about their unsightliness. They did not irritate, and they showed the usual seasonal variation. Since taking morphine she had been very moody—easily excited, then depressed. She rarely went out, and often made scenes—screaming, etc.—when the morphine allowance was finished. Her son dreaded coming home. She was admitted to the observation unit, St. Alfege's Hospital, on Aug. 7, 1941. She was pale, and the typical brownish branny patches on her skin stood out in contrast. In general the exposed areas were pigmented. There were particularly well marked pigmented patches over the manubrium sterni and nape of the neck, disposed "necklace" fashion, also on the backs of the fingers, wrists, and insteps. Her tongue was pale and very smooth. There was no fur, despite the poor appetite and the fact that she had taken so little food before admission. Her blood pressure was 110/70. There was gross oedema of the ankles

All tendon-jerks were absent. The patient said that they had been absent for years. She did not display the classical pellagrous dementia. Cerebration was quick and fairly accurate. She was hypochondriacal, and would resort to any device, from violence to wheedling, to get morphine. She was very self-centred. The blood Wassermann reaction and Kahn test were negative. A barium meal showed evidence of "gastritis" (thickening of rugae), with poor gastric tone and some initial delay followed by rapid emptying. No ulcer was demonstrated.

Progress.—The patient remained at St. Alfege's Hospital for 11 days, improving considerably and putting on weight. She had been extremely emaciated, with deep-sunken eyes, but at the time of her transfer had filled out considerably. No morphine was given, and after the first day no sedatives, although her craving persisted and she became very depressed when refused the drug. However, she soon became more reasonable and co-operative, and was willing to discuss other subjects than sedatives. On Aug. 18 she was transferred to Bexley Hospital, where a blood count showed Hb 44%, R.B.C. 2,430,000, C.I. 0.9. The patient was given large doses of iron and liver extract. Subsequent blood counts were as follows: Sept. 23—Hb 48%, R.B.C. 3,390,000, C.I. 0.71; Dec. 3—Hb 88%, R.B.C. 5,000,000. She was given nicotinic acid in 50-mg. doses six times daily, and thiamine in 50-mg. doses twice daily subcutaneously to a total of 1.8 g. Shortly after transfer she collapsed and was very ill for a few days. On recovery from this attack her physical improvement was progressive. She developed a ravenous appetite. There was at first no mental improvement, but four months after the beginning of treatment the mental state also changed for the better, and by mid-January, 1942, she had improved strikingly. She was quick, lively, cheerful, and took an active interest in her surroundings and in plans for her future. She was much less pale and the branny patches had become less evident. She was able to get about well and had put on much weight. There was still some oedema of the ankles on exertion. She was discharged home on Feb. 7, 1942.

When visited 16 months later the patient was active, able to walk several miles, and could do her housework; but unfortunately she had not co-operated in out-patient treatment, and was receiving no vitamin concentrates. She had deliberately restricted her diet on account of dyspepsia, and was taking only the small priority ration of eggs and milk which she was allowed, together with a very little bread-and-butter. She again had slight oedema of the ankles and branny patches on face, neck, arms, wrists, and hands; and her memory, especially for names, was poor. She declared that she was not taking morphia or alcohol.

Discussion

It is clear that in this case, as in most cases of vitamin deficiency, there was evidence of shortage of more than one essential factor (Sebrell, 1940). The main pellagrous picture is attributable to a nicotinic acid deficiency, but the polyneuritis and the cardiovascular disturbance suggest a thiamine lack, and the anaemia may have been due to a shortage of iron or of pyridoxine (vitamin B_6 : Unna, 1940). The exact mechanism of causation cannot be determined with certainty, but the principal factor was probably the apathy and loss of appetite induced by morphine addiction.

The effect of morphine on digestion and absorption is complex. The secretion of digestive juices is diminished (Douthwaite, 1932; Edwards and Gunn, 1941), but the effect of this reduction is counterbalanced by a subsequent increase in the gastric secretion and by the fact that the food remains longer in the stomach and intestine. Thus there is no reduction in absorption attributable to the direct action of morphine. It does not therefore appear that direct disturbance of digestive function by the drug can have been a prominent factor in the production of pellagra.

A further possibility is suggested by the work of Shideman and Seevers (1941), which indicates that chronic morphine addiction and thiamine deficiency both produce the same sort of metabolic defect (characterized by a reduction of the normal increase in oxygen output from muscle following addition of pyruvate and co-carboxylase *in vitro*). Ellinger, Hassan, and Taha (1937) noted that dehydrogenases are inhibited by narcotics. This would not account for the pellagrous features of the above case, but might be partially responsible for the development of polyneuritis and oedema. The tendency, after cessation of therapy, to relapse without a return of the morphine addiction does not eliminate any of these possibilities, since, on the one hand, permanent metabolic changes may have been caused by the prolonged habituation to morphine, and, on the other, her diet after discharge was probably inadequate.

Summary

A case of pellagra in a long-standing morphine addict is discussed. There were also polyneuritis and anaemia. Great improvement occurred with treatment, followed by relapse on cessation of therapy. Loss of appetite due to morphine was thought to be the cause of the pellagra. (The possibility that morphine may disturb metabolism more directly is considered.)

Our thanks are due to Dr. Eli Davis, deputy medical superintendent, St. Andrew's Hospital, Bow, for his help in diagnosis and treatment of the patient and for his assistance in the compilation of this report.

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MALARIA EPIDEMICS AT EXCEPTIONALLY HIGH ALTITUDES IN KENYA

BY

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Malaria in tropical and subtropical countries is usually thought to disappear once a certain altitude is reached, though the accepted "safe" height varies from place to place. Six thousand feet is usually regarded as the upper limit. Manson-Bahr (1941) quotes figures for Abyssinia which indicate that the transmission of malaria in that country ceases at 6,600 feet, though doubtful records exist of cases of benign tertian malaria at Addis Ababa at 8,175 feet. Schwetz (1942) in a review of the African literature gives limits up to 5,000–6,500 feet.

Hitherto in Kenya Colony malaria was thought to be absent from places above 7,000 feet. In June, 1941, a small farming community at Londiani (7,500–7,800 feet) was suddenly stricken by a severe epidemic of the disease. On 12 of the 14 farms at least one European became infected between June and August, and on every farm cases of malaria were numerous among the African labourers. Blood slides taken from 35 ill Africans (of non-immune tribes, and long resident in the district) were examined, and 27 showed malaria parasites. Twenty-six of these infections were *Plasmodium falciparum*, and one was *P. vivax*. This represented only a small sample of the total number of cases. The European infections were malignant tertian with the exception of a single case of benign tertian.

The Londiani district (75 miles from the provincial centre, Kisumu) comprises a small township with an Indian bazaar and native location surrounded by European farms extending for a radius of about 12 miles. There was also at the time of the outbreak a large military camp sited at Londiani. Bordering the farms there are extensive areas of forest reserve in which live African squatters numbering several thousands. The epidemic particularly affected the farms and the military camp, though in the latter many of the cases may have been relapses from infections contracted elsewhere. It is interesting to note that little malaria occurred in the isolated huts of the forest squatters.

The epidemic came to an abrupt end in August, 1941. Smaller outbreaks occurred in 1942, 1943, and 1944, at the same season.

Many of the cases were severe because treatment was delayed, the reason for the delay being that malaria was not thought of in these cold highlands. In 1943 several cases with typhoid-like symptoms and nearly moribund were seen, and these were proved to be malaria only by finding rings and schizonts of *P. falciparum* in the blood. The same curious localization of cases occurred in the later years : in November, 1943, through the kindness of the military authorities, I was permitted to examine 300 Italian prisoners of war who had been resident for at least six months in the Londiani camp, and not a single one showed malaria parasites in the blood slides (thick drop preparations).

Much of the country at Londiani is vlei land, and in rainy weather becomes waterlogged, providing ample facilities for mosquito breeding. Dammed streams, quarry pits, roadside drains, and seepages are also common anopheline breeding-places. Table I gives the results of several mosquito surveys undertaken at Londiani.

TABLE I.—*Anopheline Mosquito Surveys—Londiani*

Date	Adults*	Larvae
Dec., 1927 ..	Nil	*A. garnhami*
June, 1936 ..	*A. gambiae* (6)	*A. natalensis* and *coustani*
Nov., 1936 ..	Nil	*A. natalensis* and *garnhami*
May, 1941 ..	*A. christyi*	Nil
July, 1941 ..	*A. garnhami* (2)	*A. christyi* and *squamosus*
Aug., 1941 ..	*A. christyi* (5)	*A. christyi* (403), *squamosus* (120), and *coustani* (90)
April, 1942 ..	*A. garnhami* (6) and *kingi* (2)	*A. christyi*, *squamosus*, and *natalensis*
May, 1942 ..	Nil	*A. gambiae*
July, 1942 ..	*A. gambiae* (3) and *christyi* (6)	*A. christyi* (319), *coustani* (15), and *garnhami* (4)
June, 1943 ..	*A. gambiae* (2), *christyi* (4), *garnhami* (5), *squamosus* (5)	*A. christyi* (7) and *garnhami* (39)
Jan., 1944 ..	*A. christyi* (2) and *natalensis* (1)	*A. christyi* (255) and *squamosus* (5)

* Adults were collected from native huts, cattle-sheds, and river-banks. All *gambiae* came from dwellings.

Of the seven species represented at Londiani only one—*A. gambiae*—is a known vector of malaria. The salivary glands of all the adults collected were examined for sporozoites, but no infections were found. (The glands of one *A. gambiae* were very heavily infected with leptomonads.) These surveys indicate that *A. gambiae* breeds for a short period about May and that adults persist during the next two months. Climatic records for Londiani are shown in Table II.

TABLE II.—*Climatic Records, 1941—Londiani (7,500 feet)*

Month	Rainfall in Inches	Temperatures* in Degrees F.		
		Mean Max.	Mean Min.	½ Max. Min.
Jan.	3·46	73	45	59
Feb.	1·00	76	46	61
March ..	4·56	76	50	63
April ..	10·22	75	50	62
May ..	8·78	73	49	61
June ..	5·47	70	47	59
July	11·01	67	47	57
Aug. ..	5·05	66	46	56
Sept. ..	3·36	70	46	58
Oct.	3·33	71	47	59
Nov. ..	6·35	70	47	59
Dec. ..	2·93	70	47	59

* 1941 temperature records are not available. These figures represent the mean of 1942, 1943, and 1944 observations.

In May, 1944, the extension of malaria into the highlands of Kenya went one step further. An epidemic of a fatal disease was reported on a farm about 20 miles to the north of Londiani at an altitude of 8,300 to 8,500 feet. Twenty cases (among 220 inhabitants) showing symptoms of headache, backache, shivering, and fever were examined and blood slides were taken. Previously one woman had died after six days' illness (during the second half of which she had been semiconscious). Eight cases showed *P. falciparum* (including one with gametocytes) in the blood, and of these eight, six were proved not to have left the farm for at least six months. The fatal case was probably due to cerebral malaria. Most of the sufferers were Africans of the non-immune Kikuyu tribe and lived in huts scattered on the slopes of the hills and down by the streams. A mosquito survey revealed the presence of numerous anopheline larvae (*A. christyi, garnhami,* and *ardensis,* but no *gambiae*) and two very worn specimens of *A. gambiae* (females) in huts. Suitable breeding-places for *A. gambiae* existed in the cattle- and pig-hoof prints in the swampy sides of the streams. The epidemic began about May 24, and the last case occurred on June 7. Malaria was apparently confined to this one farm ; squatters living in the adjacent forests were unaffected. Climatic records of a B.E.A. meteorological station about 10 miles from this area and 500 feet higher are shown in Table III.

TABLE III.—*Climatic Records, 1944—Equator Station (9,000 feet)*

Month	Rainfall in Inches	Temperatures in Degrees F.		
		Mean Max.	Mean Min.	½ Max. Min.
Jan.	0·34	68	45	57
Feb.	0·67	71	46	58
March ..	2·48	70	47	59
April ..	3·85	66	48	57
May ..	5·07	66	47	57
June ..	2·66	65	46	56
July	3·86	60	47	53
Aug. ..	11·66	61	47	54
Sept. ..	6·19	63	47	55
Oct.	2·14	65	46	55
Nov. ..	4·53	64	47	55
Dec. ..	1·11	64	47	55

Temperatures on the affected farm were probably 2° F. higher.

Discussion

Malaria at these surprisingly high altitudes (7,500–8,500 feet) must be governed by three factors at least—the presence of human carriers, a range of rainfall and temperature suitable for the breeding of *A. gambiae,* and a temperature above the known minimum, to enable the parasite to develop in the mosquito.

The carrier problem is simple. Most farms employ large numbers of Africans who come from the malarious lowlands of the province, and these men, and more particularly the children who accompany them, provide a good source of gametocytes.

The climatic factors concerned in the breeding of *A. gambiae* are rainfall and temperature. This species is essentially dependent upon rainfall, and Tables II and III show that it is adequate at Londiani and at the Equator station. Various authors (De Meillon, 1934 ; Leeson, 1931) give a mean temperature of not less than 61° F. as the limit for *A. gambiae.* On the farm below the Equator station this temperature is reached in one month of the year only—in March—when the mean is 59° + 2° = 61° F. At Londiani, four months—February, March, April, and May—appear to be suitable, though the low rainfall of February invalidates that month for *A. gambiae* breeding. A sudden drop in temperature occurs in June, and this presumably inhibits further breeding until the rains and higher temperatures of the succeeding year.

Lastly, there is the sexual cycle of the parasite in the mosquito to consider, and if outside (shade) temperatures were alone to be reckoned with it is obvious that development could not occur. Wenyon (1926) states that a minimum *constant* temperature of 64.4° F. is required for *P. falciparum,* and at this temperature development probably takes three weeks at least. At the Equator station hourly readings showed that temperatures above 64° F. were attained for not more than two hours in the 24. At Londiani the mean temperature never reaches the critical figure. The answer to the problem is provided by the domestic habits of this species of mosquito. *A. gambiae* spends most of its life in human habitations—viz., small thatched huts, with no windows or ventilation, with fires burning night and day, and overcrowded at night with people and goats. In such circumstances the temperature is 5 to 10° F. higher than that prevailing outside. This suggests that the mean temperature inside huts on the "Equator farm" was 59° + 5° F., and at Londiani 61° + 5° F., during the month of May. These figures just suffice for sporogony of *P. falciparum.*

The meteorological conditions are such that the survival of *A. gambiae* throughout the year is extremely doubtful in the case of Londiani and impossible in the case of the "Equator farm," and it is reasonable to assume that the mosquito is introduced annually by road or rail transport. Partial confirmation that the latter occurs is provided by the results of searches of trains arriving at Kisumu. Sixty-three *gambiae* were caught in the coaches in the years 1943 and 1944.

It is unlikely that Wynter-Blyth's (1943) explanation of the occurrence of malaria at exceptionally high altitudes in the

Nilgiris (6,300 feet) applies here. He suggested that warm winds blowing from below brought the mosquitoes up; here they would have to be blown 20 miles, up 4,000 feet, and through a narrow twisting valley, which is inconceivable.

A hypothetical diary of events of the epidemic on the "Equator farm," based on the above considerations, is as follows:

(i) Gravid *A. gambiae* were transported to the farm in motor vehicles coming by road from a malarious valley 20 miles below. *March.*

(ii) Eggs laid and first (and only) generation emerged. *Beginning of April.*

(iii) These adults bit African gametocyte carriers. *April.*

(iv) Sporozoites in glands. *About May 13.*

(v) Human cases. *May 24 to June 7.*

(vi) Death of last infected mosquitoes. *About May 27.*

Summary

Epidemics of malignant tertian malaria began for the first time in Londiani in the year 1941 and were repeated on a milder scale in succeeding years (May to July). The altitude of the locality is 7,500 to 7,800 feet. A few cases of benign tertian malaria also occurred.

In 1944 a localized outbreak of malignant tertian malaria occurred on a farm about 20 miles to the north of Londiani, at an altitude of 8,300 to 8,500 feet. It lasted for a fortnight only.

Anopheles gambiae Giles was found at both places, and is the probable vector.

Climatic records indicate that the breeding of this species is possible at 8,300 feet only during the month of March and at 7,500 feet only during March, April, and May.

Temperature records suggest that the completion of the sporogonic cycle is possible only because *A. gambiae* spends practically all its life indoors, where the temperature is several degrees higher than outside. The external environment is too cold for development to occur.

It is probable that *A. gambiae* is reintroduced annually by road and rail transport.

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Medical Memoranda

Mediastinal Emphysema and Bilateral Pneumothorax after Tracheotomy

The following case of complications after tracheotomy is thought to be interesting enough to be put on record.

CASE REPORT

Early on Christmas morning I operated on a little girl, 2½ years old, suffering from acute asphyxia; by 10 a.m. (10 hours afterwards) the child had died. The obstruction had come on quite suddenly the previous evening. There was marked retraction of the intercostal spaces and the suprasternal notch; the throat was injected, but no membrane was seen. The temperature was 100° F. The mother stated that there had been an alteration in the voice for some time.

Tracheotomy was performed in the usual way without undue haste under light general inhalation anaesthesia. The engorged veins over the trachea were ligated to preserve haemostasis; the layers of the cervical fascia were then incised—the pretracheal transversely—and the tracheotomy tube was inserted without difficulty through an opening made into the trachea. The breathing improved as a result, but did not seem to be completely relieved. On return to the ward the child's condition was fair, but later she became cyanotic, gasped for breath, and finally stopped breathing and died.

I am indebted to Dr. Donald Teare for the post-mortem findings: "Small wart-like papillomata of the vocal cord, tracheitis, acute bronchitis, emphysema of the mediastinum (some of the bullae being 2 cm. in diameter), and bilateral pneumothorax were found." He adds the comment that "acute respiratory infection was superimposed on the papilloma of the cord so that the tracheotomy did not completely relieve the respiratory distress." Though this is the first case of this nature that I have encountered, it would appear from the literature that this complication is not so rare as might be supposed. Minor degrees of emphysema of the mediastinum probably resolve and are therefore overlooked unless a radiograph is taken, and severe conditions, with pneumothorax ending fatally, may not be properly diagnosed unless the disaster which has overtaken the lungs is recognized.

At operation a suture was placed at the top end of the wound because a deep and rather mobile trachea necessitated a long incision, as is often the case in a child, otherwise the wound was left open and lightly packed with gauze beneath the cannula. Close suturing has been thought to be a possible cause (C. Jackson). There is no reason to suppose that the pleural dome was injured at the time (another cause cited). The usual explanation given is that air enters through the wound in the cervical fascia, becomes imprisoned in the mediastinum, especially when the obstruction is not relieved, and finally reaches a tension high enough to rupture the mediastinal pleura, which is generally thin, particularly in children. When there is a pneumothorax there seems always to be a coexisting mediastinal emphysema; the pneumothorax too is usually bilateral.

Michels (1939) has suggested the preliminary use of the bronchoscope to restore an adequate airway as early as possible and so convert a struggling and restless child into a quiet one. At operation it would seem desirable to disturb the layers of the cervical fascia as little as possible after incising them. The possibility of the sudden onset of pneumothorax as a complication after operation, as shown by hyperresonance, a displaced heart, and absent breath sounds, must be kept in mind, and appropriately treated by aspiration when it appears.

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An Unusual Case of the Labyrinthine Fistula Symptom

Rarefaction and compression of air in the external auditory meatus producing nystagmus, giddiness, or other labyrinthine reactions is known as the labyrinthine fistula symptom. The syndrome is caused by the partial destruction of the bony wall of the labyrinth (commonly by chronic otitis, rarely acute), so that alterations in atmospheric pressure within the external auditory meatus are transmitted through the perforated drum membrane to the labyrinthine fluid. This syndrome is important because it indicates direct communication between the infected middle ear and the labyrinth, and in consequence requires special care.

CASE HISTORY

The patient, a man aged 35, was admitted to the West Middlesex County Hospital on Aug. 28, 1943, having had right otorrhoea of 16 years' duration. The ear had been operated on in 1927, a few weeks after onset of aural discharge, when an incomplete mastoidectomy was performed. For eight days previous to admission he had complained of right-sided headache. This had been followed five days later by the development of pain in the right ear, accompanied by true vertigo—objects in the room moving round him in a horizontal plane—and effortless vomiting. He could not say whether objects moved right to left or left to right. Once during those eight days, while lying in bed on his right side, he had felt "giddy" and vomited.

On examination there was a non-pulsating offensive purulent discharge from the right ear. No nystagmus was obtained when carrying out the so-called "labyrinthine fistula test" (rarefaction and compression of air in the external meatus). There was no tenderness over the mastoid. No abnormality of the central nervous system was found, and the C.S.F. was normal. The discharge gave a growth of anaerobic streptococci and the blood count showed a moderate leucocytosis. The history was suggestive of a labyrinthitis. However, signs of the labyrinthine fistula symptom remained negative, although the patient consistently complained of giddiness and headaches.

Three days after admission he dreamt* that he was in a motor-car which was going round and round, faster and faster, from right to left. When he awoke he was violently sick. Inquiry revealed that he had been lying with his right temporal region pressing on the edge of the mattress. Pressure was applied to that region just above the auricle, when a horizontal rotary nystagmus—slow movement to the left—occurred, and the patient had a recrudescence of the above symptoms. It was noted that the old operational scar was more extensive than usual and reached high up on the right temporal region. The E.N.T. surgeon was consulted, and it was decided that the reason for obtaining nystagmus in this unusual site was that connecting the external scar with the labyrinthine fistula

* Dreams in patients suffering from labyrinthitis with fistula symptoms were described by K. Eisinger and P. Schilder in *Mschr. Psych. Neurol.*, 1929, **73**, 314.