THE CLINICAL PRESENTATION OF POLIOMYELITIS IN THE YOUNG BANTU CHILD

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In 1951 it was reported that among urban Bantu children silent infection with the virus of poliomyelitis took place early in life, but the incidence of paralytic poliomyelitis was relatively low.[1] However, in 1956, in the largest epidemic of clinical poliomyelitis yet seen in the Johannesburg Bantu, 169 cases of acute anterior poliomyelitis were seen in the Paediatric Unit, Baragwanath Hospital, Johannesburg, in the first half of the year.

Early diagnosis of this disease to prevent admissions to the general wards was considered important, and this paper gives an account of the clinical presentation and the difficulties encountered in diagnosis.

Material

The Paediatric Unit at Baragwanath Hospital serves non-European children under 9 years of age. It is a large unit where 5,839 and 5,996 in-patients, and 73,000 and 88,000 out-patients were seen in 1955 and 1956 respectively. There are no isolation facilities for patients suffering from infectious disease.

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Between January and July 1956 119 cases of poliomyelitis were diagnosed at Baragwanath Hospital, but the records of only 99 could be traced, and these patients are studied in this paper. Of these, 18 were in-patients and the rest were out-patients. In addition, 50 patients with poliomyelitis, diagnosed elsewhere, were referred for physiotherapy, after the acute stage of the disease.

Definitions

The following terms used in this paper are briefly defined:

Non-paralytic poliomyelitis: Evidence of involvement of the central nervous system, with positive cerebrospinal-fluid findings, but without paralysis; with or without recovery of polio virus from the stools. (This was attempted in 3 patients; 2 were positive.)

Encephalitic form of poliomyelitis: Evidence of poliomyelitis, but encephalitic symptoms (coma, stupor or convulsions) dominated the clinical picture.

Bulbar form of poliomyelitis: Cranial-nerve involvement without clouding of consciousness, and with or without involvement of the spinal cord.

Spinal form of poliomyelitis: Paresis or paralysis attributable to involvement of the cervical, thoracic or lumbo-sacral cord.

The spinal cases are further described after the classification of Smith, Harris and Rosenblatt:[2]

Group I	Mild	Minimal isolated muscle weaknesses.
Group II	Moderate	Includes weakness of 2 limbs.
Group III	Moderately severe	Includes flaccid paralysis of one limb with or without isolated muscle weakness elsewhere.
Group IV	Severe	Includes flaccid paralysis of 2 limbs with or without isolated muscle weakness elsewhere.
Group V	Very severe	Includes flaccid paralysis of 3 limbs or respiratory muscle paralysis (diaphragms, intercostals, abdominals).

Presentation

67% of the patients presented with paralysis already present (Table I), although this had not always been recognised as

TABLE I. PRESENTING SIGNS AND SYMPTOMS

Paralysis	66
Fever	11
Cough and fever	7
Pain	5
'Twisted' neck	2
Convulsions	2
Sores in mouth	2
Diarrhoea	1
Nystagmus	1
Swollen fontanelle	1
Listlessness	1
Total	99

such by the mother, who might say, 'Baby won't crawl', or 'Baby has a crooked face'. Only about 1/5th gave a history of preceding feverish illness.

Sex and Age

There was the usual preponderance of boys (Table II),

TABLE II. TABLE SHOWING SEX INCIDENCE

Males	52
Females	36
Sex not recorded	11
Total	99

which has been noticed in most epidemics. The highest age incidence was between 6 and 18 months (36 cases); 3/4ths of the cases were less than 3 years old (Table III).

TABLE III. TABLE SHOWING AGE INCIDENCE

0 to 5 months	2
6 months to 11 months	15
1 year to 17 months	21
18 months to 2 years	13
Over 2 years to 3 years	19
Over 3 years to 4 years	11
Over 4 years to 5 years	6
Over 5 years to 6 years	0
Over 6 years to 7 years	3
Over 7 years to 8 years	2
Age unknown	7
Total	99

Type and Severity

More than 3/4ths of the patients showed the spinal paralytic form of the disease (Table IV); 9% were bulbar, 3% were encephalitic, and only 9% of cases showed the non-paralytic form. When the spinal paralytic cases were grouped according to severity, it was noted that only 31% fell into the mild or moderate category, while 63% were moderately severe, severe or very severe (Table V).

Cerebrospinal Fluid

Lumbar puncture, which was usually an out-patient procedure, was found essential for diagnosis in nearly all cases under 2 years of age. The cerebrospinal fluid findings in 64 of the patients are summarized below.

Of the patients with typical paralytic poliomyelitis, 5 (7·8%) gave normal cerebrospinal fluids. In the other patients the total cell count varied from no cells to 225 cells per c.mm. In half the patients it was less than 60 cells per c.mm. The cells were chiefly lymphocytes, the highest count being 189 per c.mm. In 1/3rd of cases polymorphs were less than 5 per c.mm., and in only 16 patients were there more polymorphs than lymphocytes. Of these, 7 had polymorph counts varying from 79 to 138 per c.mm. (3 were bulbar, 1 polio-encephalitic, and 3 non-paralytic in type). Where the polymorph count was high, only one patient showed a protein value of over 40 mg.%. Where the total cell count was high, the protein was usually, but not invariably, low. Half the patients lumbar punctured showed a protein value below 31 mg.%, and the highest value was 116 mg.%.

TABLE IV. CASES OF POLIOMYELITIS ACCORDING TO AGE AND FORM OF POLIOMYELITIS

Forms of Poliomyelitis	*Unknown age*	*0-5 months*	*6-11 months*	*12-17 months*	*18 mths-2 years*	*2-3 years*	*3-4 years*	*4-5 years*	*5-6 years*	*6-7 years*	*7-8 years*	*Totals*	*% Totals*
Non-paralytic	0	0	4	2	0	0	1	1	0	1	0	9	9·1
Encephalitic	0	0	1	0	1	0	1	0	0	0	0	3	3·0
Bulbar	0	0	3	1	0	1	3	0	0	0	1	9	9·1
Spinal-paralytic	7	2	7	18	12	18	6	5	0	2	1	78	78·8

TABLE V. SPINAL CASES ACCORDING TO AGE AND SEVERITY

	No. of Cases	*% Spinal Cases*	*0-5 months*	*6-11 months*	*12-17 months*	*18 mths-2 years*	*2-3 years*	*3-4 years*	*4-5 years*	*5-6 years*	*6-7 years*	*7-8 years*	*Unknown age*
Spinal Cases	78	100·0	2	7	18	12	18	6	5	0	2	1	7
I. Mild	20	25·6	0	1	5	3	6	1	2	0	2	0	0
II. Moderate	4	5·1	1	0	0	1	0	0	0	0	0	1	1
III. Moderately severe	23	29·5	0	4	6	4	5	2	1	0	0	0	1
IV. Severe	18	23·1	0	1	6	3	5	1	1	0	0	0	1
V. Very severe	8	10·3	1	1	1	1	1	2	1	0	0	0	0
Unclassified	5	6·4	0	0	0	0	1	0	0	0	0	0	4

COURSE OF THE DISEASE

Any complete study is notoriously difficult with Bantu patients. Of the 99 patients, 5 died (2 encephalitis, 1 bulbospinal and 2 very severe spinal), 9 were not paralysed nor suffered any disability, and 72 other patients were seen after their discharge from hospital (there was no follow-up at all in 13 cases).

Of 31 patients who were seen 1 month after the onset of their disease, 2 were cured (a mild spinal case and a combined bulbar and mild spinal case), 3 bulbar cases showed slight improvement, and 2 other bulbospinal cases showed improvement of the bulbar palsies only. The remainder were 24 spinal cases who were unchanged.

Of 19 children who were followed up for 2 months, 1 bulbar and 13 spinal cases were unchanged, 2 very severe cases had improved and could now be classified as severe, 2 moderately severe had improved to mild, and 1 initially mild spinal case showed improvement.

Of 22 children followed up for times varying from 3 to 6 months, 18 showed no change, 1 moderately severe spinal case had improved to mild, 2 mild spinal cases showed very considerable improvement though weakness could still be detected, and 1 patient who had been classified as encephalitic and spinal now showed no paralysis but still had very slight nystagmus.

Differential diagnosis illustrated by clinical case notes

As McMath *et al.*[3] point out, poliomyelitis may be difficult or even impossible to diagnose in the *pre-paralytic stage.* Examination at this time may reveal equivocal signs of meningeal irritation with doubtful Kernig and Brudzinski signs and often very slight neck rigidity. They consider the 'tripod' and 'kiss the knees' signs, and the presence of pyrexia accompanied by retention of urine, as valuable diagnostic evidence. However, in children of 3 years and less, as in most of the Baragwanath group, the patient's cooperation is seldom secured sufficiently to elicit these signs. Cases 1-9 were all admitted with diagnoses referable to meningeal irritation, and all gave abnormal cerebrospinal fluids.

Case 1, a male aged 8 months, *case* 2, a male aged 1 year and 3 months, *case* 3, a female aged 1 year and 4 months and *case* 4, a male aged 9 months, all had neck stiffness and high fever and were admitted as cases of *meningitis.* Case 2 became paralysed next day, and later developed respiratory involvement, whilst case 1 was found on admission to have mild spinal paralysis and a bulbar palsy. Cases 3 and 4 did not develop paralysis.

Cases 5 and 6 were less acutely ill and were admitted as probable cases of *tuberculous meningitis.* Case 5, a male aged 2 years, presented with a 'sore neck' and had neck stiffness for 2 days in hospital before developing a weakness of the left leg, when it was learnt that a sibling had been admitted to another hospital with poliomyelitis. Case 6, a male aged 4 years, was so uncooperative and apparently hysterical for some days in the ward that he could not be properly examined, and his mild paralysis was not immediately discovered.

Case 7, a male aged 1 year and 4 months, with high fever, was admitted as a possible *encephalitis or tuberculous meningitis* because he was thought in the out-patients department to have an upper-motor-neurone weakness of the facial nerve. After observation in the ward it became clearly lower-motor-neurone in type, and the cerebrospinal fluid showed 119 polymorphonuclear leucocytes and 47 lymphocytes per c.mm., with normal chemical findings.

Case 8, a male aged 1 year and 3 months, was admitted as an *encephalitis,* with neck stiffness and increased cells and protein in the cerebrospinal fluid but without paralysis.

Case 9, a female aged 1 year and 3 months, was admitted as *neck stiffness* for further investigation. On admission she was found to have generalized weakness, and was still very severely paralysed 3 months later

The pre-paralytic stage has been shown to last longer in young infants; the average interval between the date of onset of illness and the first detection of paralysis was 5·5 days among infants under 1 year of age as contrasted with 3·5 days among children and adults.[4] In 5 cases (children) in which the diagnosis is known to have been missed at Baragwanath out-patients, and who later were referred by fever hospitals, the diagnoses on their record cards (just a few days before paralysis set in) varied from pharyngitis to gastro-enteritis. Cases 10 and 11 are examples of pyrexial children in the pre-paralytic stage of poliomyelitis who escaped diagnosis and were admitted.

Case 10, a female aged 3 years and 10 months, had a history of fever and pain in the chest and X-ray evidence of right middle-lobe consolidation. She was admitted with the diagnosis of *broncho-pneumonia,* and next day there was pain in the back with muscle spasm. The cerebrospinal fluid showed 84 polymorphs and 10 lymphocytes per c.mm. and a protein level of 82 mg.%. She was referred to the fever hospital, where a mild paralysis ensued. Weakness of the right shoulder-girdle muscles was still present 2 months later.

Case 11, a female aged 4 years, had *abdominal pain for investigation* as her admission diagnosis, and was considered a case of appendicitis by the referring doctor. She had vomited with her severe abdominal pain 3 days before. On admission she was found to have tenderness and pain in limb muscles as well as abdomen and she developed paralysis.

The painful muscles in Case 11 should have indicated the probable diagnosis, for the paralytic phase of poliomyelitis is commonly heralded by tenderness, stretch pain and fibrillary contractions in muscles, with increase of the deep reflexes.[3] The fibrillary contractions in very young patients are usually masked by subcutaneous fat, but the other signs and symptoms sometimes give rise to great difficulty in diagnosis. The increased reflexes caused case 12 to be admitted as an apparent left hemiparesis, and it was only a few days later that poliomyelitis was suspected.

Case 12, a male aged 1 year and 4 months, was admitted with the diagnosis of *hemiparesis with encephalitis.* He had been unable to walk for some days and although he would not use his left arm or stand on his left leg, the jerks were brisker on that side. Two days after admission he developed some doubtful weakness of the right arm, and on the 4th day his left knee and ankle jerks became diminished for the first time. On his 9th hospital day all his reflexes were present and apparently equal, but there was a mild left quadriceps weakness. His cerebrospinal fluid on admission showed 5 polymorphs and 49 lymphocytes per c.mm. with a protein of 77 mg. A week later the cell count was normal, but the protein was still 56 mg. Type-I polio virus was grown from the stool of this patient. He was apyrexial throughout his stay in hospital.

On account of the presence of all reflexes on admission, the following case was not suspected of spinal poliomyelitis.

Case 13, a male aged 1 year and 8 months, with a history of mild diarrhoea and convulsions for 2 days, was admitted in *coma* with marked trismus. Muscle tone was poor and there was no neck rigidity. All reflexes were present and equal. His cerebrospinal fluid contained 130 polymorphs and 95 lymphocytes per c.mm. and was normal biochemically. He died 18 hours after admission, and at post-mortem was found to have changes typical of polio-encephalitis, and poliomyelitis in the anterior horn cells of the cord.

Case 13 is an example of how some cases of polio-encephalitis rapidly become comatose before paralysis can be diagnosed. Such widespread polio-encephalitis is almost invariably fatal.[5] The picture of coma, trismus and convulsions was seen in another in-patient at the time of the poliomyelitis epidemic, and the diagnosis of polio-encephalitis was mistakenly made, for neither laboratory nor post-morten evidence of the disease was found. Both these cases have been fully described elsewhere in a discussion on trismus.[6] This is an interesting and well described sign occurring in poliomyelitis, and attributed to mid-pontine tegmentum involvement at the site of the motor nucleus of the V cranial nerve.

In the paralytic phase of the disease, when *lower motor neurone paresis* develops, diagnosis becomes much easier, but that confusion still arises when the patient is very young is shown by the admission to Baragwanath Hospital of several infants with paralysis. Post-diphtheritic polyneuritis is not uncommon at Baragwanath Hospital and it is easy to understand, therefore, how this diagnosis may be made on an extensively paralysed patient, when seen in the early stages of an epidemic.

Case 14, a female aged 3 years, was admitted as a *polyneuritis* on account of generalized weakness. Next day it was appreciated that in the upper limbs the paralysis was asymmetrical, and the cerebrospinal fluid contained 38 polymorphs and 140 lymphocytes per c.mm. Later Type-I polio virus was grown from her stool.

The amyotonia congenita syndrome due to infantile spinal muscular atrophy may give rise to confusion in the small infant; both conditions result from pathology in the anterior horn cells.

In case 15, a female aged 2 months, 2 convulsions had occurred the night before admission and there was a sudden onset of 'limpness'. The baby was admitted with the diagnosis of possible *amyotonia congenita* or *polyneuritis*. All limbs appeared weak and flaccid, except the right arm, where some muscle tone was present. The right biceps and triceps jerks were the only reflexes that could be elicited. There was some paralysis of intercostal muscles and breathing was chiefly diaphragmatic. The cerebrospinal fluid contained 5 polymorphs and 19 lymphocytes per c.mm. and the protein was raised to 71 mg.%. A month later the patient still had a flaccid paralysis of the left arm, right leg and both glutei.

In the differentiation of poliomyelitis from amyotonia congenita, the history may help, fever and a sudden onset favouring the diagnosis of poliomyelitis. The paralysis is sometimes bulbar and may involve the muscles of respiration in both conditions, but is symmetrical in distribution in amyotonia congenita. Asymmetry in an extensively paralysed small baby may only be demonstrable after careful and patient examination. The cerebrospinal fluid in case 15 was characteristic of poliomyelitis; in amyotonia congenita the cerebrospinal fluid is normal. Final proof is offered by the virus laboratory.

Cases 13 and 15 showed convulsions, but these occur so rarely in poliomyelitis that, in spite of a flaccid left arm in a 5-week-old infant (case 16), the admitting officer who saw him in a convulsion felt that the diagnosis of poliomyelitis was so unlikely that he admitted the patient to the general wards.

Case 16, a male aged 5 weeks, was admitted with the diagnosis *monoparesis for investigation*. His left arm had been noticed to be paralysed for 1 day, and he had been feverish for 2 days. A generalized convulsion occurred just before admission. There was a flaccid paresis of the left arm. Subdural taps were negative and the cerebrospinal fluid contained 29 polymorphs and 48 lymphocytes per c.mm. and 71 mg. of protein per 100 ml. Twelve hours later the right leg was thought to be moving less briskly than the left and the patient was transferred. When seen a month later the arm was still paralysed and some weakness of both legs was present.

In New York in 1949 and 1950 convulsions occurred in only one infant among 92 under 1 year with poliomyelitis.[4] In the two very young paralytic Baragwanath cases, cases 15 and 16, the convulsions may be attributed to 'febrile convulsions', but in case 13 the convulsions were almost certainly due to the encephalitis, and it is known that the encephalitic form of the disease sometimes causes convulsions even in adults.[7]

Only about 1/5th of the paralysed patients gave a clear history of a minor illness, and although it has been said that in about 40% of paralytic cases a minor illness can be distinguished,[5] it must be remembered that the patients were for the most part under 3 years of age, and more than half of these below 18 months, and also that history-taking in a busy out-patient department among a multi-language group is often rather brief. There were some extremely observant mothers who noticed a slight weakness some days before the medical officer was sufficiently convinced to subject the child to lumbar puncture.

In each of these several cases the cerebrospinal fluid showed changes typical of poliomyelitis.

In the diagnosis of paralytic poliomyelitis, pseudo-paralysis may give rise to confusion, and the pseudo-paralysis associated with congenital syphilis is seen not uncommonly at Baragwanath Hospital. The pseudo-paralysis of scurvy, however, is not seen, as scurvy is exceedingly rare in Bantu children, although a common disease in adults.[8] The pseudo-paralysis caused by osteomyelitis was mistakenly diagnosed as poliomyelitis at Baragwanath Paediatric Out-Patients during this epidemic. Some of the out-patients were of special diagnostic interest.

The mother of *case* 17, a male aged 9 months, noticed 'funny movements of the eyes' for 1 day. On examination remarkable coarse nystagmoid movements of the eyes were present, and there was a doubtful weakness of the sternomastoids. The child was cheerful and did not look ill. There were 11 polymorphonuclear leucocytes and 105 lumphocytes per c.mm. in the cerebrospinal fluid. The chemistry was not known until the following day, and the baby was nearly admitted to the general wards with a diagnosis of tuberculous meningitis. However, as this disease rarely occurs under 1 year at Baragwanath Hospital without radiological evidence of gross chest tuberculosis, an out-patient chest X-ray was taken. This appeared completely normal and the child was sent to the Fever Hospital with a diagnosis of poliomyelitis, which proved to be correct. He later developed a lower-motor-neurone paralysis of his left leg. This recovered, but some months later very slight eye movements could still be detected.

These coarse movements of the eyeballs on attempted fixation, known as opsoclonia and differing from true nystagmus, which however is also seen, have been described by several observers.[5, 9] The studies of Baker and Cornwall[10] and Matzke and Baker[11] have shown that the cerebellum and midbrain are frequently implicated in the pathology of poliomyelitis, even although clinical manifestations of such involvement are uncommon.

Two children made their first visit to out-patients with *sores in the mouth* and both were diagnosed as herpetic stomatitis. One became mildly paralysed 4 days later and still had weakness a month later, and one became moderately severely paralysed 9 days later, but then developed involve-

ment of the respiratory musculature in the Fever Hospital and died after a week. It is interesting to speculate whether these children did in fact have herpangina due to the Coxsackie-A virus, which virus is often isolated in proven cases of poliomyelitis.[12]

DISCUSSION

It has recently been suggested that acute poliomyelitis is not infectious and patients can safely be admitted to a general ward.[13] Anyone who has personally seen cross-infection occur would disagree. In 1949 several long-term cases developed paralytic poliomyelitis after the admission of 2 cases of poliomyelitis to their cubicled infants' ward. These patients had diseases such as leukaemia, Wilms's tumour, hydrocephalus.[14] Horstmann *et al.* have said that 'on the basis of virus isolations and serological evidence, poliomyelitis infection may be said to be as contagious as measles among susceptible individuals in a family setting'.[15] Despite great care and the use of diagnostic lumbar puncture in the outpatients department, 18 patients were admitted to Baragwanath Hospital. Of these, 16 have been discussed; the remaining 2 patients were known to have poliomyelitis, but one, a fatal case, had to be admitted as he was *in extremis* and too ill to send the long journey (20 miles) to the nearest fever hospital, and the other was admitted owing to incorrect information about the duration of the disease. No case of cross-infection was noted as a result, but the turn-over of patients in Baragwanath Hospital is extremely rapid.

There was a preponderence of very young children amongst the poliomyelitis cases, and this often made diagnosis difficult. Elsewhere it is generally agreed that poliomyelitis is showing an increasing incidence in the older age-groups. Smith *et al.*,[2] who show an increasing incidence of the non-paralytic form of the disease with age, suggested that the more frequent recognition of these forms is the cause of the apparent increasing incidence in older age-groups. However, an increasing incidence with age has also been found among paralytic forms of poliomyelitis.[16]

In New York, among 92 cases in children less than 1 year old, non-paralytic forms were infrequent, totalling only 9·8%, compared with 44·7% in children aged 5-15 years.[4] In London the percentage of paralytic cases under 1 year was higher than that for any other group.[4] At Baragwanath Hospital, on the other hand, of the 17 babies under 1 year with poliomyelitis, 4 (23·52%) showed no paralysis; only 9 out of the 99 children with poliomyelitis were non-paralytic cases, and 6 of these 9 were under 18 months. In view of their recognition at an early age, it seems unlikely that non-paralytic forms in the older age-groups were missed. Presumably when the Bantu also show a trend towards older age-incidence, the percentage and age of the non-paralytic forms will increase.

Medalie[13] has shown that the age incidence in patients in Boksburg-Benoni Hospital in 1956 was lower among the non-Europeans than the Europeans. This may be due to economic and hygienic factors rather than racial factors. In America, Negroes appear to suffer from this disease as frequently as Whites.[17, 2] The shift in incidence of poliomyelitis to the older age-groups appears greater in the families of higher economic status in America. In these it is seldom seen at all under one year.[18]

The percentage of paralytic poliomyelitis was higher in the Baragwanath series than that reported in most recent epidemics elsewhere, and bulbar forms were fewer.[9, 2, 4, 19-21] Encephalitic forms of poliomyelitis varied in New York in incidence from 2% to 8%.[2, 17] At Baragwanath 3% of the cases were encephalitic.

The severity of the cases was striking. Smith *et al.*[2] found only 35% of their spinal cases fell into the moderately severe, severe and very severe groups, whereas 63% of the Baragwanath cases were in these categories.

The cerebrospinal fluids of the Baragwanath cases did not show any unusual features and the findings, including the occurrence of normal fluids, are similar to those described in most epidemics.[2, 22]

The follow-up of these patients is quite inadequate because, although most authorities agree that the maximum degree of recovery in muscle function in poliomyelitis occurs in the first 6 months, recovery often continues for a couple of years. The most severely afflicted can expect the greatest degree of permanent weakness, and on this basis the young Bantu cases described can be expected to give rise to a considerable number of cripples. The need for early diagnosis, difficult though it may be in this age-group, and for immediate isolation of patients, is stressed.

SUMMARY

In the largest epidemic of poliomyelitis yet seen in the Johannesburg Bantu, which occurred in 1956, 169 cases of acute anterior poliomyelitis were seen in the Paediatric Unit, Baragwanath Hospital, Johannesburg, in the first half of the year.

The clinical presentation, sex, age, form of disease and severity of 99 cases are described and discussed.

The preponderance of very young cases is discussed. They presented special diagnostic problems and made diagnosis difficult; 16 children who initially escaped diagnosis and were admitted to the general wards are discussed.

Non-paralytic forms of the disease were relatively few, and mostly under 18 months. Bulbar cases were relatively few. The severity of the cases was striking.

Some data on the immediately post-isolation progress of the cases is presented.

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