# POLIOMYELITIS IN NAIROBI 

J. R. Harries, M.D., M.R.C.P., D.C.H., D.T.M. \& H.

Physician, and

W. E. Lawes, D.A., F.F.A.R.C.S., D.T.M. \& H.<br>Anaesthetist, Respiratory Unit, King George VI Hospital, Nairobi

In November 1956 there was a sharp increase in the number of admissions of poliomyelitis to the Respiratory Unit, Nairobi. It soon became obvious that a serious epidemic was developing.
We thought that it might be of interest to readers to present an account of the racial incidence and types of cases, methods of treatment, differential diagnosis, and the causes of death. The unit accepted cases from Nairobi and from all parts of East Africa. The cases admitted from other hospitals were primarily life-threatening. Many were transported by air, a flying team being sent out from the unit for this purpose.
The unit possesses 4 Radcliffe and one Engström intermittent positive-pressure respiration (IPPR) pumps, 4 tanks, one Spiroshell cuirass respirator and 2 rocking beds. During the epidemic one Beaver IPPR pump and 7 additional tanks were borrowed. At the height of the epidemic 5 IPPR machines, 6 tanks and 2 oxygen tents were in use at the same time.

## Cases Admitted

224 cases were admitted to the unit during the period November 1956 to September 1957. Only one of these cases was non-paralytic. Of this total 52 were Europeans, 18 Asians and 154 Africans.

## Age Groups

The age and sex incidence of each racial group are presented in Tables I and II. As can be seen there was a considerable difference between the age distribution in the different races. In the African group, 146 of the 154 admissions ( $94 \cdot 8 \%$ ) were under the age of 5 years; of these 146 cases 115 were under 2 years of age. In the European group 28 of the 52 admissions ( $53.8 \%$ ) were over 20 years of age. All but one of the 18 Asians admitted were under 15 years of age.

## Types of Cases

Among the 154 Africans, 39 could be classified as lifethreatening; the respective figures for Europeans and Asians were 19 out of 52 and 6 out of 18 .

For the purpose of this paper we have used the following definitions of life-threatening poliomyelitis:
'Bulbar paralysis': pharyngeal paralysis with inability to swallow.
'Bulbo-spinal paralysis': pharyngeal paralysis plus involvement of the muscles of respiration.
'Spinal paralysis': paralysis of intercostals and/or diaphragm.

## OUTSTANDING FEATURES

## African Cases

Half of the Africans had severe diarrhoea on admission and some were considerably dehydrated. Many were anaemic; one had a haemoglobin level of $35 \%$ (Sahli) in spite of profound dehydration.
A distinctive feature among the African cases which was not seen in the other groups was the high proportion of 'wet lungs' ( 20 out of the 39 life-threatening cases). This condition was mainly due to paralysis of the abdominal muscles with or without diaphragmatic or intercostal paralysis, leading to an inability to cough. Measles or an upper-respiratory-tract infection superimposed upon this condition, or nursing in the supine position, soon led to a state of 'wet lung' with generalized rales and, in some cases, consolidation and/or atelectasis. Some of these cases gave a history of a febrile illness with paralysis of the limbs some weeks before and had been admitted to a general hospital as cases of bronchitis or bronchopneumonia before being sent to us.

## Asian Cases

Of the 6 'life-threatening' cases, 2 showed an encephalitis as the predominant feature; one other had bulbo-spinal poliomyelitis and the remaining 3 had involvement of the muscles of respiration. An Asian youth of 15 years who showed a bulbar paralysis with polio-encephalitis appeared to have paralysis of the respiratory centre. He was unable to swallow and had had a tracheotomy performed before admission to the unit. During transit over a distance of 100 miles he had stopped breathing and had needed artificial respiration. On admission he began to breathe on his own. This spontaneous respiration lasted for some 20 hours, during which time he maintained a normal oxygenation of arterial blood. There was no evidence of paralysis of his intercostals, diaphragm or arms and legs. With no warning of respiratory failure he again stopped breathing and required IPPR treatment. Arterial blood analysis taken within a few minutes of this acute respiratory failure showed a pH 7.31 and $\mathrm{Pa}^{*} \mathrm{CO}_{2} 60 \mathrm{~mm} . \mathrm{Hg}$, but the respiratory centre did not appear to respond to this degree of respiratory acidosis.

* For this symbol for arterial $\mathrm{CO}_{2}$ tension see Federal Proceedings (1950): 9, 602.

It might be argued that this respiratory acidosis was primarily due to paralysis of respiratory muscles but the respiratory failure was of extremely abrupt onset and there was no evidence of respiratory obstruction.

## European Cases

In this group there was a high incidence of bulbar and bulbo-spinal involvement ( 12 out of the 19 life-threatening cases).

The total admissions included 14 married European women, of whom 6 were pregnant. One miscarried at 3 months, one had a stillbirth at 36 weeks and 3 live babies were delivered at term, one by Caesarean section. The remaining patient is 6 months pregnant and has been an in-patient for 3 months. The Caesarean baby was delivered during the period of infectivity and the virus was isolated from the meconium and cord blood. The virus isolations were done by Dr. J. Gear in Johannesburg and both proved to be Type I.

Of the 10 adult European males admitted, 8 showed respiratory involvement, 6 requiring respiratory aids.

## TREATMENT

Statistical details of treatment are shown in Table III (A, B, C and D). Further particulars are given below:

## 1. Bulbar Paralysis

These cases were treated by posture (i.e. in the face-down position and by raising the foot of the bed), pharyngeal suction and tube or parenteral feeding. Seven cases were so treated with no deaths.

## 2. Bulbo-spinal Paralysis

Seven severe adult cases were treated with tracheotomy and IPPR machines in the manner described previously by many writers. ${ }^{6,10,9,4}$ Of these, 5 were first nursed in tank respirators until bulbar paralysis developed; tracheotomy was then performed and the patient ventilated by IPPR. Two severe cases in children both under the age of 2 years were treated in the face-down position without tracheotomy, in a tank specifically designed to allow the use of this position. There were 2 deaths in this group of 9 , both being adults with severe bulbo-spinal encephalitis.

Two less severe cases in children under the age of 5 were successfully treated with posture, tube feeding and suction, and were nursed in oxygen tents. One adult female was successfully treated with posture, tube feeding and suction.

## 3. Spinal paralysis, i.e. paralysis of intercostals and/or diaphragm

Of the 17 cases who needed respiratory aids, 16 were treated in tanks and one by IPPR as he was admitted with total atelectasis of one lung which it was not possible to clear in a tank. Adult cases were put into tank respirators when their vital capacities fell to $30 \%$ of the expected normal. Of the 16 treated in tanks, 5 died (all infants under 2 years of age). The moment to use tank respirators in infants was judged by a falling or increasing respiratory rate and pulse rate, and any other indications of respiratory distress such as the use of accessory muscles and alae nasi.

Of the cases who did not need respiratory aids, 12 were treated with posture and suction and nursed in oxygen
tents, and 11 others were treated by posture and suction alone.
Four infants whose predominant paralysis was of the abdominal muscles were treated by abdominal binders, posture and oxygen tents.

All cases with 'wet lungs' were given antibiotics.

## Hypothermia

Seven cases were treated by hypothermia of varying degree. This form of treatment was only used in cases showing hyperpyrexia (temperature of over $104^{\circ}$ ) where previous experience has shown that a high mortality rate could be expected. ${ }^{2}$ Lassen ${ }^{7}$ found a mortality rate of over $90 \%$ in these cases.
There were 2 deaths in the 7 cases so treated.

## Cross Infection

Considerable inconvenience was occasioned by cross infection. A Staphylococcus aureus phage type 80 was isolated among other bacteria from tracheotomy wounds, bronchial secretions, and infected urines. Four members of the staff developed acute septic conditions of the hands; 3 were of a serious nature. Again a $S$. aureus phage type 80 (penicillin resistant) was isolated from each case.
differential diagnosis, etc.
Among the cases admitted as suspected poliomyelitis were the following:

Landry's paralysis in 2 cases showing bulbar spinal paralysis; these were successfully treated with cortisone; one case required respiratory aid. Herpes zoster in a middleaged European with an abnormal CSF; the predominant features were severe pain in one leg with paralysis followed by the typical herpetic eruption. Three cases of benign lymphocytic chorio-meningitis. A case of post-mumps encephalitis in a European adult. Two cases of pertussis encephalitis in African infants. Two hemiplegics, both being young African males. Two African infants showing osteomyelitis of the femur. A European adult female with a Coxsackie infection, who showed severe pain of the intercostals and diaphragm which embarrassed respiration. A fatal case of pneumococcal meningitis with a normal CSF in a European schoolboy. A European adult with cerebral malaria. An African adult with transverse myelitis. Two European adult females with hysteria.

## C.S.F. Findings

Some of the CSF findings did not fit into the so-called classical picture. The cell count ranged from 5 to over 1,000 . In cases showing these extremes of the cell count poliomyelitis was not diagnosed unless typical paralysis was evident.

## Causes of Death

Out of the total of 223 paralytic cases there were 64 with life-threatening disease. This high incidence was occasioned by the selective nature of our admissions; the great majority of cases from hospitals outside of Nairobi were of a serious nature. Of the 64, 11 died, 8 deaths occurring in the African group. Of these 7 showed 'wet lungs' on admission.

Post-mortem examinations were performed in 10 cases, in 7 of which there was obstruction to respiration in the lower respiratory tract due to muco-pus in 6 cases and to inhaled milk in the 7th. In one other case the predominant

TABEE I. TOTAL ADMISSIONS* BY RACE, SEX AND AGE


TABLE II. LIFE-THREATENING CASES AND DEATHS, ETC. BY RACE AND AGE

|  | 0-5 years |  |  | 5-20 years |  |  | 20-50 years |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Total | $\begin{aligned} & A f . \\ & 146 \end{aligned}$ | $\begin{gathered} \text { As. } \\ 13 \end{gathered}$ | Eur. | $\overline{A f}$ | $\begin{gathered} \text { As. } \\ 5 . \end{gathered}$ | $\begin{gathered} \text { Eur. } \\ 14 \end{gathered}$ | $A f .$ | As. 0 | $\begin{gathered} \text { Eur } \\ 28 \end{gathered}$ |
| Resp. cases * | 36 | 4 | 1 | 3 | 2 | 1 | 0 |  | 17 |
| Resp. cases as \% of total | 24.6\% | 30.7\% | 10\% | 50\% | 40\% | 7.7\% | 0 |  | 60.7\% |
| Deaths .. . |  | 1. | 0 | 1. | 0 | 0 | 0 |  | 2 |
| Mortality rate | 19.5\% | 25\% | 0 | 33.3\% | 0 | 0 | 0 |  | $11.7 \%$ |
| Age group as \% of all ages | 94.8\% | $72 \cdot 2 \%$ | 19.2\% | 4\% | 27.8\% | 27\% | 1.2\% |  | $53.8 \%$ |

* Life-threatening poliomyelitis

TABLE III A. AFRICAN CASES AND DEATHS BY CLASSIFICATION AND TREATMENT AND BY AGE


TABLE III B. EUROPEAN CASES AND DEATHS BY CLASSIFICATION AND TREATMENT AND BY AGE


TABLE III C. ASIAN CASES AND DEATHS BY CLASSIFICATION AND TREATMENT AND BY AGE


TABLE III D. ALl CASES AND DEATHS BY Classification and treatment and by age

$P . E .=$ Polioencephalitis. $B .=$ Bulbar paralysis. $B . S .=$ Bulbo-spinal paralysis. $A b d .=$ Paralysis of abdominal muscles, without intercostals or diaphragm. $S p .=$ Paralysis of intercostals and/or diaphragm with or without abdominal muscles. Posture etc. $=$ Prone position, elevation of foot of bed, pharyngeal suction. Posture $+0_{2}=$ Above plus oxygen tents. Deaths in brackets.
findings were in the brain, no other obvious cause of death being found. One case, who died within 24 hours of the cessation of induced 'mild hypothermia', ${ }^{1}$ showed a massive thrombosis in the right auricle extending through the tricuspid valve into the right ventricle and from there into the pulmonary artery.
Another case died during induced 'moderate hypothermia' ${ }^{1}$ and showed changes not dissimilar to those described in experimental animals by Knocker (1955) and Grey (1957). ${ }^{3}$
Of the 8 deaths in African children, 4 occurred while the children were in tanks and 4 in cases treated by posture, suction, antibiotics, and oxygen tents. One of these latter cases had a tracheotomy.

## Respiratory Obstruction by Secretions

The most serious difficulty in the treatment of life-threatening poliomyelitis in African infants during this epidemic was not the poliomyelitis per se but the lower-respiratorytract obstruction caused by the accumulation of secretions. In 3 infants who died the main respiratory muscles involved were the abdominals, whilst involvement of the intercostal muscles or diaphragm was minimal. In many of these cases it was difficult to decide whether or not a respiratory aid was necessary. All had raised respiratory rates and some were slightly cyanosed. Tracheotomy was performed on one of these infants to facilitate suction; this was one of the 3 infants who died.
In these cases of 'wet lung' where evidence of respiratory paralysis was insufficient to warrant the use of a respiratory aid, treatment by posture, abdominal binders to help coughing and nursing in an oxygen tent seemed reasonably effective.

## Improvement in Mortality Rate

In the past 5 years the mortality rate for life-threatening poliomyelitis has declined. Among the factors responsible for this can be listed the introduction of IPPR by Bjorn Ibsen ${ }^{5}$ in 1954.
It has been suggested by Trueta ${ }^{11}$ that tracheotomy should be avoided if possible in acute poliomyelitis, not because the mortality rate is increased, but because weaning is made more difficult. In our experience tracheotomy with IPPR in adults is sometimes a life-saving measure, though this method carries with it its own specific complications and difficulties and requires considerable experience to achieve success. This is possibly shown by figures from our own unit: In our first season we treated 5 adults with IPPR with 3 deaths ( $60 \%$ mortality), whereas this season 8 adults were so treated with 2 deaths ( $25 \%$ mortality). Other factors that may have
contributed have been more detailed investigation of electrolyte and fluid balances and an improvement in hypothermic techniques.
In our first season 3 patients showed hyperpyrexia, and all 3 died. This season 7 patients showed sustained temperatures of over $104^{\circ} \mathrm{F}$; they were all treated with hypothermia and 2 of them died.
Hypertension in acute poliomyelitis may be alarming, e.g. a systolic pressure of over 220 mm . Hg. In many cases this would appear to be central in origin, or at least not due to hypercarbia, for in 3 of our cases who showed high blood pressure readings the arterial $\mathrm{CO}_{2}$ levels were normal.

Lassen ${ }^{8}$ states that the mortality rate is increased in cases showing hypertension. Our 3 cases were treated with subcutaneous Ansolysen, which effectively reduced the blood pressure. All cases survived.

## SUMMARY

A brief description of some of the features of the 1956-57 epidemic of poliomyelitis in Nairobi are presented, particular attention being paid to the age incidence in the various racial groups.

The importance of impairment of coughing due to paralysis of the abdominal muscles is emphasized as a cause of respiratory embarrassment.

The methods of treatment used in the Respiratory Unit, Nairobi, are summarized and a brief analysis of the causes of death is given.

Some reasons for the improvement in present-day mortality rates are put forward. Among these are thought to be the introduction of intermittent positive-pressure respiration, induced hypothermia, detailed attention to electrolyte and water balances, and the use of hypotensive drugs.

## REFERENCES

1. Delorme, E. J. (1956): Proc. Roy. Soc. Med., 49, 368.
2. Engström, C. G. (1954): Brit. Med. J., 2, 666.
3. Gray, T. C. (1957): Lancet, 1, 383.
4. Harries, J. R. and Lawes, W. E. (1955): Brit. Med. J., 1, 454.
5. Ibsen, B. (1954): Proc. Roy. Soc. Med., 47, 72.
6. Lassen, H. C. A. (1953): Lancet, 1, 37.
7. Idem (1954): Proc. Roy. Soc. Med., 47, 70.
8. Idem (1955): Wld. Hith. Org. Monogr. Ser., no. 26.
9. Macrae, J., McKendrick, G. D. W., Sefton, E. M. and Walley, R. V. (1954): Lancet, 2, 21.
10. Smith, A. C., Spalding, J. M. K. and Russell, W. R. (1954): Lancet, 1, 939.
11. Trueta, J. (1956): Handbook on Poliomyelitis, p. 82. Oxford: Blackwell.
