risk. Hot, humid summers, sea-ports, overcrowded communities with a low standard of environmental sanitation and scanty, restricted and unprotected water supplies in certain areas facilitated the introduction of cholera into South Africa.²

A Cholera Surveillance Programme for the mining industry was started in November 1973. During March 1974, two eastern Transvaal coalmines and 1 goldmine yielded cholera-positive sewer pads, and on 25 March 1974 the sewer of a western Transvaal goldmine also became positive. Two healthy cholera carriers were found on this mine during a rectal swab survey. The epidemic was contained by the administration of oxytetracycline and mass vaccination of all mineworkers.³ In 1978, 3 confirmed cases of cholera in South African tourists were notified, none of whom had acquired the disease locally.⁴

The first case of cholera in South Africa in an open community, was diagnosed on 2 October 1980 at Shongwe Hospital in KaNgwane. An epidemiological investigation was launched on 8 October 1980. The one common factor found among all patients was that they lived on farms at Malelane and had drunk water from the Crocodile-Malelane irrigation canal.⁵

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Clinical features

This cholera pandemic was caused by *Vibrio cholerae* El Tor Inaba serotype. El Tor is a hardier organism and survives for longer periods in the environment than the classic form; it is also excreted for longer periods in stools after infection and 75% of cases are asymptomatic.¹ Persons infected with this organism remain mobile and may spread the disease, shedding millions of vibrios in their stool for about 2 weeks.

In severe cases cholera causes profuse diarrhoea of sudden onset, which is explosive in character. It also causes typical rice-water stools and severe dehydration ensues. The stools are odourless, except for a mildly fishy smell. Effortless and projectile vomiting is common and dehydration is marked in patients with both diarrhoea and vomiting. A South African patient was shown to have lost 25 litres of fluid in 24 hours per rectum. Such profound dehydration is frequently accompanied by subnormal temperatures. In a Natal study6 it was found that the majority of patients admitted to hospital were severely dehydrated and shocked, with 72% presenting with systolic blood pressures below 100 mmHg; blood pressures were unrecordable in 10% of patients. The severe electrolyte imbalance following such a dramatic derangement of the normal gastro-intestinal metabolism is the cause of the severe leg muscle and abdominal cramps often encountered in untreated patients.

Epidemiology

Being part of the seventh pandemic, the entire recorded presence of cholera in the general South African community will, in this article, be referred to as the 'cholera epidemic'. The individual phases, on the other hand, will be referred to as 'epidemic periods'.

The surveillance of cholera in South Africa was based on bacteriologically proven specimens of *V. cholerae* El Tor; in all, 25 251 positive cases were identified, all of the Inaba serotype.

Seven cholera epidemic periods, designated Cholera I - VII, occurred in South Africa between October 1980 and July 1987. The duration, extent and case-fatality rate of the various periods are summarised in Table I.

Time

The individual outbreaks by month of occurrence are illustrated in Fig. 1 and Table II.

Cholera has a seasonal pattern that appears to follow the ambient local patterns of rainfall and temperature (Fig. 2).⁷

Place

The initial cases were all from KaNgwane and the Malelane area in the eastern Transvaal, in close proximity to the Mozambique border, whence spread extended upstream along the courses of the Crocodile and Kaap Rivers and then further afield (Fig. 3).4 More than 99% of all cholera patients fell ill in

IABLE I. NO. C	F CASES AND DEATHS FR	OM CHOLERA IN	SOUTH AFRICA	, 1980 - 1987
	Duration	No. of cases	No. of deaths	Case-fatality ratio (%)
Cholera I	Oct 1980 - Jul 1981	3 786	42	1,1
Cholera II	Aug 1981 - Jul 1982	11 141	218	2,0
Cholera III	Aug 1982 - Jul 1983	7 638	62	0,8
Cholera IV	Aug 1983 - Jul 1984	1 977	20	1,0
Cholera V	Aug 1984 - Jul 1985	568	4	0,7
Cholera VI	Aug 1985 - Jul 1986	134	2	1,5
Cholera VII	Aug 1986 - Jul 1987	7	0	0
Total	The state of the s	25 251	348	1.4

	1000	In-		Cholera epid	demic period	WATER THE WA	Manual State	Jeth dre
Mo.	1 1980/81	II 1981/82	III 1982/83	IV 1983/84	V 1984/85	VI 1985/86	VII 1986/87	VIII 1987/88
Aug	-	1	101	62	7	12	4	0
Sep		0	92	64	1	7	0	0
Oct	259	19	220	189	6	0	0	0
Nov	334	476	442	199	1	0	3	0
Dec	360	913	488	303	7	2	0	0
Jan	1 180	2 540	1 232	345	30	47	0	0
Feb	572	2 826	1 684	155	102	52	0	0
Mar	268	1 450	1 263	169	53	11	0	0
Apr	575	1 407	844	178	124	2	0	0
May	202	902	844	182	175	1	0	0
Jun	33	486	300	99	39	0	0	0
Jul	3	121	128	32	23	0	0	0
Total	3 786	11 141	7 638	1 977	568	134	7	0

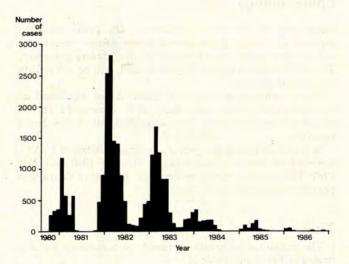
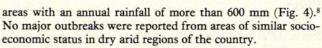


Fig. 1. Bacteriologically proven cases of cholera per month, Cholera I - VII.



The last 3 bacteriologically proven cases of cholera were reported in November 1986 from Eshowe and Greytown in Natal.

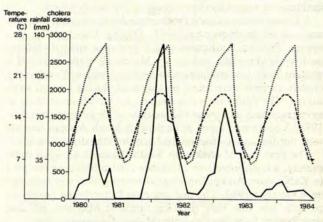
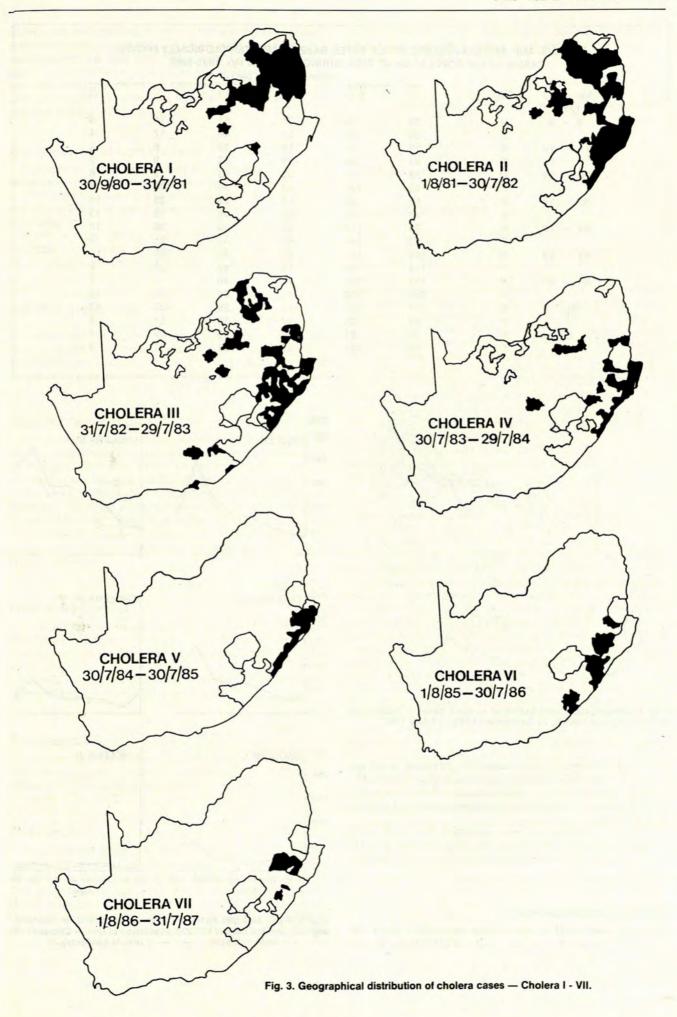


Fig. 2. Average minimum temperature, average rainfall and number of bacteriologically proven cases of cholera by month, August 1980 - July 1984 (. = average rainfall; - - - = minimum temperature; — = cholera cases).

Subjects

The age-specific attack rate was found to increase with advancing age (Table III; Fig. 5).⁴ During Cholera I, II and III the attack rate for female subjects was higher than for male subjects, while for Cholera IV, V and VI the attack rates were almost the same for both sexes. The age-specific attack rates,



CAS	3E3/100 000 1	POPULATION A					
	Cholera epidemic period						
Age group (yrs)	Sex	1	11	III	IV	٧	VI
0 - 9	M	39	58	39	51	15	4
	F	38	54	31	46	12	4
10 - 19	M	29	61	31	19	8	2
	F	44	63	29	13	9	3
20 - 29	M	38	55	32	23	8	8
	F	63	102	44	30	20	14
30 - 39	M	58	80	45	29	23	11
	F	79	122	62	34	12	13
40 - 49	M	79	117	55	46	30	16
	F	85	144	63	30	21	2
50 - 59	M	100	113	51	48	38	13
	F	114	142	74	42	25	7
60 - 69	M	187	195	88	40	7	7
127	F	200	224	145	73	57	16
70+	M	117	161	99	59	107	11
	F	89	181	97	35	51	0
Overall	M	56	83	41	35	17	7
	F	69	102	47	33	17	7

Rate

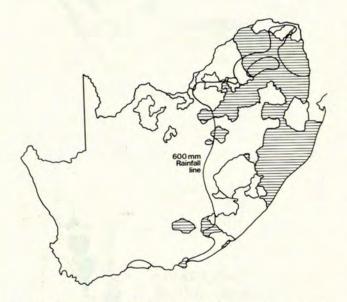


Fig. 4. Geographical distribution of cholera cases in relation to areas of highest rainfall, 30 September 1980 - 31 July 1987.

comparing Cholera I with Cholera II, increased in all age groups. Of the 1977 patients diagnosed during Cholera IV, 1968 were black, 6 Asian, 2 coloured and 1 white.8 During the other epidemic periods, the overwhelming majority of patients were also black

Cholera is a disease of poor sanitation and poor living conditions and, in South Africa, was most prevalent in fairly densely populated rural communities of low socio-economic status.

Mode of transmission

During Cholera III a matched-pair case-control study was successfully conducted in the village of Moletlane. It was

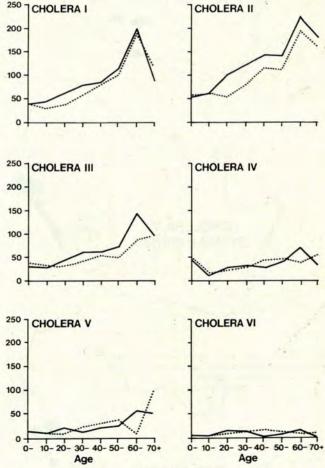


Fig. 5. Age- and sex-specific attack rates, based on bacteriologically proven cases/100 000 population at risk — Cholera I -VI (---- = male subjects; ———— = female subjects).

shown that consumption of open river water was positively associated with an increased risk of contracting the disease.⁹ Anecdotal reports suggested that shared food brought into miners' quarters after a week's leave in a cholera-infected rural area was accompanied by an increased risk of food-borne infection. A similar impression was formed after an investigation on a Natal mission reserve.¹⁰

Environmental surveillance

Throughout the epidemic, open water courses and sewage effluents were regularly and repeatedly tested for the presence of pathogenic *V. cholerae* and often found to be positive. In the eastern Transvaal spread along water courses was distinctly retrograde.¹¹

Every so often cholera struck urban communities. Such outbreaks were always very limited in extent, pointing, *interalia*, to the protective effect of safe and lavish water supplies.

Control measures

In response to the introduction of cholera to the RSA, the then Department of Health, Welfare and Pensions devised and implemented a cholera containment strategy, which embodied the following principles, several of which are quoted verbatim.¹²

Cholera surveillance

'An attempt must be made at bacteriological diagnosis in all cases of gastro-enteritis presenting at hospitals and clinics. It is essential to educate all health workers regarding cholera and to create an awareness of possible cholera cases

'All health workers must be equipped with a cholera pack containing: (i) screw-top specimen bottles containing alkaline peptone water; (ii) sterile rectal swabs; (iii) disposable gloves; (iv) wooden spatulae for collecting faecal material; (v) laboratory request forms; (vi) instruction for collection and dispatch of specimens'

Oral rehydration

'Oral rehydration of patients with diarrhoea . . . will cure many cases and save many lives In the case of cholera all but the most severe cases can be successfully treated in this way'

Mineral substitutes, electrolyte solutions and ion exchange preparations were used for oral rehydration.

Foodhandlers

'All foodhandlers in the potential cholera areas must be subjected to gastro-intestinal surveillance' This was followed by the specific instructions.

Contacts

Contacts were defined as members of the household of an infected person. They were given tetracycline 1 g immediately and 250 mg 6-hourly for 4 doses. Contacts were confined to their homes until the course of tetracycline had been completed. Health education was provided on a wide front with special emphasis on personal and food hygiene and the provision of specific instructions regarding the preparation and use of safe water. Ensuring the safety of the available water supplies

involved protection of the water source, boiling water and the use of chlorine — even household bleaches.

General

"... Disinfect houses, drains and surroundings with bleaching powder.

'Proper disposal of stools and vomitus.

Burn refuse and garbage.

'Wash soiled clothes after soaking in cresol solution.

'Chlorinate domestic water'

Unsolved problems

To date certain problems remain unsolved:

- 1. How does *V. cholerae* survive during the inter-epidemic periods? The maximum incubation period for *V. cholerae* is 5 days. It is on record that 18 incubation periods, with no cases being reported during October and November 1985, and only 2 in December, were suddenly followed by an upsurge in the occurrence of the disease (Fig. 1: Table III). Three theories have been proposed to explain this: (*i*) persistence of long-term or typhoid-like carriers; (*ii*) persistence of the organism in a free-living, altered or adapted form, capable of reverting to a pathogenic variety; and (*iii*) continuous year-round transmission by subclinical cases. At present it is accepted that (*iii*) best explains the continuation of the disease.
- Primary health care in the prevention of the disease: in certain deep rural areas the accessibility and outreach of primary care services is wanting, so that timeous intervention was difficult. This matter is receiving continuous attention and progress is certainly being made, albeit at a pace that could and should be increased.

Conclusions

The communities most severely affected were blacks in rural or deep rural, relatively high rainfall areas, such as in Natal, KwaZulu and KaNgwane. A detailed analysis of cases in Umzinto showed that at district level an epidemic would pass through a community, with cases occurring no further apart than the accepted maximum incubation period of 5 days.¹³ Most probably, what was seen was a spread from one virgin local community to the next, i.e. a continuous sequence of localised epidemics rather than real endemicity. This is largely borne out by the age-specific attack rates per epidemic period, of which there were 7.

One might perhaps be initially inclined to attribute the fall in incidence and the ultimate subsidence of cholera to the control measures instituted. While it is true that field staff did excellent work and often had to improvise, particularly at the height of the epidemic, it is probable that the disappearance of the disease was also effected by those unknown factors listed above, which continue to baffle scientists.

The single most important challenge, epidemiologically and otherwise, is to solve the riddle of the organism's survival during non-epidemic periods. The first cases diagnosed in any new outbreak are the primary source of information and must be vigorously investigated. In the meantime it is good counsel to be wary of cholera and to maintain low-level, on-going surveillance in those areas that bore the brunt of the disease. The recent reappearance of appreciable numbers of cholera patients in neighbouring Mozambique, with 126 reported cases and 14 deaths from 28 April to 4 May 1990 and 374 cases and 8 deaths from 5 - 12 May 1990, 14 merely serves to underscore the urgent need to increase our awareness and vigilance.

We should like to thank the field staff on whose diligent reporting this entire study is based; the laboratories for submitting all the reports; Professor Margaretha Isaäcson for her many invaluable and sustained contributions to the investigation and containment of cholera, and Mariëtte Muller for her meticulous maintenance of the cholera database. The Director-General of the Department of National Health and Population Development is thanked for permission to publish.

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