POLIOMYELITIS IN WEST AFRICA.

BY
Brigadier G. M. FINDLAY,
Captain J. R. ANDERSON,
Royal Army Medical Corps,
AND
Captain M. H. K. HAGGIE,
Royal Army Medical Corps.

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Although poliomyelitis is of world-wide distribution it has received comparatively little attention in the tropics, largely, it would seem, because of the absence of epidemics in these regions. In West Africa, for instance, there is no recorded epidemic of poliomyelitis either among Africans or Europeans. Nevertheless the disease is well known to the local inhabitants. Our respected friend Kojo Asare, a medicine man who carries on an extensive practice in the forest region of the Gold Coast, informs us that he has seen numerous cases of acute paralysis in children: certain of these cases die while others partially or wholly recover. He carefully distinguishes between the paralysis of young and of old people (hemiplegia) from which there is no recovery.

The Hausas also recognize the disease for they have a word "Inna," literally "mother," which, according to our informants, is always used with reference to infantile paralysis although Bargery (1934) in his Hausa dictionary translates the word as "hysterical paralysis." In Hausa the word Inna also refers to an evil female spirit, another name for whom is Doguwa, literally the "tall woman." This spirit is commonly believed to give children infantile paralysis, the phrase used being "Inna has drunk his leg" which is exactly similar to the supposed modus operandi of a witch in Ashanti. Incidentally Doguwa or Inna is regarded as having two manifestations, Doguwa na Gida (of the house) and Doguwa na Daji (of the bush). The former is by far the more dangerous and will paralyse the limbs of anyone who displeases her (cf. Tremearne, 1914). She appears as a character in the famous Bori dances where the spirits of disease are thought to possess their devotees. Her song at this dance is "Whomsoever she seizes, he will lose his hand. Whomsoever she seizes, he will lose his feet." It is of interest that many of the Western Fulani people (Taylor, 1931) in the Gold Coast and French Guinea use the word "Inna" for mother. Those, however, who live in Nigeria in Hausa country do not employ and often do not know this word, probably because of its evil connotation (Saunders, 1945).

Clinical observation suggests that cases of residual paralysis involving the legs are not uncommon over a wide area in West Africa and from time to time brief references to paralysis are to be found in the Annual Medical Reports of the British West African Colonies. These refer to the treatment, usually as outpatients, of persons who are regarded as suffering, or having suffered, from poliomyelitis. Only very occasionally is an acute case recorded. Turner (1932), for instance, observed an acute febrile illness with subsequent paralysis in a child in Southern Nigeria, while during the present war we have seen what appeared to be acute cases in children and young persons as far apart as Kano in Northern Nigeria and Winneba on the Gold Coast littoral. In adult Africans the disease would seem to be very rare. McAlpine (1945) and Seddon et al. (1945) have reported its extreme rarity in adult Indians and Maltese.

So far, however, no attempt appears to have been made to isolate the virus of poliomyelitis in British West African Colonies or to determine the incidence of immune bodies to poliomyelitis among the indigenous population. The mobilization of large numbers of African troops, who were often required to serve a thousand miles or more from their country of
origin, and the presence in West Africa of numerous young European soldiers, who had never before been in the tropics, would, it was thought, provide an opportunity for studying the behaviour of poliomyelitis under tropical conditions.

**Poliomyelitis in the Armed Forces in West Africa.**

Unlike Egypt and Malta where during the present war extensive epidemics of poliomyelitis have been recorded among Service personnel and in Malta also among civilian children, no outbreak of poliomyelitis has occurred in West Africa either among military or civilians. Despite the fact that approximately 150,000 West African troops have been at risk the incidence of poliomyelitis has been extraordinarily small; only seven cases having been recorded in the five years from July, 1940 to July, 1945. With the exception of one at Kumasi, Gold Coast, in January, 1945, the other six cases all occurred in 1944: for this year the rate was 0·08 per 1,000 strength.

The distribution was as follows:

- **Abeokuta, Nigeria** ... 2 cases March.
- **Freetown, Sierra Leone** ... 1 case April.
- **Lagos, Nigeria** ... 1 June.
- **Sekondi, Gold Coast** ... 1 September.

The two Africans at Abeokuta belonged to different units but might have bought food in the same "Mammy" market; the oldest patient, a Kroo, was 28 years of age. All these patients recovered; their symptoms were comparatively mild. It is thus obvious that among African soldiers from 18 to 40 years of age poliomyelitis is rare. Among nearly 1,600 West African troops invalided from India and Burma for various complaints there was one case of poliomyelitis.

Among European troops of age-groups similar to those in the Middle East and Malta, though there was also no epidemic of poliomyelitis, the incidence and also the death-rate was higher than among Africans. Among approximately 40,000 British Service personnel at risk in the five years 1940–1945 there were admitted to Army hospitals 11 cases with 4 deaths. Of these 11 persons 8 were officers and 3 other ranks. The incidence of poliomyelitis in the West African Command among British officers and other ranks is shown in Table I, where a comparison is made with figures for India and North African Commands (McAlpine, 1945). Thus, although the figures are fortunately small, there is a greater proportion of officers than men among the victims of poliomyelitis in West Africa as in other Commands: three officers and one British other rank died, a mortality comparable with that among European troops in the India Command in 1943 and 1945.

It is of interest that in the West African Command there are two other diseases which are more frequent among officers than among men—amebic dysentery and infective hepatitis.

The distribution of poliomyelitis cases in Europeans in time was as follows:

- **August, 1942** ... 2 cases
- **November, 1942** ... 1 case
- **March, 1943** ... 1
- **April, 1943** ... 1
- **May, 1943** ... 1
- **August, 1944** ... 1
- **March, 1944** ... 1
- **April, 1944** ... 1
- **May, 1944** ... 1
- **June, 1944** ... 1
- **July, 1944** ... 1
- **August, 1945** ... 1
- **September, 1945** ... 1
- **October, 1945** ... 1
Poliomyelitis in West Africa

With the exception of the two occurring in August, 1942, at Port Loko, Sierra Leone, there was no known relationship between any of the cases: they occurred within a fortnight of each other. The officers lived in different Messes and the survivor of the two stated that he was unacquainted with the other. There were no known civilian cases in the area at the time that these two officers were affected. Other cases occurred in Ibadan and Lagos, Nigeria; Accra, Gold Coast; and Freetown.

It will be seen that the majority of cases among Europeans and Africans occurred in the period March to October, the period of the rains. From November to March the dry Harmattan wind blows from the Sahara: pneumonia and cerebrospinal meningitis become epidemic among Africans.

CLINICAL SYMPTOMS.

The fatal cases in Europeans all ran a very acute course with symptoms of a rapidly ascending involvement of the cord ending in respiratory paralysis or quadriplegia. The biphasic character of the symptoms was much in evidence and in two cases *falciparum* parasites were found in the blood during the preliminary period of fever, which subsided after mepacrine treatment but not because of, anti-malarial drugs.

Since so little is yet known of the virus infections endemic in West Africa the question arose whether these cases were due, not to the poliomyelitis virus, but to an agent of the type of B virus which, normally present in the saliva of monkeys, causes an ascending paralysis in man. No opportunity of isolating the virus responsible for these rapidly fatal cases of ascending myelitis presented itself till July, 1945, when an officer died from a fulminating attack. The following are briefly the clinical and pathological notes of this case, which was very similar to other fatal cases:

Lieutenant X.Y., aged 21 years, had been in the Gold Coast and Nigeria for six months in good health. Immediately before the attack he had travelled from Ibadan to Lagos, Nigeria; thence for three days he had come by road convoy through Dahomey and Togo to Accra where he arrived 9.7.45. For the next two days he felt a little out of sorts and on 11.7.45 he was admitted to hospital with a temperature of 102° F., headache, shivering, pains in the back: the spleen was not enlarged. He confessed that he had not been taking mepacrine 0·1 gramme per day with regularity and rings of *Plasmodium falciparum* were found in his blood.

He was placed on mepacrine treatment (0·8 gramme in the first twenty-four hours) and by the evening of 12.7.45 his temperature was normal and he was symptom free. On the evening of 19.7.45, he complained of slight pain in the neck and left shoulder and also had some frequency of micturition. On the morning of 20.7.45, his temperature was 101·8° F., pulse 126 and he complained of pains in the back and abdomen as well as in the neck and jaw. He had no headache. He vomited twice in the morning but apart from a furred tongue there were no abnormal physical signs. His blood was negative for malaria parasites but as the frequency of micturition continued the mid-stream urine was cultured and yielded *Bact. coli.*

During the day he was alternately shivering and sweating; pain in the neck and shoulder became pronounced; abdominal tenderness was more marked on the right than the left side. The neck and shoulder pains were slight but still present. He vomited again three times during the day but in the evening his temperature was only 100·8° F., with pulse 108. He was treated as a possible case of pyelitis and given sulphathiazole and pot. citrate. On 21.7.45, his condition was unchanged but at 16.00 hours he complained of weakness in the left shoulder with tingling in the left hand; the pain in the abdomen and back was much less. At 21.00 hours there was weakness of the left palatal muscles. He was unable to abduct the left arm while the adductors were weak. Movement of the left forearm and arm was impaired and the left grip was very weak. Biceps and triceps jerks on the left side were only just obtainable, the supinator jerk was normal.

A provisional diagnosis of poliomyelitis was made.

22.7.45: Diplopia now present with severe frontal headache: difficulty of articulation, nasal voice, neck stiff; left side of palate paralysed; tongue tremulous; fine sustained nystagmus on moving eyes to the left; weakness of lumbar muscles and intercostals; swallow...
ing normal; bladder function normal; no feces passed for the last forty-eight hours. Left biceps and triceps jerks still just obtainable but the left supinator jerk was absent.

Leucocytes: Total count 10,600 per c.mm.: polymorphonuclear cells 65 per cent, lymphocytes 24 per cent, mononuclears 9 per cent, eosinophils 1 per cent.

Cerebrospinal fluid: Clear fluid under pressure; 200 cells per c.mm.; polymorphonuclear cells 47 per cent, lymphocytes 53 per cent. Protein 60 mgm. per 100 ml.; sugar normal.

By the evening paralysis had greatly increased so as to involve the muscles of the pharynx; temperature and pulse rising; restless and sleeping only for short periods.

23.7.45: 07.00 hours: respiration suddenly failed with the onset of pale cyanosis; put into Drinker’s apparatus; marked fibrillary twitching of the left biceps. Throughout the day respiration became more difficult and oxygen was given continuously; consciousness was lost only fifteen minutes before death which occurred from circulatory failure and oedema of the lungs at 19.30 hours.

The post-mortem was performed within thirty minutes of death. Apart from hypostatic congestion in the lungs, enlargement of the Peyer’s patches in the small intestine and engorgement of the mesenteric lymph nodes there were no macroscopic lesions. Histologically the only noteworthy lesions were in the central nervous system. The olfactory lobes, cerebral hemispheric and mid-brain showed no lesions. In the pons and to a greater extent in the medulla there was perivascular infiltration with lymphocytes but no degenerative changes in the neurones and little or no infiltration of the grey matter. In sections of the cord at all levels there was extensive perivascular cuffing with lymphocytes and polymorphonuclear leucocytes; little meningeal infiltration was noted. The grey matter of the cord showed considerable infiltration more especially in the region of the anterior horn cells. The infiltrating cells were mainly polymorphonuclear leucocytes except in the cervical region where round cells and polymorphonuclear leucocytes were about equal in number. At all levels of the cord the anterior horn cells had for the most part disappeared on the right side or were obscured by masses of polymorphonuclear leucocytes which seemed to be engaged in neuronophagia. In the lumbar region a few pale ghost-like homogeneous remains of anterior horn cells were distinguished. On the left side the anterior horn cells were more numerous but in many the nuclei stained uniformly and were eccentric. In the few nuclei of some neurones with sections stained by Giemsa’s method could be seen small amorphous eosinophilic masses lying in the nucleoplasm. The histological changes were thus consistent with a diagnosis of anterior poliomyelitis.

Distribution of virus in the tissues.—As soon as it became obvious that myelitic symptoms were developing, blood and cerebrospinal fluid were removed and injected intracerebrally into mice and monkeys. No symptoms developed in these animals. At the post-mortem brain, cord and mesenteric lymph nodes were removed aseptically, ground up to form a 10 per cent suspension in broth and injected intracerebrally into mice and monkeys. Feces were removed from the small and large intestines and, after treatment with ether, were injected intraperitoneally into monkeys.

The results of the monkey inoculations are shown in the table.

<table>
<thead>
<tr>
<th>Material injected</th>
<th>Species of monkey injected</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Olfactory lobes</td>
<td>Cercothecus aethiops centralis</td>
<td>Survival, no symptoms</td>
</tr>
<tr>
<td></td>
<td><em>Papio papio</em></td>
<td></td>
</tr>
<tr>
<td>Frontal lobes</td>
<td><em>Erythrocebus patas</em></td>
<td>&quot;</td>
</tr>
<tr>
<td></td>
<td><em>Cercothecus mona roloway</em></td>
<td>&quot;</td>
</tr>
<tr>
<td>Pons</td>
<td><em>Cercocebus torquatus</em></td>
<td>&quot;</td>
</tr>
<tr>
<td>Cervical cord</td>
<td><em>C. aethiops centralis</em></td>
<td>Paralysis 20th day</td>
</tr>
<tr>
<td>Dorsal cord</td>
<td><em>C. mona roloway</em></td>
<td>9th</td>
</tr>
<tr>
<td>Mesenteric lymph nodes</td>
<td><em>C. aethiops centralis</em></td>
<td>Survival, no symptoms</td>
</tr>
<tr>
<td>Feces from small intestine</td>
<td><em>E. patas</em></td>
<td>Death 12th day, but no lesions</td>
</tr>
<tr>
<td>Feces from large intestine</td>
<td><em>C. mona roloway</em></td>
<td>Survival, no symptoms</td>
</tr>
<tr>
<td></td>
<td><em>E. patas</em></td>
<td>Paralysis 8th day</td>
</tr>
</tbody>
</table>
Symptoms in all monkeys were the same, slight hyper-excitability quickly followed by weakness and paralysis of the hind legs, extending upwards to the trunk and arms.

Blood and brain of each monkey were cultured for sterility when the animals were killed and in no case was any bacterial contamination present. The histological changes in all monkeys were typical of an acute poliomyelitis; numerous haemorrhages were found through the mid-brain. The cord of a Cercopithecus ethiops monkey dying with paralysis after injection of cervical cord was further injected intracerebrally into two other monkeys, Papio papio and E. patas in doses of 1 c.c. of a 10 per cent suspension. The monkeys developed paralysis on the seventh and eighth days after inoculation respectively. The lesions in the cord and brain of these two monkeys were similar to those in the primary passage monkeys. Similarly an emulsion of cord from the Cercopithecus monkey developing symptoms after injection of pons was inoculated into a Mona monkey which developed symptoms on the sixth day after inoculation.

Attempts to transmit the virus by intracerebral injection to mice and to bush rats Cricetomys gambianus failed.

It is of interest that one Patas monkey at death was found to have a heavy infection of Plasmodium kochi in the blood. All monkeys when examined for malaria parasites a week or two earlier had been negative.

It will thus be seen that the virus was demonstrable in the patient’s large intestine, cervical and dorsal cord and pons. The frontal and olfactory lobes showed no virus and histologically exhibited no lesions.

**DISCUSSION.**

The observations here recorded show that poliomyelitis undoubtedly exists in West Africa; in addition sera from West Africans kindly examined by Dr. Peter Olitsky of the Rockefeller Institute, New York, have shown the presence of neutralizing antibodies to the Lansing strain of poliomyelitis virus. These serological tests together with clinical and pathological observations show that poliomyelitis virus is widely distributed in West Africa. In view of the demonstration by Schlesinger, Morgan and Olitsky (1943) that there exists in the Middle East a strain of poliomyelitis virus similar to the original Lansing strain the pathogenicity of the West African virus to the cotton-tail rat is being investigated.

The extreme rarity of the disease among young African soldiers is remarkable in view of the fact that a low standard of hygiene and the presence of flies is universal while many Africans have been stationed away from their country of origin. The lower incidence of poliomyelitis among negroes than among whites has a striking parallel in the United States of America where it has been often noted in epidemics that negroes are attacked less frequently than whites (cf. Lewis, 1942): the resistance of the negro would seem to be due more probably to an acquired immunity than to any racial resistance.

Among Service personnel in West Africa there has fortunately been no outbreak such as occurred during the war in Malta and the Middle East; nevertheless both the incidence and mortality have been greater among European than among African troops.

There are at present two rival theories of the spread of poliomyelitis; on the one hand it is believed to spread as a droplet infection, the portal of entry being the nasopharynx, on the other it is thought to contaminate food and water, and to enter through the alimentary tract.

Seddon et al. (1945) in discussing the means of propagation of the Malta epidemic unhesitatingly accept the droplet nuclei theory of spread.

On the other hand since Trask, Vigner and Paul (1938), confirming earlier work, showed how easily the poliomyelitis virus could be obtained from the human faeces there is overwhelming evidence that the whole human alimentary tract from the mouth and pharynx to the colon may be a portal of entry for the virus (Sabine and Ward, 1941). In addition, poliomyelitis virus has been isolated from sewage, flies and more recently by Ward, Melnick and Horstmann (1945) from food contaminated by flies during an epidemic.
So far as it goes the evidence from West Africa is in favour of the theory of spread by feces, flies and contaminated food. The disease occurs throughout the year as does amoebiasis and infective hepatitis but is more common in the rains: flies are even more abundant at this period and it is never easy to wean the African from the idea that the whole of Africa is a ready-made latrine. On the other hand in West Africa diseases transmitted by droplet nuclei have marked seasonal incidence. Pneumonia and cerebrospinal meningitis as well as smallpox all have their greatest prevalence during the dry dusty months of the year, when the Harmattan blows, decreasing in incidence as soon as the first rains appear at the end of March.

If poliomyelitis is carried as a droplet infection it is strange that officers both in India, the Middle East, North Africa (McAlpine, 1945) and West Africa should be more liable than British other ranks to the disease, for officers tend to live in less crowded quarters; on the other hand they invariably employ more Mess servants, and dine out more frequently, while the general supervision and hygienic standards of Officers' Messes are notoriously poor.

It has already been mentioned that in the West African Command the other diseases to which officers are more liable than British other ranks are amoebic dysentery and infective hepatitis, both of which are transmitted by the alimentary canal.

Finally, although it is unsafe to argue from a single instance, the distribution of the virus in the body of the case here described lends some support to the theory of spread by contaminated feces.

The experimental evidence shows that West African monkeys, *Papio papio*, *Cercopithecus aethiops centralis*, *Erythrocebus patas*, *Cercopithecus mona roloway* and *Cercocebus torquatus* are susceptible to a West African strain of poliomyelitis. Previous experiments have shown that the first two species, *P. papio* and *C. aethiops centralis*, are also susceptible to a strain of poliomyelitis virus sent from the Middle East by Major C. E. van Rooyen, R.A.M.C.

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**CONCLUSIONS.**

Poliomyelitis is endemic in West Africa but is rare among West African soldiers. Its incidence among European Service personnel in the tropics is greater than among African soldiers. Officers are more predisposed to infection than other ranks. The virus has been isolated from the pons, cord, and large intestine of a fatal case in West Africa.

West African monkeys, *Papio papio*, *Cercopithecus aethiops centralis*, *Cercopithecus mona roloway*, *Cercocebus torquatus* and *Erythrocebus patas* are susceptible to poliomyelitis virus.

Sera from Africans in West Africa contain immune bodies to the Lansing strain of poliomyelitis virus.

**REFERENCES.**


