The findings as shown justify me in stating as my considered opinion that all deaths from suspected accidental infant suffocation should be the subject of post-mortem examinations, both from the point of view of correct statistical information and in some cases to relieve the mother from mental torture or reproach. Despite their grief, the relief of parents is often very apparent when informed that the death was due to circumstances beyond their control. Careful inquiries made in every case have revealed that there was a complete absence of prodromal symptoms or any departure from the normal sufficient to justify the parent in seeking medical advice—in other words, death was entirely unexpected, even in those cases in which the cause of death was found to be respiratory or due to otitis media. It would appear that an infant could develop bronchopneumonia or capillary bronchitis without complications, or even accompanied by otitis media, and die suddenly without warning, as is shown by a considerable number of the 229 deaths from these causes.

Quite a number of the children in all groups were found prone or with the face turned into the pillow—borne out by post-mortem hypostasis—suggesting death from obstruction to the air passages; and in the absence of other factors one might naturally conclude that death was caused by mechanical means.

In the 11 cases of death from inspiration of vomit, inquiry into the circumstances of how and when the child was fed did not seem to have any direct bearing on the death.

The total numbers of deaths due to mechanical suffocation in bed, cot, or cradle, etc., in England and Wales, taken from the Registrar-General's Returns, for the years 1940, 1941, and 1942, were 308, 379, and 358, respectively (*B.M.J.*, 1945, 1, 602). If the Birmingham figures are any guide, in view of the fact that the population is approximately one-fortieth of that of the country, it would appear that these figures are much too high, and that they would be corrected by the universal procedure of post-mortem examination.

My experience is that doctors who, at the time of reporting a death, honestly believed that a child had been suffocated by mechanical means have frequently expressed their surprise on attending the post-mortem examination, which showed that death was not due to these causes but to other factors. Consequently, I consider that every medical practitioner should act warily in such cases, and should so qualify his opinion as to the cause of death as to necessitate the coroner concerned using his powers to obtain the best opinion possible, by means of a post-mortem examination.

It will be observed from Table I that all cases of children asphyxiated while in bed with the parents occurred during the first four months, and that all cases of asphyxia by mechanical

TABLE II.—Seasonal Incidence, 1938–44 (7 years)

	Jan.	Feb.	Mar.	April	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Total
Respiratory infections with or without contributory cause	40	27	31	17	16	7	10	7	19	22	24	37	257
Asphyxia in bed with parents	2	1	.3	—	2	—	1	—	—	2	5	2	18
Asphyxia while sleeping alone	—	2	—	—	—	—	—	—	2	—	1	1	6

Summary

Attention is drawn to the fact that statistics relating to infant suffocation are misleading and err on the side of over-estimation. Circumstantial evidence that suffocation was the cause of death is not sufficient.

Accurate information regarding the true position in a city of 1,000,000 inhabitants revealed an incidence very much lower than is generally accepted.

During the years 1938 to 1944, of 318 deaths considered as possibly due to asphyxia only 38 were shown to have been so caused; 18 of these resulted from mechanical suffocation while in bed with some other person, and 6 from mechanical suffocation while sleeping alone. The remaining 280 deaths were definitely ascribed to natural causes, principally bronchopneumonia, often with an associated otitis media.

A post-mortem examination is indispensable as part of the routine investigation in every case where suffocation is considered a likely cause of death. Evidence in support of this is furnished by the Birmingham deaths, which show such a pronounced fall from 1929 onwards, when this procedure was adopted.

The number of deaths in infants reported to the coroner during the past three decades shows a very marked and progressive fall. This is undoubtedly due to many factors, including improved social and housing conditions, and the activities of the child welfare section of the public health authorities; but in the group in which death was the result of mechanical suffocation I feel that the present statistics for the whole country do not do justice to the real improvement that has taken place in recent years.

The remedy rests in the first instance with the medical practitioner, and, failing him, with the coroner.

POLYNEURITIS AFTER JUNGLE SORES

A SERIES OF TWENTY-ONE CASES

BY

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Chart showing annual deaths from mechanical suffocation while in bed with parents, or others, in Birmingham from 1916 to 1944.

means occurred during the first seven months of life. Seasonal incidence in deaths from respiratory infections and mechanical asphyxia is shown in Table II. Annual deaths from suffocation while in bed with parents or others occurring in Birmingham from 1916 onwards are shown in the accompanying chart.

In a recent campaign in the Burmese jungle British and Indian troops were fighting for upwards of five months under appalling geographical and climatic conditions. During the operation many of these men developed multiple indolent ulcers, mainly on their limbs, and commonly known as "jungle sores." It was noticed that several of those who had suffered from jungle sores contracted a peripheral neuritis later. This paper describes a series of such cases. Owing to war conditions it has been impossible to make satisfactory laboratory investigations; for the same reason the literature on the subject was not available. Therefore this description must be considered from the purely clinical standpoint.

Clinical Data

Twenty-one cases of peripheral neuritis were seen in hospital. Of these, 11 were admitted complaining of symptoms referable to their neuritis; nine because of malarial relapse, and one with jungle sores. All of them had suffered from jungle sores, but, except for Case 19, the ulcers were healed at the time of admission. The number of ulcers on each man varied from 2 to 40, with an average of 14. The commonest sites were the lower leg and forearm. Ulcer scars were also found on the wrists, ankles, arms, buttocks, and shoulders, in that order of frequency. The average healing time was 8 weeks, but varied from 4 to 17. The description of the jungle sores as given by the patients in no way differed from the unhealed sores seen by us. A typical one was about $\frac{1}{4}$ in. in diameter, circular or oval in shape. It presented a punched-out appearance, with a necrotic sloughing

base and unhealthy purple edges; but the local reaction was slight, and was confined to the margins of the ulcer.

The clinical course of all cases in the series was remarkably constant, and the sequence was as follows. In 16 cases the first symptom of nervous involvement was blurring of vision, the common complaint being that after reading two or three pages the print became blurred and reading was impossible. This symptom was usually noticed about seven weeks after the jungle sores began. The earliest it appeared was in four weeks and the latest 12. The average duration of blurred vision was four weeks, the eyesight returning to normal after this time. In all patients there was disturbance of sensation. In four cases it synchronized with blurring of vision, but in the remainder it occurred three weeks later. The average time between the onset of jungle sores and the appearance of sensory disturbance was 10 weeks. Sensory symptoms, consisting of tingling, numbness, and coldness of the extremities, were often severe enough to make the men report sick. In the milder cases these paraesthesiae were the only complaints; however, others later complained of difficulty in walking and writing. The picture in its most severe form was completed by progressive weakness of the limbs spreading to involve hips and shoulders.

corresponding reflex was absent or sluggish. Muscle tone was normal in all cases and wasting of muscles did not occur. Except for these findings in the C.N.S., physical examination was negative. The cases fell into the age group 20-30. The general condition of all was good. No anaemia, glossitis, or malnutrition was observed. Clinically there was no cardiac abnormality and the pulse was normal in rate and rhythm. The gastro-intestinal tract was also normal.

Mild Cases

The cases may be classified according to the severity of their disability. Eleven were classified as mild—i.e., disability unnoticed except when attempting physical exertion. A typical example of this group was Case 8.

Case 8.—Admitted to hospital for malaria. Also complained of tingling and numbness of hands and feet. Stated that he found it difficult to march more than a mile. Jungle sores began in Burma, followed 8 weeks later by blurring of vision. Four weeks later the paraesthesiae started and the jungle sores healed. No history of diarrhoea. On examination, nutrition good. No evidence of avitaminosis or anaemia. Scars of healed jungle sores on legs. All systems except C.N.S. normal. C.N.S. examination: pupils regular, equal, reacted to light and accommodation; all cranial nerves

Synopsis of Cases*

Case No.	Jungle Sores		Accommodation		(a) Sensory Symptoms. Onset (weeks)	Sensation		Gait Affected ?	(b) Weakness		Tendon Reflexes	(c) Disability Group
	No.	Healing Time (weeks)	Affected ?	(a) Onset (weeks)		Anaes.	Posture Affected ?		Arms	Legs		
1	22	17	Yes	12	16	+	+	+	++++	+++	All abs.	2
2	8	6	"	7	8	-	+	+	+++	+++	A.J.s and K.J.s abs.	1
3	30	13	No	N.A.	13	+	+	+	L > R	L > R	All abs.	2
4	12	4	Yes	4	4	±	+	±	+++	+++	Rt. A.J. and Rt. K.J. abs.	1
5	6	4	"	4	8	+	±	-	+	+++	A.J.s and K.J.s abs.	1
6	16	8	"	4	8	+	±	+	++++	+++	All abs.	3
7	15	10	"	5	8	+	±	-	+++	+++	A.J.s and Z.J.s abs.	1
8	25	12	"	8	12	+	±	±	+++	+++	A.J.s and K.J.s abs.	1
9	5	10	"	6	10	±	±	±	+++	+++	All abs.	1
10	2	8	"	6	10	±	±	±	+++	+++	"	2
11	10	6	No	N.A.	6	+	+	±	+++	+++	"	1
12	30	8	Yes	8	7	+	+	+	++++	+++	"	3
13	8	8	No	N.A.	10	+	+	+	+++	+++	"	2
14	7	12	Yes	12	14	+	+	+	+++	+++	A.J.s and K.J.s abs.	2
15	40	9	"	6	6	+	+	+	+++	+++	All abs.	2
16	8	6	"	4	8	-	±	+	+++	+++	"	1
17	10	8	"	12	16	+	±	±	+++	+++	"	1
18	6	8	No	N.A.	12	+	±	±	+++	+++	A.J.s and K.J.s abs.	1
19	8	11	"	N.A.	14	+	±	±	+++	+++	All abs.	3
20	14	8	Yes	6	15	+	+	+	+++	+++	"	2
21	14	16	"	12	16	-	-	±	±	++	"	1

(a) Time of onset reckoned from beginning of jungle sores.

(b) + = Weakness of grip/foot.

++ = Weakness up to and including forearm/leg.

+++ = Weakness up to and including upper arm/thigh.

++++ = Weakness up to and including shoulder/buttock.

(c) 1 = Disability only on exertion.

2 = Routine life impeded.

3 = Bed-ridden.

In addition anaesthesia of the extremities, astereognosis, and ataxia were present. From a shuffling walk with feet apart the gait deteriorated until walking was impossible.

Examination of the nervous system in these cases showed the characteristic signs of a polyneuritis. In addition to this, paralysis of accommodation was a marked feature of the disease. However, in the few cases in which eyesight was blurred during stay in hospital the pupils reacted to light and to accommodation. The phenomenon was subjective, and was shown by defective near vision. There was paresis of a lower motor neurone type, peripheral in distribution; maximum weakness being in the hands and feet. The weakness spread centripetally, but recovery took place in the reverse direction. Loss of power was more pronounced in the extensor than in the flexor muscle groups; although characteristically bilateral, it often predominated on one side. The distribution of the paresis in no way followed that of the jungle sores. Impairment of sensation followed the distribution of the paresis, all forms being affected, including postural sense. In the severe cases astereognosis and ataxia were present. Increased muscular tenderness was rare. Vasomotor disturbances were seen in a few cases, as evidenced by blueness and coldness of the extremities. It was observed that the area affected by paraesthesiae was larger than that of actual anaesthesia. Tendon reflexes of the affected limb were either absent on admission or rapidly disappeared. Even when there was minor disturbance of motor and sensory function the

normal; bilateral weakness of grip and dorsiflexion and plantar flexion of feet; no other muscular weakness; no disturbance of muscle tone; slight impairment of all forms of sensation over both feet; postural sense slightly impaired; ankle- and knee-jerks absent bilaterally; all other reflexes present. Gait fair while wearing light shoes; with boots, dragging of toes and walking with feet abnormally wide apart, this being accentuated when the eyes were closed. In two weeks paraesthesiae had disappeared. In three weeks power was returning to normal, and the patient was discharged from hospital, knee- and ankle-jerks still being absent.

Moderately Severe Cases

In seven cases, classed as moderately severe, the disability was sufficient to impede the patient's everyday life. Case 1 is an example.

Case 1.—Admitted for symptoms referable to polyneuritis. Nineteen weeks before admission jungle sores started, and 12 weeks later he noticed blurring of vision. Three weeks before admission, onset of increasingly severe tingling in hands and forearms. Simultaneously walking became difficult. Weakness was progressive, until on the date of admission he could scarcely walk. Fine movements of hands were clumsy. Abnormal physical signs: Pronounced bilateral weakness of grip and forearms; moderate weakness of upper arms; marked weakness of feet, legs, and thighs; some degree of left foot-drop; in all limbs extensor muscle groups were weaker than the flexor; partial anaesthesia of "glove-and-stockings" type bilaterally; postural sense very poor; astereognosis and ataxia marked. All tendon reflexes absent. Progress: Power returned

steadily; in two weeks foot-drop disappeared. After three weeks fine movements of hands became possible and anaesthesia was absent. Patient was discharged after six weeks; shortly beforehand paraesthesiae had gone, but although power greatly improved, it would not permit severe exertion.

Severe Cases

Of the series only three cases became bed-ridden; one of these is described.

Case 12.—Gave a history of jungle sores beginning two months previously; finally healed a few days before admission. Present complaints: tingling of extremities, unsteadiness of gait, and blurring of vision. These occurred at approximately the same time as the sores healed. On examination, general condition good. No evidence of avitaminosis or anaemia. Scars of healed jungle sores on legs and forearms. All systems normal except C.N.S. C.N.S. examination: pupils and cranial nerves normal; weakness of grip—L.>R.; slight weakness of extensors of wrist and dorsiflexors of ankle; no anaesthesia; upper-limb tendon reflexes present; ankle- and knee-jerks absent on both sides. Two days after admission:—Tendency to foot-drop, L.>R.; impairment of all forms of sensation up to knees; astereognosis present; biceps jerks now absent. There followed a rapid increase in weakness, maximal peripherally and spreading towards the trunk. Ten days after admission:—Patient unable to move either arms or legs; muscles of both hips and shoulders weak; sensation bilaterally impaired up to the elbows, and on legs up to and including the perineum; no disturbance of sphincters; all tendon reflexes absent; cremasteric and abdominal reflexes present. One month after admission:—First signs of recovery appeared; power of hips and shoulders began to return. Six weeks after admission:—Able to lift arms above head; power returning to upper arms and thighs; foot-drop improved; area of anaesthesia greatly diminished. Eight weeks after admission:—Able to use hands and stand with support; extremities still weak and with impaired sensation; all tendon reflexes absent. The paraesthesiae, which had disappeared at the time of maximum anaesthesia and had returned in the early stages of recovery, now left him entirely. At this point the patient was transferred to a rehabilitation centre.

Discussion

There is no doubt that these patients were suffering from a polyneuritis, the aetiology of the condition being the main point of interest. Chemical poisons such as arsenic can be excluded as the aetiological agent, since no patient gave a history of exposure to this. Acute infective polyneuritis is excluded on the natural history of the disease, the onset in the series being insidious and the course afebrile. It remains to decide whether the polyneuritis is the result of a vitamin deficiency or is due to a bacterial toxin. Cases of vitamin B deficiency were seen in troops engaged in the same operation; the picture presented by these was primarily one of wasting and diarrhoea, with glossitis. Polyneuritis, when present, was mild and responded quickly to intensive vitamin therapy.

In the cases under discussion the polyneuritis, as has been shown, could be completely disabling, and was not influenced by adequate doses of the vitamin B complex. Moreover, the nutrition of all cases was good, despite the conditions under which they had been living, no glossitis and no diarrhoea being noticed.

Since a constant association between jungle sores and a subsequent polyneuritis was noticed, it is likely that a toxin, elaborated by bacteria infecting these ulcers, was the responsible agent. Clinically a marked similarity exists between jungle sores and the desert sores seen in troops of the M.E.F. It was found that a number of desert sores were infected with diphtheria bacilli; similarly these organisms have been isolated from jungle sores. In our series of polyneuritic cases, except for one patient, the sores were healed at the time of admission. From the unhealed sores of this case (No. 19) diphtheria bacilli were isolated. However, a week after admission this patient developed faucial diphtheria; therefore it is difficult to decide whether the polyneuritis was due to the faucial or to the extrafaucial infection.

The time of onset of polyneuritis indicated the latter. The outstanding clinical point in favour of a diphtheritic aetiology is the paralysis of accommodation noted in a large percentage of cases. It is also important to note that the neuritis was not locally related to those areas affected by jungle sores. In a few cases neuritis of the legs and arms followed when the ulcers had been present only on the arms, and vice versa. The loss of accommodation can also be explained by assuming a blood-borne spread of the toxin. This form of generalized peripheral

neuritis is seen in both faucial and extrafaucial diphtheria, and as a clinical entity differs from other types of polyneuritis. The number of ulcers did not influence the severity of the disease; similarly, neither did the duration of unhealed jungle sores. A feature of interest was the fact that although Indian troops taking part in the same operation developed jungle sores, no case of a subsequent polyneuritis was discovered. It is well known that diphtheritic infection is rare in Indians.

From the above facts it is felt that all the cases represent a polyneuritis of diphtheritic origin. The value of Schick testing was obvious, but although every attempt was made to obtain the toxin none was available. Electrocardiographic studies would also have been valuable. The origin of a diphtheritic infection incurred in the jungle is obscure. These cases of polyneuritis were drawn from all the units concerned in the operation. If a carrier was the source of infection it is likely that the cases would have been grouped in one unit. All troops came into contact with the native population, and it is more likely that the infection was introduced from them. However, no details are known of the incidence or carrier rate of diphtheria in the Burmese.

From a military point of view this disease can cause a serious wastage of man-power. Even the mild cases will be unfit for jungle warfare for six months from the onset of their disability. The severe cases will probably take a year to recover fully. These men neither had been immunized against diphtheria nor had previously suffered from the disease. If the diphtheritic origin of this polyneuritis is accepted the question of prophylactic immunization will arise for troops engaged in future jungle operations.

Summary

Twenty-one cases of polyneuritis subsequent to jungle sores are described.

The course of the disease followed a constant pattern.

Sixteen out of 21 cases had loss of accommodation.

In reviewing the aetiology it was considered most likely that the polyneuritis followed a diphtheritic infection of the jungle sores.

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ACUTE INFECTIVE POLYNEURITIS

BY

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A form of acute polyneuritis apparently of infective origin is well recognized by neurologists but has received little attention in English medical textbooks. It is probably more common than polyneuritis due to the classical toxins, and seems often to be a source of diagnostic confusion. The following case, which appears to be typical, seems therefore worth recording.

Case Report

An air-gunner aged 23 was admitted to his station sick-quarters with a left facial palsy. On the day of admission he rapidly developed "pins and needles" in arms and legs, with marked muscular weakness. The condition was recognized as a generalized one, and he was transferred to hospital. There was no sphincter disturbance. On admission to hospital it was elicited that he had had a bad cold and chill a fortnight previously. Apart from appendicectomy a year ago, he had always been healthy and never a heavy drinker. On examination he was afebrile, but appeared ill. A complete left facial palsy of the lower motor neurone type was present, but the other cranial nerves were normal. The fundus oculi was healthy. There was marked muscular weakness of arms and legs, with winging of the right scapula. The proximal muscle groups, indeed, were particularly affected. Tendon reflexes could still be obtained on admission, but within a few days had entirely disappeared from the legs and left arm, and were feeble in the right arm. The muscles were flaccid and toneless. The upper abdominal reflexes were present, but not the lower; the plantars were flexor. No reliable sensory changes could be discovered, but the paraesthesiae were the cause of much complaint. The throat was clear, the nose a little congested. No K.L.B. could be isolated from a swab. Other systems were normal. Lumbar puncture on the day after admission showed a clear fluid under normal pressure, with no cells; protein,