YELLOW FEVER AND ITS PREVENTION

FIG. 1.—A good example of a Native Village, kept in excellent order and free from puddles and odds and ends. Observe the stand pump. Kenema, Sierra Leone Protectorate.

[*Frontispiece.*

YELLOW FEVER
AND ITS PREVENTION

A MANUAL FOR MEDICAL STUDENTS AND PRACTITIONERS

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WITH ILLUSTRATIONS

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JOHN MURRAY, ALBEMARLE STREET, W.
1911

OTHER WORKS BY THE SAME AUTHOR ON YELLOW FEVER.

YELLOW FEVER PROPHYLAXIS IN NEW ORLEANS, 1905.

REPORT ON YELLOW FEVER OUTBREAK, BRITISH HONDURAS, 1905.

MOSQUITO OR MAN? 1909.

HEALTH PROGRESS AND ADMINISTRATION IN THE WEST INDIES, 1910.

TO THE LATE

SIR ALFRED LEWIS JONES, K.C.M.G.

FOUNDER AND GENEROUS BENEFACTOR OF THE
LIVERPOOL SCHOOL OF TROPICAL MEDICINE

WHOSE INTENSE SYMPATHY WITH THE SUFFERING OF HIS FELLOW MEN, VIVID IMAGINATION, AND GREAT GRASP OF AFFAIRS, STIMULATED THE AUTHOR TO TRAVEL AND TO ACQUIRE KNOWLEDGE IN DISTANT PARTS OF THE TROPICAL WORLD, THIS VOLUME IS DEDICATED

IN

AFFECTIONATE AND GRATEFUL REMEMBRANCE.

PREFACE

ALTHOUGH I cannot say, like the illustrious historian[1] of the plague at Athens, "having myself had the distemper," I can repeat with him "having seen others suffering under it, I will state what it actually was, and will indicate, in addition, such other matters as will furnish any man who lays them to heart, with the knowledge and the means of calculating beforehand, in case the same misfortune should ever again occur." This work is, in fact, a summary of my experiences and investigations in New Orleans early in 1905, in British Honduras and Central America late in 1905, in Barbados and other islands of the West Indies, and British Guiana in 1909; and lastly, in Sierra Leone, the Gold Coast, Southern Nigeria, in West Africa in 1910, to all of which places I was sent out to investigate the subject of yellow fever. I have treated the subject-matter historically, geographically, and from the point of view of symptomatology and treatment, pathology, diagnosis, epidemiology, entomology, and prophylaxis, and I hope that the manual will be of practical value to the young medical student who intends taking up work in the tropics. Having already in my book on *Health Progress and Administration in the West Indies* sketched the history of yellow fever and its prevention in the West Indies and in Central America, I have in the following pages laid special stress upon the question of yellow fever in West Africa, believing that if the existence of the disease is fully recognised,

[1] Thucydides, ii., 48.

and the simple measures which I advocate are followed, the conquest of West Africa will be free from the ghastly set-backs which the pioneers of Central and South America and the West Indies experienced during the many years in which they were engaged building up commerce and civilisation.

RUBERT BOYCE.

THE UNIVERSITY, LIVERPOOL,
December 1910.

ACKNOWLEDGMENTS

In my previous works on yellow fever I have acknowledged my indebtedness to the numerous medical officers and administrators who so liberally assisted my investigations in the past.

In the present volume, which embodies my experiences in West Africa, I desire to especially thank Drs Rice, Kennan, Strachan, Graham, Garland, and Pickels, and their assistants, all of them colleagues with whom I worked in the 1910 outbreak. For great hospitality and advice I especially desire to record my thanks on the Gold Coast to the late Sir John Rodger, K.C.M.G., and Mr Furley, D.C.; in Nigeria to Mr Thorburn, C.M.G., and Mr James; in Sierra Leone to Sir Leslie Probyn, K.C.M.G.

I also desire to express my indebtedness to my colleagues Mr Robert Newstead, M.Sc., and Professor Howard Kelly of Baltimore, for advice and assistance in the preparation of this work. For the reproductions 44, 46, 47, 51, and 52, I am indebted to Dr White of New Orleans; for charts 6, and 13 to 16 to the work of Dr Guiteras of Havanna, and to Faget's work for the reproduction of several very interesting temperature and pulse curves.

I desire also to thank Colonel Sutton, R.A.M.C., and the Colonial Office for valuable assistance rendered in enabling me to consult original reports.

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PART I

HISTORY AND GEOGRAPHICAL DISTRIBUTION OF YELLOW FEVER

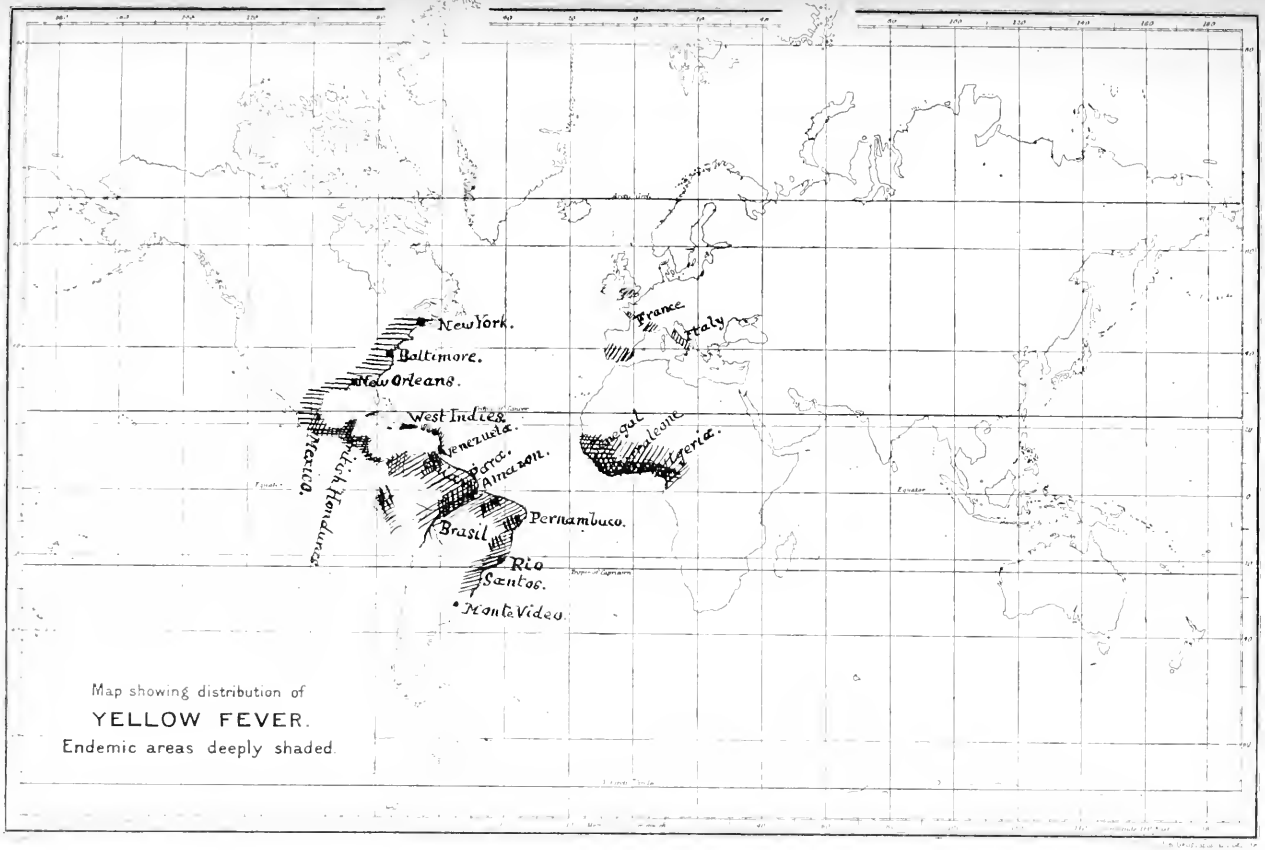

FIG. 2.—Chart to show yellow fever distribution. It is difficult to construct a complete chart, because Yellow Fever is fast disappearing from places where formerly it was endemic. The epidemic areas in Southern Europe and in North America have disappeared, and endemic areas in Central America, the West Indies, and South America have been greatly reduced.

[*To face p.* 2.

CHAPTER I

HISTORY OF YELLOW FEVER IN CENTRAL AMERICA

THE fragmentary historical evidence which we possess, tends to show that yellow fever existed amongst the native races of Central America when the Spaniards arrived.

It is stated to have been known to the ancient Mexicans. It was found in Columbian times amongst the peoples inhabiting the New World. Centuries afterwards evidence points to a similar endemic foothold on the West Coast of Africa.

But, although it is not fruitful to speculate where a disease like yellow fever or malaria, both intimately bound up with the progress of the human race, first made their appearance, nevertheless useful practical information is gained by reviewing the historical data in connection with the progress of both diseases. The information so gained is of use from the epidemiological standpoint, for it shows clearly how disease follows closely upon the expansion of civilisation, whether the lines along which it travels be religious, military, or commercial.

For example, it is to the monks who accompanied the Spanish and Portuguese races in their numerous expeditions to the New World in the Middle Ages that we owe the little we possess to-day of the "discovery," or rather chronicling of the sudden outbursts of the new and mysterious disease which so seriously impeded the founding of settlements in the New World.

CHIEF REFERENCES FOR GENERAL HISTORY AND DISTRIBUTION OF YELLOW FEVER

HIRSCH (AUGUST)—*Handbook of Geographical Pathology, New Sydenham Society*, vol. cvi., London, 1883.

AUGUSTIN (G.)—*History of Yellow Fever*, New Orleans, 1909.

The 1852 *Commission, Whitehall, London*, "Yellow Fever."

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BÉRENGER-FÉRAUD (L. J. B.)—*Traité de la fièvre jaune*, Paris, 1890.

HUMBOLDT (A.)—*Essai politique sur le royaume de la nouvelle Espagne.*

STERNBERG (W.)— *Handbook of the Medical Sciences.*

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HISTORY OF YELLOW FEVER IN CENTRAL AMERICA

(*From Sixteenth to Twentieth Centuries*)

Yellow fever is stated to have been known in pre-Columbian times, and that it was called "Matlazahuatl" by the ancient Mexicans. It is also said to have been present in Vera Cruz since 1509. In other words, yellow fever was one of the established indigenous diseases when the early explorers arrived from Europe. After the sixteenth and seventeenth centuries there appears to have been a pause, and it is not until the nineteenth century that yellow fever becomes again prominent, owing no doubt to commercial expansion in the eighteenth and nineteenth centuries.

In Mexico, as stated above, yellow fever appears to have been known to the ancient Mexicans; and at Vera Cruz, the large port in the Gulf of Mexico, the disease is said to have been endemic since 1509. The reason why our knowledge of yellow fever in Mexico goes back further than in other American republics, is no doubt due to the fact that the country was the seat of an old civilisation, and that it early attracted adventurers. When in later ages other ports and towns were opened to Europeans and North American commerce, yellow

fever immediately made its appearance in epidemic form. In fact, yellow fever followed closely the trade routes, whether by road, rail, or sea, and whether along the coast, in the interior wilder districts, or up on to the mountains.

Wherever newcomers settled, the disease broke out, in some cases no doubt from an imported source, but as often as not from the endemic source. Vera Cruz, being the great port, has persisted as a centre of yellow fever to modern times, the disease manifesting itself in larger or smaller epidemics, or as annual sporadic cases depending upon the number of non-immunes present in any one year, and therefore influenced by the movements of troops or settlers.

In like manner, the ports of Tampico and Progresso have been, since the early part of the nineteenth century, the seats of epidemics and sporadic cases. Large epidemics have been recorded at Tampico in 1843 and in 1847. Merida, the capital of Yucatan, is still an endemic focus. In Mexico city, built at an elevation of 7450 feet, only imported cases of yellow fever have, it is stated, occurred.

Vigorous anti-yellow fever operations have been undertaken in the chief towns of Mexico, notably at Vera Cruz.

References For Yellow Fever in Mexico

Liceaga (E.)—"Yellow Fever in Mexico," *American Health Association Annual Convention*, 1893.

Boyce (Sir R. W.)—*Report upon Outbreak of Yellow Fever in British Honduras in* 1905, London, 1906.

Liceaga (E.)—"Yellow Fever in Mexico," *Transactions of the XV. International Medical Congress*, Lisbon, 1906.

Augustin (G.)—*History of Yellow Fever*, New Orleans, 1909.

Parker, Beyer, and Pothier—*Report of the Working Party No. I.*, Yellow Fever Institute, Washington, 1903.

Bolletim extraordinario del Consejo superior de Salubridad Mexico.

Manuel Carmona y Valle, Leçons sur l'étiologie et la prophylaxie de la fièvre jaune, Mexico, 1885.

Seidelin (H.)—"Actiologie des Gelben Fieber," *Berliner Klin. Wochen.*, *No.* 18, 1909; "Experiences in Yucatan," *Yellow Fever: Journal of Trop. Med.*, November 1910.

In British Honduras yellow fever was no doubt endemic in the early part of the seventeenth century at the time of its settlement in 1630.

Analysis of the Surgeon-General's returns show that in 1886 there were 17 cases; in 1887, 16 cases; probably some cases in 1889; in 1890, 19 cases; in 1891, 3 cases; in 1905, 40 to 50 cases.

Since 1905 no primary cases have been recorded in Belize. After that year anti-mosquito regulations were adopted.

References for Yellow Fever in British Honduras

Boyce (Sir R. W.)—*Report to the Government of British Honduras upon the Outbreak of Yellow Fever in* 1905, London, 1906.

Lawson (R.)—"Yellow Fever," *Transactions of the Epidemiological Society*, London, 1860, vol. iii.; also *Brit. and Foreign Med. Chir. Review*, 1862.

In Guatemala yellow fever is still probably endemic. At Livingston, a fruit port on the Atlantic side, a yellow fever epidemic was recorded in 1891; another in 1905; no doubt, however, sporadic cases have occurred in intermediate years. In 1905 severe epidemics occurred in the interior towns as Zacapa and Gualan, and isolated cases at Puerto-Barrios, and other small towns. The 1905 outbreaks were probably due to the opening up of the country by railroads, and in consequence of the increase of non-indigenous non-immunes. It is noteworthy that the disease went under the name "railway fever." At Zacapa, 700 cases were reported in 1905.

References for Yellow Fever in Guatemala

Boyce (Sir R. W.)—*Report to the Government upon the Outbreak of Yellow Fever in British Honduras*, 1905, London, 1906.

Reports of the Public Health and Marine Hospital Service, Washington.

In Spanish Honduras epidemics are recorded in the early part of the nineteenth century (Bérenger-Féraud), 1803, and later in 1850. But there can be little doubt that yellow fever

has been endemic from Columbian times (1502) to the present period. In 1905 commercial and railway extension took place and at the principal port, Puerto Cortes, yellow fever broke out in 1905, when 50 deaths were recorded. The new arrivals were the first to succumb. From the seaport the disease is supposed to have spread to the interior; one town—San Pedro—reported 621 cases.

References for Yellow Fever in Spanish Honduras

Boyce (Sir R. W.)—*British Honduras, loc. cit.; Washington Health Reports, loc. cit.*

Bérenger-Féraud (L. J. B.)—*Traité theorique et pratique de la fièvre jaune*, Paris, 1890.

In Nicaragua yellow fever commences to be recognised about the middle of the nineteenth century. Cases have been recorded at Bluefields, a small fruit port on the coast. Yellow fever was present in the interior in 1905. The disease is no doubt endemic.

References for Yellow Fever in Nicaragua

Boyce (Sir R. W.)—*Loc. cit.*

Bérenger-Féraud (L. J. B.)—*Loc. cit.*

Lawson (R.)—*Loc. cit.*

In Costa Rica—the chief seaport is Port Limon—yellow fever appears to have only commenced to attract attention since 1890, that is, since commercial expansion took place. Yellow fever is probably endemic. Outbreaks are recorded in 1901 and 1903, also numerous small outbreaks up to date (1910).

References for Yellow Fever in Costa Rica

Washington Health Reports, loc. cit.

Augustin (G.)—*Loc. cit.*

In Salvador.—This republic is on the Pacific side of Central America, and therefore more excluded from the lines of traffic common to the Atlantic side.

Yellow fever commences to be recorded about the middle of the nineteenth century, 401 cases being recorded in San Salvador in 1868. Small outbreaks and sporadic cases are of very frequent occurrence.

References for Yellow Fever in Salvador

Cornilliac—*Recherches chronologiques.*
Guzman—Thèse, Paris, 1869.

Panama, was no doubt until recent years an endemic centre of yellow fever. The construction of the Isthmian Canal, and the giving over to the Isthmian Canal Commission of the Isthmian Canal Zone, as well as the placing of other towns in the vicinity under modern sanitary supervision, has effected the complete disappearance of endemic yellow fever. As showing the immense strides in yellow fever preventive measures, it is worthy of note that the French in the first year of their attempt to construct a canal had a death-rate of 60·8 per thousand, the American Commission a death-rate of 15·8 per thousand employees. Since 1905 there have been no cases of yellow fever.

References for Yellow Fever in Panama

Public Health Reports, loc. cit.
Boyce (Sir R. W.)—*Health Progress, loc. cit.*
"Isthmian Canal Commission, 1908," *Annual Report. Dept. of Sanitation.*
"Isthmian Canal Commission, 1906," *Bulletin No.* 2. *Laboratory of the Board of Health.*
"Isthmian Canal Commission," *Reports of Health Officer*, 1905-1907 (Georgas, W. G.).
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CHAPTER II

HISTORY OF YELLOW FEVER IN SOUTH AMERICA

(From Eighteenth to Twentieth Century, chiefly Nineteenth Century)

In *Venezuela* yellow fever is still endemic, and has preserved in all probability an unbroken record from pre-Columbian times; numerous epidemics are recorded in the seventeenth, eighteenth, and nineteenth centuries. There was an epidemic at Ciudad Bolivar in 1907, and in the same year the disease is stated to have been continually present in La Guayra and in Carácas. Cases are also recorded in 1910.

In January 1910, the President of Venezuela created a Department of Hygiene in the Administration, to be under the immediate charge of a director, who should also act as secretary of the Superior Council of Hygiene and Public Health. Already the department has issued a memorandum to the public dealing with yellow fever prevention.

References for Yellow Fever in Venezuela

Boyce (Sir R. W.)—*Health Progress, loc. cit.*

Hirsch (A.)—*Geographical and Historical Pathology, loc. cit.*

Beauperthuy (L. D.) — *Travaux scientifiques de Louis Daniel Beauperthuy, Docteur en médecine des Facultés de Paris et de Carácas*, Bordeaux, 1891.

"Outbreak of Yellow Fever in Venezuela," *Boston Medical and Surgical Journal*, Oct. 1910.

In British Guiana.—Yellow fever was also no doubt endemic when it was first settled in the early part of the seventeenth century. It is not, however, until later in the eighteenth and

in the nineteenth century that yearly regular records of outbreaks amongst sailors and new arrivals generally are recorded.

In 1840 the mortality from yellow fever was such that in a few months 69 per cent. of all the white troops had perished. After this period there is a lull and outbreaks are not again recorded until 1881 and 1888, and since the last date no cases appear to have been chronicled. This improvement dates back to the sixties, when sanitary reform, just as in other countries, began to gradually banish the endemic type of the disease.

References for Yellow Fever in British Guiana

Blair (N.)—*Some Account of the Yellow Fever Epidemic of British Guiana*, London, 1858.

British Guiana Medical Annual, 1896.

Report of the Commission upon the General and Infantile Mortality, British Guiana, 1906.

Boyce (Sir R. W.)—*Health Progress and Administration in the West Indies*, London, 1909.

In French Guiana evidence exists that the fever which impeded the progress of the gold mining enterprises of 1871 was yellow fever. This disease is in every probability endemic. Numerous epidemics are recorded in the eighteenth century and also during the nineteenth century.

References for Yellow Fever in French Guiana

Cotholendy (F.)—*Quelques considérations sur les endémies de la Guyanne*, Thèse, Paris, 1857.

Esquerra (Carlos)—*Contribution à l'étude de la fièvre de Magdalena*, Paris, 1899

Esquerra (Domingo)—*Memorie sobre les fiebres Magdalena*, Magdalena, 1872.

Garnier (A.)—"La fièvre jaune à la Guyanne avant 1902, et l'épidémie de 1902," *Annales d'hygiène et de Méd. Col.*, Paris, 1904.

Jacquier (P. V.)—*Essai sur l'hygiène de la Guyanne française*, Thèse, Paris, 1835.

Dorvau (F. H.)—*Considérations sur l'hygiène et la pathologie des chercheurs d'or à la Guyanne française*, Thèse, Montpellier, 1876.

Gomez (Proto)—"Los mosquitos y la fiebre amarilla," *Revista medica de Bogota*, No. 15, 1887.

In Dutch Guiana the history of yellow fever is similar to that of the two preceding colonies: epidemics were common and

attracted attention from the end of the eighteenth century to the nineteenth century, and 15 cases were described in 1908 to 1909.

References for Yellow Fever in Dutch Guiana

Hirsch (A.)—*Loc. cit.*

Boyce (Sir R. W.)—*Health Progress in the West Indies, loc. cit.*

In Brazil when commercial development set in yellow fever came into prominence; thus in 1850, 4000 deaths from yellow fever were reported in Rio.

Numerous epidemics are also recorded in the sixties and seventies, and in the year 1894, 5000 died in Rio from that disease alone.

The following table shows the annual and monthly deaths from yellow fever in Rio from 1896 to the present time. The table also shows how as the result of anti-mosquito measures, taken in the year 1903, the disease has been stamped out, and is now no longer endemic:—

Deaths from Yellow Fever in Rio de Janeiro since 1896.

Year.	January.	February.	March.	April.	May.	June
1896	690	986	1433	557	171	34
1897	38	65	88	66	36	16
1898	19	116	310	278	178	82
1899	138	235	258	101	40	22
1900	57	49	89	50	21	13
1901	24	55	83	66	38	22
1902	48	86	223	218	208	128
1903	184	219	270	148	44	20
1904	5	7	9	8	10	4

Year.	July.	August.	September.	October.	November.	December.
1896	24	16	4	19	12	28
1897	5	2	1	...	4	4
1898	54	30	14	12	17	30
1899	17	7	14	11	19	35
1900	7	6	3	7	7	5
1901	19	5	15	16	9	10
1902	85	54	38	39	36	121
1903	16	12	4	4	3	4
1904	4	1	2	...	30	...

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Fernandez (S. G.)—A profilaxia da febre amarela no Para," *Revista Propagador da medicina natural e Beneficente*, August 1910.

Otto u. Neumann—"Studien über Gelbfieber in Brasilien," *Zeitschr. f. infects. Krankh.*, 1905.

Pedro Torres Caleira—*Mangrove Tree Destruction and Yellow Fever*, Rio, 1884.

Gouy (E.)—*Histoire de la fièvre jaune au Brazil*, Paris, 1884.

Ribas (E.)—*Instructions sanitaires tendant à éviter la Propagation de la fièvre jaune*, S. Paulo, Brazil, 1903.

Barreto, Barros, and Rodrigues—*Rapport des expériences faites à l'hôpital d'isolement de St Paul*, 1903.

Durham (H. E.), Myers (Walter)—*Notes on Sanitary Conditions obtaining in Para.* Yellow Fever Expedition. Liverpool, 1901; *Report of Yellow Fever Expedition to Para*, 1900. Memoir VII., Liverpool School of Tropical Medicine.

Thomas (H. W.)—*La sanidad publica de Iquitos*, 1905; "Report of the 1905-1909 Expedition to the Amazon," *Annals of Tropical Medicine*, vol. iv., Liverpool School of Tropical Medicine.

Publications of the Instituto Oswaldo Cruz, Rio de Janeiro, 1909.

Bartarelli (E.)—"Die Bekämpfung des gelben Fiebers in Rio de Janeiro," *Wien. Klin. Rundschau*, 1906, vol. xx., pp. 897-99.

Strain (W. Loudon)—*Yellow Fever; its Mode of Discrimination, S. Paulo Epidemic*, 1892-1893.

During the period of trade expansion, the disease spread from the older established and larger centres of commerce to every new settlement, either along the coast or into the interior. Yellow fever also appeared for the first time in surrounding republics, where before it had been overlooked or had not appeared; as fast, in fact, as these countries unfolded to commerce.

Whilst yellow fever has ceased to be endemic in Rio, it still retains its endemic character at various trade centres along the Amazon; but here also steps have recently been taken to get rid of it.

There is no doubt that both malaria and yellow fever have existed endemic side by side, especially in the towns along the river Magdalena. "Magdalena fever" is only another place name of yellow fever. Epidemics have been recorded in 1830, 1837,

1860-1865, 1870-1871, and 1884. Along the rivers Magdalena, Orinoco, and Amazon, yellow fever has been from the earliest times endemic, and to-day they remain strongholds.

From the preceding statements, it is clear that places exist in Central and South America where yellow fever is still endemic, much as it was when the Spanish Conquistadores landed. It will also be noticed that yellow fever does not again commence to be chronicled until the period when military operations, gold mining, industrial developments, railway and canal construction and steam-ship routes open up anew these countries.

From this period through the *nineteenth century*, there is a second great recrudescence of yellow fever, lit up from the old slumbering endemic foci, as well as imported afresh from other centres. This present century, in striking contrast, is marked by the curtailment or disappearance of yellow fever in consequence of anti-mosquito measures.

It will also have been noted that yellow fever did not remain confined to the endemic foci in Brazil, the Guianas, Colombia, Ecuador, and Venezuela in the nineteenth century. It also spread to other commercial centres in South America, where there is no evidence that the disease was naturally endemic. For example, epidemics are recorded in Peru in 1842, 1852, 1854, 1869. Cases were recorded at Callao in 1889. Similar outbreaks took place in the seventies in Monte Video and Buenos Ayres and Ascension.

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Hirsch (A.)—*Handbook of Geographical and Historical Pathology*, London, 1883.

Clemow (F. G.)—*The Geography of Disease*, Cambridge, 1903.

Sodré (A. A. A.) and Couto (M.)—*Das Gelbfieber, Spec. Path. and Therap. Nothnagel*, Wien, 1901, Bd. V.

CHAPTER III

HISTORY OF YELLOW FEVER IN THE WEST INDIES

WE can, I think, come to no other conclusion than that yellow fever was endemic in these islands at the time of their invasion by the Latin races in the sixteenth and seventeenth centuries. The regularity with which yellow fever broke out amongst the white newcomers in their settlements is proof of this.

In 1493 an epidemic is recorded at Isabella in St Domingo about the time of its foundation by Columbus.

In 1508 one in Porto Rico,
In 1620 to 1648 in Cuba,
In 1635 in Guadeloupe,
In 1648 in St Kitts,
In 1649 in Martinique,
In 1649 in Barbados,
In 1655 in Jamaica,
In 1665 in St Lucia.

After the seventeenth century, or the age of Latin military and missionary enterprise, yellow fever receded in prominence, until British military and commercial activity set in with the rise of British naval supremacy, then once more yellow fever comes into prominence. In the eighteenth, and earlier part of the nineteenth century, it constitutes the fever of the principal towns of the Islands, the West Indian "endemial" or "acclimatising" fever, which every newcomer was certain to get soon after his arrival. It attacked, in regular epidemic form, the troops which from time to time were sent out from England;

FIG. 3.—The Bed of a Water-course, made in concrete, to prevent the formation of puddles and the breeding of mosquitos. Trinidad, 1909. (From *Mosquito or Man?*)

[*To face p.* 14.

because the arrival, in an endemic centre of yellow fever, of from one to three hundred troops was sufficient to light up an epidemic in almost any year, the medical military records of the Windward and Leeward commands are of the greatest interest to the student from this standpoint. They show that yellow fever appeared in an unbroken line from 1815 to 1846 in the island of Barbados and at intervals to 1846. Similar records exist for the islands possessed by other nations.

A West Indian military report of the time states: "The arrival of a stranger at almost any time or season in the West Indies was sufficient to develop yellow fever." From these well-authenticated records we must conclude that yellow fever persisted in its endemic form amongst the regular inhabitants of the towns, be they whites, creoles, or blacks, until comparatively modern times.

In the modern period, the growth of hygiene bringing in with it cleaner yards and streets, and above all a pipe-borne water supply, did away with the old-time wells, barrels, and innumerable receptacles employed to store the rain, river, or well water. This reform (commencing about 1850) struck at the root of endemic yellow fever. It reduced the breeding-places of the *Stegomyia* to that point at which yellow fever ceases to be endemic.

From about the year 1850, however, small outbreaks of yellow fever occurred from time to time, not from the presence of an endemial disease but as the result of imported cases, or imported infected *Stegomyia* from distant centres where the disease was still endemic, viz., Venezuela and the Amazon. The imported infection caused an outbreak limited in extent solely by the local supply of *Stegomyia* and the number of non-immune inhabitants. These points were clearly brought out by investigations which I conducted in 1909 in the British West Indies, and by those which a French Commission conducted at the same time in Martinique and Guadeloupe.

In the case of Barbados, the fever attacked the coloured and

black population with more frequency than the white. This, I concluded, showed that endemial yellow fever had long ceased to exist, and that in consequence the majority of the dwellers in the island were non-immunes; it also showed that there was a sufficient supply of *Stegomyia* to give rise to numerous cases, provided that infection were imported from without. Investigation showed that this in every probability did actually occur in a crowded, poor district in Bridgetown, the principal town of the Island. In this particular quarter it was shown that the *Stegomyia* was breeding in abundance, and might have readily been infected from some mild or unrecognised case of yellow fever arriving from the Amazon, or other centre where there was yellow fever, and within the time limit of the incubation of the disease.

In the case of the other West Indian islands in which a few cases of yellow fever were recorded, the evidence likewise was all in favour of importation. In the case of the French islands, Martinique and Guadeloupe, the Commission inclined to the belief that the outbreaks in those islands were not due to imported yellow fever but to endemial yellow fever. This endemial fever was of such a mild type that it went under the name of "inflammatory fever," in consequence its true nature was not appreciated. From this mild form of yellow fever the mosquito became fully infected, and was able to communicate it in its virulent form to new arrivals, or to returning residents who had resided some time out of the colony. In support of their contention they showed that the islands since 1649 had been continually the seat of outbreaks of the disease down to the year 1895, and they contended that in the intervals in all probability, mild, unrecognised cases of yellow fever occurred, and that these kept up continuous infection in the *Stegomyia*. If we may assume the preceding reasoning to be correct, it shows how in the West Indies yellow fever was endemic to commence with, *i.e.*, about 1493, how it long continued to remain so, and how the magnitude and frequency of the epidemics

increased coincident with military and commercial expeditions and the growth of towns, until altered hygienic conditions commencing about the years 1850 to 1860 had in many of the islands brought about the cessation of the continuous connected outbreaks of yellow fever; had in other words stopped the flow of endemial yellow fever. These sanitary changes had not, however, brought about the complete destruction of the *Stegomyia*. In consequence we find localised and limited outbreaks of yellow fever down to the year 1909, due, in the majority of instances, to importation of infection from without.

In the case, however, of the Martinique and Guadeloupe epidemic, the recrudescence appears to have been due to the persistence in all probability of the original endemial yellow fever common to all the islands in the fifteenth and sixteenth centuries.

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KERAUDREN (P. F.)—*De la fièvre jaune observee aux Antilles et sur les Vaissaux du roi considéree principalement sous le rapport de sa transmission*, Paris, 1823.

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BLAIR (N.)—*Some Account of the Yellow Fever Epidemic of British Guiana*, London, 1850.

DIXON (NODES)—*Observations on the Inflammatory, Endemic, or Yellow Fever in the West Indies*, London, 1819.

WILSON (T. G.)—"Observations on the Yellow Fever Epidemic at Bermuda," 1864, *Lancet*, 1872.

The following are the chief historical data connected with yellow fever in the principal West Indian islands:—

Grenada.—Yellow fever is said to have been introduced in 1793 from West Africa, and in consequence was called "Bulam fever." It certainly could have readily been introduced; but in all probability the fever was naturally endemic to this as to other islands of the group. In 1818 the mortality amongst the troops is given as 21 per cent. The last cases, probably endemic ones, were recorded in 1881.

REFERENCES

BOYCE (Sir R. W.)—*Loc. cit.*
Annual Medical Reports of Grenada.

St Vincent.—Yellow fever is recorded regularly in the nineteenth century until 1822; it then fell back. In 1909 five cases were recorded, due, it is stated, to an imported case.

REFERENCES

The Second Report on Quarantine, Yellow Fever, London, 1852.
BOYCE (Sir R. W.)—*Loc. cit.*
Annual Medical Reports of St Vincent.

St Lucia.—In the year 1796, 800 troops perished of yellow fever in the year. It was very prevalent in the nineteenth century. In 1827 the mortality amongst the troops was stated

FIG. 4.—A Concrete Roadside Drain, Port of Spain, Trinidad.

[*To face p.* 18.

to be 21 per cent. No cases have been recorded in recent years.

References

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Levacher (M. G.)—*Guides des Antilles*, Paris, 1834.

Trinidad and Tobago.—Yellow fever was very prevalent in the nineteenth century up to 1818, when the mortality reached 30 per cent. in both islands. In 1821 it was 25 per cent. After this period no doubt isolated cases continued to occur. Thus in 1907 there were 50 cases; in 1908 150 cases; in 1909 1 case is reported. Yellow fever is, however, not now endemic, but Trinidad is very close to and in constant communication with Venezuela, which is an endemic focus.

References

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St Kitts or Christopher.—In the seventeenth and eighteenth centuries outbreaks are recorded.

References

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Antigua and Monserrat.—Yellow fever is recorded from 1817-1836. In the 1816 epidemic in Antigua it was noticed that no natives were attacked, which is proof that the disease was endemic.

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FURLINGE (G.)—"Report on Certain Cases of Yellow Fever, St John's, Antigua," *Lancet*, 1850.

In Dominica the death-rate in 1817 was 29 per cent. amongst the troops.

In Tortola, Nevis, and the Bahamas yellow fever occurred regularly in the nineteenth century.

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BLAIR (N.)—*Loc. cit.*

BOYCE (Sir R. W.)—*Loc. cit.*

Jamaica.—In 1780, 3500 deaths are recorded in a period of four years. From 1817-1847 the mortality was very high amongst the white troops, but almost nil amongst the black. After this period the cases became very much fewer up to the year 1889. There was an outbreak, however, with 38 deaths, in 1897-1898.

In 1899 there were some very suspicious cases diagnosed as remittent fever. In 1904 7 cases with 3 deaths were reported, and in 1905 there was 1 death from yellow fever in Kingston. The chief factors in putting an end to the endemic yellow fever which prevailed in the nineteenth century were a pipe-borne filtered water supply, first introduced in the year 1897, and increased attention to general sanitation.

The disease is now no longer endemic in Kingston, nor probably in any part of the island.

REFERENCES

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JACKSON (R.)—*A Treatise on the Fevers of Jamaica*, Philadelphia.

HUNTER (JOHN)—*Diseases of the Army in Jamaica*, 1788.

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Second Quarantine Report, 1852, "Dr A. Browne's Report."
BOYCE (Sir R. W.)—*Loc. cit.*, 1910.
Annual Medical Reports of Jamaica, especially Reports of Dr Donovan and Dr Mosse.

At *Port Royal* in Jamaica there is also evidence of an unbroken history of yellow fever from the commencement of the nineteenth century down to the year 1904. The record from the year 1878 is as follows:—

Year.	Cases.	Deaths.
1878	16	11
1882	14	6
1883	4	3
1885-6	22	6
1890	8	1
1891	3	3
1897	8	6
1900	1	1
1901	1	1
1902	1	1
1903	6	1
1904	1	...

Some of the above might have been imported cases.

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BLAIR (N.)—*Some Account of the Last Yellow Fever Epidemic of British Guiana*, London, 1850.
"Epidemic of Malarious Yellow Fever on Board the *H.M.S. Doris* off Port Royal, 1873," *Lancet*, vol. i., 1875.

Barbados.—Epidemics are recorded in 1647, and yellow fever was known as Kendal's disease in 1691. Then came the usual pause, followed in the nineteenth century by a continuous outbreak coincident with military and commercial expansion. The disease appeared for forty years in uninterrupted succession.

Yellow fever up to this period was endemic—about the middle of the nineteenth century, when sanitary water reforms took effect and epidemics became less in magnitude and frequency; one is recorded in 1881. Since then none are recorded until 1909, when the black and coloured population were more affected than the white, showing that the disease was probably not endemic but imported.

References

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Boyce (Sir R. W.)—*Loc. cit.*

Harrison (J. B.), and Moxly (J. H. S.)—*Reports upon Experiments connected with Yellow Fever*, Barbados, 1884.

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Cuba.—In 1762, during the siege of Havana, 8000 soldiers and sailors were stricken with yellow fever. The disease was known in 1620 as the "pest of Havana," and remained endemic there until 1890, and in other parts of the island until 1898.

In 1900 proof was experimentally furnished that the *Stegomyia* was the sole transmitting agent of yellow fever—a fact which in 1881 Finlay of Havana had also taught. In 1909 there was no yellow fever in Havana, due entirely to anti-*Stegomyia* measures. Yellow fever is in fact at the present time being driven out of the whole island.

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Faundleroy (P. C.)—"Some Notes on the Last Epidemics of Yellow Fever in Cuba," *New York Med. Record*, December 1909.

Puerto Rico.—The seat of epidemics in the nineteenth century, since when much has been done to get rid of endemic yellow fever.

References

Hirsch (A.)—*Handbook of Geographical and Historical Pathology.*
Augustin (G.)—*Loc. cit.*
Boyce (Sir R. W.)—*Loc. cit.*, 1910.

Martinique and Guadeloupe.—These islands have been visited by repeated epidemics during the seventeenth, eighteenth, and nineteenth centuries, and from a recent report there is reason to believe that yellow fever persisted in its endemic form to 1909, when a considerable outbreak occurred in Martinique, not as the result of importation. It was originally supposed that yellow fever was introduced into Martinique in the year 1649 from Siam, hence the name *maladie de Siam.*

References

Second Report on Quarantine, 1852.
Belot (C.)—*Fièvre jaune à la Martinique*, Rochefort, 1881.
Mervilleux (G.)—*Considérations sur l'hygiène des troupes à la Guadeloupe*, Bordeaux, 1887.
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Launoy (L.)—"Contribution à l'étude de l'épidémiologie amarile de Martinique," *Annales de l'Institut Pasteur*, Décembre 1909.

The Danish West Indies.—St Thomas, St Bartholomew's, and St Martin have been the seats of numerous outbreaks in the eighteenth and nineteenth centuries.

Reference

Hirsch (A.)—*Loc. cit.*

Hayti or Saint Domingo, Republic of Hayti and San Domingo.—Epidemics are recorded very many times during the seventeenth, eighteenth, and nineteenth centuries. At the present time the disease is confined to diminishing areas, and is not regarded as the serious disease of the island.

References

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CHAPTER IV

THE HISTORY OF YELLOW FEVER IN NORTH AMERICA

THE history of yellow fever in North America and in Europe is of great practical interest.

In the first place, it will be seen that the rise, progress, and disappearance of the disease in these regions is absolutely corroborative proof of the mosquito doctrine; and in the second place, it shows how disease may be carried; how it follows trade routes and commercial movements, but cannot establish itself permanently because certain conditions are not fulfilled. All these points have a very direct bearing upon the progress of the disease to-day. Admitting that the primary endemic focus of yellow fever in the New World is Central and South America and the West Indies, it can be readily understood how North America was probably invaded by the disease before Europe. The former, as we shall presently see, was comparatively close to the endemic centres of infection.

History in North America

Yellow fever reached North America in the middle of the seventeenth century—1668. That portion of North America which is now the United States, was then just colonised by British, Spanish, and French, and was dotted over with numerous small settlements, more especially along the Atlantic and Gulf coasts, or along the great waterways. The small settlements established during the last part of the eighteenth

century and the first half of the nineteenth century grew up with great rapidity into large commercial cities and ports.

In those days of rapid development and comparative poverty, overcrowding was the rule, and there was little attention paid to sanitation of any kind. The water was obtained from wells or roofs, therefore cisterns, barrels, and no doubt all kinds of water-containers were common. In wet weather puddles formed everywhere for there was no proper drainage. In other words, the conditions then prevalent were such as are now only to be found in some primitive West African or Central American coast town. These conditions would favour no doubt the propagation of the *Stegomyia* and probably enabled it, by means of the artificial warmth of the overcrowded houses, to survive through the cold season. Thus there was in those days a continuous supply of *Stegomyia*, brought about by artificial means.

Intercourse between the then British, French, and Spanish settlements in North America, and the corresponding settlements in the West Indies and Central America was constant by means of the old wooden ships of the period. We know, moreover, that the then proverbial unhealthiness of the West Indies and their perpetual condition of unrest, produced by frequent changes of government at home and rebellions within, drove the colonists to seek a safer and more settled home in North America, and English, French, and Spanish colonists migrated in their hundreds to the more peaceful North America. Then as soon as they established themselves, and prosperity increased, there arose the demand for labour, and the slave importation commenced. Given, therefore, the existence of the *Stegomyia*, we can see that they could not long remain uninfected.

For convenience, in order to illustrate the progress of yellow fever in North America, it will be simpler to divide the chief ports into those placed on the Atlantic side, namely:— Charleston, Baltimore, Philadelphia, New York, and Boston;

FIG. 5.—A Row of large Rain-water Cisterns, placed close up against the backs of the houses. They have been hastily protected by "cheese cloth," to prevent the breeding of the *Stegomyia*. New Orleans, 1905.

[*To face p.* 26.

and those in the south, known as the Gulf ports, viz., Galveston, New Orleans, Mobile, Pensacola, and Key West. The climatic conditions of the southern ports differed from those in more northern latitudes; the conditions were more favourable to the prolonged propagation of the mosquito carrier.

Nevertheless the conditions were never so favourable as those obtaining in the West Indies or Central America. In late autumn and in winter there was a cold period which tended to put an end to the breeding of the *Stegomyia*. Naturally, the further north the port was situated, the more effective did the inhibitary action of the cold become. The foothold of the *Stegomyia* in North America was in a position of unstable equilibrium; given a mild, frostless winter succeeding the usually intensely hot summer, and the *Stegomyia* could survive to the following year; but given a cold season and the *Stegomyia* must have perished in enormous numbers. Perhaps, however, not completely. For it must not be forgotten, even in countries where there is a cold season, provided the season is not too cold or too prolonged, that there may exist conditions of temperature in overcrowded houses in the slums of large towns, which enable the *Stegomyia* not only to survive through a winter but even to propagate, and thus to keep up the species from one year to another. But under these conditions sooner or later the chances are that a colder winter than usual will bring the species to an end.

Not only in the Gulf ports like New Orleans, but also in the Atlantic ports like New York, the evidence furnished by the outbreaks points to the survival at that period, not only of infected adult *Stegomyia*, but also to the survival of the species, as we shall presently see. But at all times, under these conditions, the survival of the mosquito was exceedingly doubtful and a matter of accident, and this explains why yellow fever could not get a prolonged endemic foothold in the Atlantic towns; and if by the accident of favourable seasons the species bred for many years in succession in the Gulf towns, it was every

severe cold season reduced enormously in numbers, so that although there might not be enough mosquitos present to keep up the endemic character of the disease, yet sufficient existed to give rise to local outbursts when a case of yellow fever was imported from some locality where that disease was present.

Galveston in Texas.—From the year 1839 epidemics of considerable size were common down to the year 1867. In 1839, when the population of Galveston was only 5000, the number of deaths from yellow fever is given as 250. In 1867, when the population was 22,000, the deaths from yellow fever were 1150. From the constant manner in which epidemics succeeded one another, it is very probable that the disease gained an endemic foothold. On the other hand, Galveston was a port very liable to continuous infection from without.

Since 1870 the disease has not appeared in epidemic form, but histories of imported cases have been frequently recorded.

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KEATING (J. M.)—*History of a Yellow Fever Epidemic of* 1878, *in Memphis, Tenn.*

LA ROCHE (R.)—*Yellow Fever*, Philadelphia.

New Orleans.—There is every reason to believe that this city, during its very early history and occupation by the French and Spanish in the eighteenth century, was regularly visited by yellow fever. Not, however, until the city enlarged in the nineteenth century, as the result of commercial development in connection with cotton and sugar, did yellow fever attain the proportions that rendered it notorious. From the year 1822, probably from a still earlier date, there is an unbroken record of outbreaks and epidemics until 1860. During those thirty-eight years we may safely assume that yellow fever had become endemic, that conditions existed in the slums of the old Gulf

port, which enabled the *Stegomyia* to tide over the winter season.

After 1860 there was a fall in the annual mortality from yellow fever, but outbreaks were recorded in 1867, 1874. In 1878 an epidemic occurred, with a record of 4046 deaths; in 1897, 298 deaths took place. Between this date and the last epidemic in 1905 the notification of cases of yellow fever was frequently made. So that there is the possibility that from 1822 to 1905 the disease existed in an endemic form in the city. During this period, however, there were no doubt countless opportunities for imported infection by ships arriving from Havana and other infected ports.

References

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Augustin (G.)—*History of Yellow Fever*, New Orleans, 1909.

Boyce (Sir R. W.)—*Yellow Fever Prophylaxis in New Orleans*, 1905; Memoir XIX., Liverpool School of Trop. Medicine.

Thomas (P. F.)—*Traité pratique de la fièvre jaune observée à la Nouvelle Orleans*, Paris, 1848.

Report of the Commission of New Orleans on the Epidemic of Yellow Fever of 1853, New Orleans, 1854.

Faget (J. C.)—*Fièvre Jaune*, Paris, 1875.

Mobile, Alabama.—From the end of the eighteenth century into the early part of the nineteenth century, and at a subsequent period, there is evidence in favour of yellow fever having been endemic for a number of years. It has long ceased to be endemic.

Pensacola, Florida.—Here also there is evidence in the nineteenth century of endemic yellow fever existing for a number of years. There was a considerable outbreak in 1905.

Key West, Florida.—The seat of constant small outbreaks in the nineteenth century.

From the preceding evidence there is much in favour of yellow fever having gained a temporary endemic foothold in the Gulf ports. Climatic conditions to commence with, and

subsequently the introduction of stricter hygiene and better water supplies, turned the scale against the *Stegomyia*, and the endemic character of the disease disappeared, although small outbreaks continued to occur at intervals.

Atlantic Ports—Charleston, South Carolina.—Yellow fever was of constant occurrence in the nineteenth century—so regular, that in all probability it was for some time endemic.

Baltimore, Maryland.—As has so frequently happened in other parts of the world where yellow fever is liable to occur, there has long been present in Baltimore a fever which was known as *bilious remittent*, but which, there is now good reason to suppose, might have equally well been a mild form of yellow fever. Other significant facts recorded are that the bilious remittent fever originated in and usually remained confined to the squalid, overcrowded, exceedingly ill-kept quarters in the port, and that the abundance of mosquitos was a matter of comment, and that the physicians of the period were in favour of the local rather than the imported origin of the disease.

On the whole, the evidence is in favour of yellow fever having been endemic for a number of years, the *Stegomyia* persisting in the overheated, overcrowded slums close to the wharves. One of the earliest and most severe epidemics occurred in 1794, when there were 360 deaths; another severe epidemic took place in 1800, when there were 1197 deaths. Through the eighteenth century small outbreaks or sporadic cases were repeatedly recorded up to the year 1876.

References

Morris (J.)—*Transactions American Public Health Association*, 1877-1878.

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Augustin (G.)—*History of Yellow Fever*.

Philadelphia.—This city also furnishes another proof that conditions existed at the end of the eighteenth century and through the nineteenth century—in fact, from its foundation—

that allowed the *Stegomyia* to persist from one year to another, for there can be little question that yellow fever for a period became endemic, viz.—

1699	220	deaths
1741	240	„
1793	4044	„
1797	1292	„
1798	3506	„
1799	1015	„
1803	3900	„

These very high death-rates signified that, owing to the constant tide of immigration at the period, there were always present non-immunes in large numbers. For this was the period when the little settlement of 3000 was growing into a great city of 100,000, in the first decade of the nineteenth century. This was also a period when wells abounded and drainage hardly existed, and refugees were pouring in from the disturbed West Indies. It was also noted at this period that whilst the newcomers from Europe contracted the disease, those from the West Indies remained free—no doubt, because they were immune, having already had the disease.

References

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Rush (B.)—*Medical Enquiries and Observations*, Philadelphia, 1809 (4 vols.); consisting of essays on the non-contagiousness of yellow fever and accounts of various epidemics and sporadic cases, also other works by the same author.

Second Report on Quarantine, 1852. Gillkrest on opinion of physicians of America on yellow fever.

Rush (B.)—*An Account of the Bilious Remittent or Yellow Fever as it appeared in Philadelphia in* 1793, Philadelphia, 1794.

New York.—During the growth of this town from a village to a metropolis, commencing at the end of the seventeenth century and passing through the eighteenth and nineteenth centuries, there is a long record of outbreaks.

Some of the epidemics were of large dimensions. In 1668

there were 370 deaths; in 1803, 606 deaths; in 1856, 538 deaths. In 1870 an outbreak also occurred. The disease, as in the case of Baltimore, possessed a focal character, indicating that in a particular locality conditions existed for the survival and even multiplication of the mosquito. In the case of New York, the evidence is not conclusive enough to enable it to be said that yellow fever had gained an endemic foothold.

It can be said, however, that conditions existed which made it possible for the *Stegomyia* to multiply in large numbers in certain localities (see *ante*), so that if yellow fever was introduced it could readily spread.

REFERENCE

AUGUSTIN (G.)—*Loc. cit.*

Boston.—In this town the history of yellow fever shows that the outbreaks were comparatively small, and for the most part limited to the ships, and in all probability to the immediate neighbourhood of the shipping. It shows that the imported *Stegomyia* was not able to gain a continuous foothold, but that in certain favourable seasons it was capable of multiplying, and therefore capable of giving rise to a localised outbreak, much as in the case of the seaports in Europe.

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CHAPTER V

HISTORY OF YELLOW FEVER IN EUROPE

THE appearance, progress, and disappearance of yellow fever in Europe in the eighteenth and nineteenth centuries teaches us the same instructive lesson as did North America. For the same reason as in that continent, and from much the same causes, yellow fever gained a series of temporary footholds. It is somewhat doubtful, however, whether it ever became endemic; but it certainly was often epidemic and raged with great violence, and the outbreaks succeeded one another with great frequency. The causes which led to the appearance of the disease were those which operated in North America—growth of commercial intercourse, and the emigration of colonists to the West Indies, Central America, and Brazil, at first chiefly derived from the Peninsula and Italy, then from France, and later on from North Europe. As the Latin races had been first to colonise in the New World, so they were the first, when returning home, to import yellow fever into the Old World.

In the eighteenth and nineteenth centuries the hygienic conditions of the chief ports of Spain, Portugal, Italy, and the South of France were very primitive. As in North America, so here, there existed overcrowded filthy quarters, to be found in the old South European ports close to the shipping; and from observation of to-day it is easy to understand how readily the *Stegomyia* could have swarmed in the warm months of the year, and how even they could have survived through the winter time, in the hot, overcrowded houses in the poorer parts of the

seaports. Infection was then of almost daily occurrence, for each sailing vessel returning from Cuba and the West Indies, or Central America and Brazil, was almost certain to have cases of yellow fever on board, as well as numbers of infected *Stegomyia;* so that although a considerable distance existed in time and space between the infected foci and the Spanish ports, nevertheless the constant arrival of infected ships brought the endemic foci very close to Europe.

There appears to be considerable evidence that yellow fever was known as early as the sixteenth century in Spain, epidemics of what might have been the disease being recorded in 1501, 1515, and 1589. But in order to marshal the well-authenticated facts, I will describe the progress of the disease in Cadiz, Málaga, Cartagena, Gibraltar, Majorca, Lisbon, and Barcelona; Leghorn in Italy; Marseilles, Brest, St Nazaire, in France; and Swansea and Southampton in England.

It is quite possible that yellow fever might have been brought in in the sixteenth century with the ships returning from the New World in that century.

References

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FERMON (Dr DE), *Révue critique*, Paris, 1829.

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Reports on the Pathology, Therapeutics, and General Ætiology of the Epidemic of Yellow Fever which prevailed in Lisbon, 1857, *Brit. and Foreign Med. Review*, vol. xxv., 1860.

Cadiz.—Yellow fever is stated to have broken out in 1730, 1731, 1736, 1764, 1800, 1802, 1805, and 1810. Investigations were made into the nature of the epidemics of 1730-1731. Inquiry into the 1810 epidemic showed that in the six

outbreaks previous to 1805 the original source of infection could not then be traced. In 1761 an official report stated that from the experience of the trade relationship between Havanna and Cadiz, yellow fever could not be regarded as a contagious disease. Other epidemics followed in 1813, 1819, and 1821; the latter date appears to be the last of the epidemics.

Devèze, in his traité, gives an account of the symptoms of the epidemic of 1800.

References

Villalba—*Epidemiologia espanola.*

Gillkrest in *The Report on Quarantine*, 1852.

Devèze (Jean)—*Traité de la fièvre jaune*, Paris, 1820.

Arejula (J. Manuel de)—*Breve descripcion de la fiebre amarilla padecida en Cadiz y pueblos camarcanos en* 1800, *en Medinasidonia en* 1801, *en Malaga en* 1803, *y en esta misma plaza y varios otros del regno on* 1804.

Fellowes (Sir James)—*Reports on the Pestilential Disorder of Andalusia, at Cadiz*, 1800, 1804, 1810, 1813, London, 1815.

Málaga, Cartagena, and Passages.—These are also South Spanish ports which were frequently infected in the eighteenth and nineteenth centuries by returning ships. Therefore, as at Cadiz, the *Stegomyia* must have found suitable conditions for propagation. Outbreaks of black vomit are chronicled at Málaga in 1741, 1803, and 1810; but even at an earlier date (1678-1688) the port was visited by an epidemic which might have been yellow fever.

The ports of Cartagena and Valencia are said to have been visited by an epidemic in 1648, and epidemics are recorded at Cartagena in 1804-1812. Yellow fever broke out at the little port of Passages; it is stated that at that time the houses in Passages were crowded, filthy, and badly ventilated. Previous outbreaks are stated to have occurred in 1780 and 1791, and even in the periods 1808-1809 and 1813-1814 both English and French troops quartered there are said to have suffered from a fever which might have been yellow fever.

References

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Audouard (F. M.)—*Relation historique et médical de la fièvre jaune qui a régnée à Barcelona en* 1821.

Barcelona.—The history of yellow fever does not go so far back as in the case of the other seaports, but it gained in intensity in the nineteenth century, related without doubt to the very rapid commercial progress of that seaport. Outbreaks were recorded in 1803 and 1810. The great epidemic occurred in 1821, and attracted at the time investigators from many countries—French, British, and American.

The disease was imported by ships returning from Havanna, and the history of its development is that of all similar towns—at first the shipping people and those who visited the ships, then the dwellers in surrounding houses. The population of Barcelona was then 150,000, of which only 70,000 remained in the port during the fever, and of these 20,000 are stated to have died of the disease. In 1870 and 1883, small outbreaks occurred amongst the crew and workmen discharging infected ships in the port.

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Palma (in Majorca) was infected during the great epidemic of Barcelona, which swept over Southern Spain. Previous invasions were recorded in the early part of the nineteenth century.

Gibraltar.—The epidemics of yellow fever which once prevailed at this military station attracted a great deal of attention in the early part of the nineteenth century, and there are numerous official reports and documents available in connection with it.

The history of yellow fever at Gibraltar has a very great scientific interest, for it tends to show that that disease had actually become endemic for a time, just as we have seen in the case of certain Gulf ports in the Southern States of North America. If old accounts are to be trusted, yellow fever appeared as early as 1649. In 1727 there was an epidemic of doubtful nature, of which 500 of the garrison died.

In 1798 and 1799 a disease precisely similar to that well known in the West Indies produced great mortality amongst the newly arrived regiments. In 1800, 257 deaths occurred from the same fever in the garrison. In 1804 a severe epidemic occurred: of a population of 15,000, 4864 civilians and 869 soldiers died.

In 1813 there were 899 deaths. In the intervals between the epidemics from 1800, it appears upon the statement of Gillkrest (*loc. cit.*) that sporadic cases of the disease were of annual occurrence. If the observations were correct, it would appear that the disease had gained an endemic foothold. It is worthy of note that it *was observed that the progress of yellow fever was stopped, as it is always found to be, by the setting in of a cold wind from the North.*

A military port like Gibraltar was, it must be well understood, extremely liable to infection from without, not only by troopships returning from the West Indies and Africa, but also by ships arriving from infected ports in Spain.

The 1828 epidemic was remarkable for having brought to a head the fierce controversy which raged at the time as to whether yellow fever was contagious or non-contagious. It is obvious that experienced observers and clinicians, not suspecting that the disease was mosquito-carried, were all the time at a complete loss how to explain fully the sudden appearance, curious mode of spreading, and sudden disappearance of the disease. The majority of observers were certain, as will be shown in another chapter, that the disease was not contagious. On the other hand, great observers were equally certain that

the disease could be carried from point to point by infected persons. Both schools of investigators were correct in their facts; the missing link in the contradictory views was the unsuspected mosquito, as we shall see. In this epidemic it was noted by Gillkrest how, over and over again, those members of a family who nursed those of them who were sick very often escaped; wives slept with their infected husbands, and mothers with their sick children, yet they by no means always contracted the disease.

The non-contagionists laid great stress upon the local origin of the disease, a belief which has survived even up to the present time in many parts of the world. It was also noted how those of a family living in the upper storey of a house often escaped, whilst those on the ground floors became infected. The want of complete protection afforded by isolating the non-infected from the infected was also often observed during the progress of the epidemic. The non-contagionists laid stress upon the fact that in the epidemics in Spain the advent of cold weather caused the outbreak to decline; they reasoned that the heat favoured the local or miasmatic cause. They further noted how yellow fever clung to certain localities and even houses; then in despair Gillkrest exclaims, "It must be confessed by all unprejudiced persons, that the closest investigations have failed in the discovery of the *sine qua non* cause of yellow fever. And, as for the contagionists," he added, "they may dwell on the exotic origin of yellow fever, but they have always a secret feeling that its development is dependent upon an assemblage of circumstances. In a few words, we may say with Arejula, while the days lengthen and the sun approaches our hemisphere, we may be sure that the fever, which has so afflicted us of late years, will not attack us; but when that luminary begins to retire from us, and during the whole period of its retiring, we may fear it, especially if we have had a sterile season, and if hot and dry winds have prevailed for many days successively;" and with many more arguments of the

same nature the contagionists and non-contagionists groped along until the *sine qua non* cause was discovered many years afterwards.

The following note by my colleague, Professor Robert Newstead, is of interest because it shows that the *Stegomyia* still survives (1910) in Southern Europe, and clearly demonstrates how it was possible for large epidemics of yellow fever to break out in Southern Europe.

"*Stegomyia fasciata* (*calopus*)" *in Malta*

"The mosquito was decidedly the commonest species which frequented the dwellings of man in Valetta during the months of July, August, and the beginning of September, and was found to be more domesticated than either *Culex fatigans* or *C. pipiens*. Its favourite breeding-places are shallow receptacles in yards and gardens, tanks, fire-buckets in hospitals, or in any receptacle holding water which had been left unchanged for a few days. A vessel of water left in a bedroom for a few days was found to contain a number of larvæ of this mosquito; these subsequently produced imagines, so that there can be no doubt as to the identity of the insect.

"The commoner species of mosquitos met with in Malta are—

(1) *Culex pipiens*,
(2) *Culex fatigans*,
(3) *Stegomyia fasciata*,

of which the last named formed 16·5 per cent. of the captures.

"*Culex pipiens* is by far the commonest species, and was found breeding in enormous numbers in cesspools. It was rarely seen in the main dwellings, being much less frequently seen in such places than either the *Stegomyia* or its near relative, *Culex fatigans*.

"The *imagines* make their appearance in some numbers usually at the end of May, and are said to be most plentiful during the month of June. In the year 1906, for some unaccountable reason, this mosquito was rarely found until the month of August; but during my stay in the island (1910) it

was present during the months of July, August, and September. I was informed on good authority that *imagines* also occur during the winter months, so that in all probability this species hybernates in the adult stage during the cooler months of the year, a habit common to many species in the colder portions of Europe. I can find no records of its occurrence in the larval stage during winter, but it is just possible that they do so, seeing that there are no frosts in Malta, though the temperature may be very low at times."

When trade routes became diverted from the southern parts of Europe to the more northern, we find yellow fever in France penetrating to the ports of Brest in 1802, l'Orient and St Nazaire in 1861, and even in England, at Swansea, in 1865. These outbreaks, however, are strictly limited to the ships which brought the infected cases or the infected *Stegomyia.*

It is not a question of the *Stegomyia* breeding in any of these seaports. The climatic conditions render that impossible. A ship coming from a yellow fever centre in the West Indies, or from Brazil, came into port. Infected *Stegomyia* were living on board just as they would have done in the West Indian town from whence they came. In other words, the ship was but a floating infected house, the conditions on board being suitable to the developmental cycle of the *Stegomyia*, both as regards temperature, water and food supply, very much as on shore in a tropical town. If, therefore, labourers or visitors went on board and moved about the interior of the ship, or commenced to work the cargo in the holds, they naturally exposed themselves to infection, and an outbreak of yellow fever took place, not from yellow fever contracted in the town, but from the bites of the infected *Stegomyia* on the ship. When the ship left the port nothing was heard of the disease, unless by some stray chance an infected *Stegomyia* had escaped from the ship to an adjacent goods shed or labourer's cottage close by the wharf, when of course it might equally well infect those whom it bit.

In 1909 an outbreak occurred at St Nazaire, due to infected *Stegomyia*, and, like all previous ones, limited to the ship and those who come in contact with it, and exposed themselves to the bites of the infected *Stegomyia* which had been taken on board at Martinique during the epidemic in that island in 1909.

CHAPTER VI

HISTORY OF YELLOW FEVER ON SHIPS

A CONSIDERATION of the mode in which yellow fever is carried shows that it is par excellence a disease especially suitable for transport by ships, especially by the sailing-ships common in the fifteenth and sixteenth centuries.

The history of yellow fever shows that it was as a matter of fact one of the most frequent of the diseases common to ships in those centuries; so common that "ship's fever" or "yellow jack" were the familiar names for yellow fever.

Moreover, because yellow fever was of such frequent occurrence on ships when far out at sea, a few medical men to-day reason that the disease cannot be mosquito-carried, but must be due to some miasm arising from waterlogged ballast or special cargoes, or from the bilge. As a matter of fact we know that the *Stegomyia calopus* is very frequently found on ships, and that it can be transported alive with great ease over long distances. Given a ship which has to remain against a wharf, or alongside of which lighters come off from the shore to discharge and load, it has been proved that the *Stegomyia*, in common with other mosquitos, readily enters the ship. It is for this very reason that ships trading with mosquito-breeding ports are now being screened, and that the officers usually sleep under mosquito nets.

Careful and systematic search has been made of the holds, engine room, galley, sleeping and living quarters of the crew and officers of ships arriving from mosquito coasts, and the

Stegomyia has been repeatedly found. Eighty-two vessels coming from ports infected with *Stegomyia* were subjected to careful examination, and in spite of the fact that the voyage had lasted from 13-20 days. The *Stegomyia* was found by Dr Grubbs in three cases; Dr King of St Lucia also found living *Stegomyia* in ships coming from Barbados in 1909, Dr Durham on ships trading on the Amazon. I met with a similar instance in the case of a steamer which sailed from Belize in 1906.

Therefore it is beyond dispute that living *Stegomyia* may be carried great distances over sea by means of ships. In the old days of sailing-ships there was, however, another factor which made it a matter of certainty that ships trading in the tropics not only could transport *Stegomyia*, but also *breed* them during the voyage.

In the sailing ship days it was not a question of condensation water, it was imperative to store drinking water in numerous casks and tanks for the long voyage. There were no artificial mineral waters; reliance had to be placed on the storage tanks and casks, or the rain water collected in the sail during the voyage.

But this class of water is precisely the kind in which the adult female mosquito deposits her eggs. Therefore, if a mosquito was once introduced on board, it could propagate freely; further, as very often the stock of water was renewed at the ports of call in the *Stegomyia* zone, both larvæ and eggs of *Stegomyia* were no doubt taken on board together with the water.

For practical purposes, in the old-time wooden ships, the *Stegomyia* found as satisfactory a breeding-ground as in any village on shore, and if anything the food supply, *i.e.*, man's blood, was nearer at hand than on shore. No wonder then that yellow fever was the *ship's fever* of the eighteenth and nineteenth centuries, and that the history of yellow fever abounds with innumerable instances of the then supposed or real transmission of yellow fever from one port to another. The slave-ships were

very rightly looked upon with great suspicion, and one writer went so far as to state that yellow fever was the price which Europe paid for the slave trade. No doubt the old slave-ships were infested with *Stegomyia.*

The Frigates were also especially prone to foster yellow fever and the naval medical records teem with innumerable instances of the outbreaks of yellow fever on board. Indeed, so bad did the reputation of some ships become in the service, that they had to be ultimately abandoned and scuttled, for it was thought that the yellow fever miasms actually clung to their timbers.

Another factor which greatly conduced in the sailing-ship days to mosquitos getting on board, was the length of time which the ships remained in ports to load and unload. Since steam iron ships have replaced wooden ships, the danger of carrying infected mosquitos has greatly lessened, and still less of course is the chance of breeding them on board, for the drinking water is to a large extent obtained by condensation.

In consequence, yellow fever is now comparatively rare on ships, instead of the rule.

This fact alone proves very conclusively the relationship of the *Stegomyia* to yellow fever. In spite, however, of its rarity, yellow fever does from time to time break out on board ship. It is of course obvious that a passenger or member of the crew may leave an infected port with infection, and the disease may develop in transit; such a case corresponds to an imported case on land. But if there are *Stegomyia* on board, they can spread the disease from the infected person to others on board, or infected *Stegomyia* taken in at the port of departure may live for weeks in the ship, and when finally disturbed at the end of the voyage, or during transit, start an outbreak. There are innumerable instances of the latter method of outbreaks. Examples are found at Marseilles, Brest, Saint Nazaire (1909), Swansea, etc., etc. (see also preceding chapter).

The following are a few of the chief outbreaks of yellow fever on ships. In 1726 a great outbreak occurred on

our fleet lying off Porto Bello, from probably yellow fever. In 1741 an outbreak on the fleet under Admiral Vernon off Cartagena; again in this same fleet off Porto Bello in 1742. In 1776 in Spanish ships *Ayel* and *Astrea* on their way out to the West Indies. In 1783 an outbreak on the Spanish fleet. In 1785 on a Spanish ship. In 1793 on a Spanish cruiser bound from Cadiz to the West Indies, and also on a Spanish squadron. In 1794 on two British war-ships. In 1801 on board a ship carrying Irish emigrants to New York. In 1802 on a French fleet bound from Tarentum to St Domingo. In 1803 on a ship from Portsmouth to New York. In 1802 on the Spanish fleet. In 1807 in a French squadron in the bay of Cadiz. In 1814 in a squadron of Spanish cruisers. From this date to the middle of the nineteenth century, on innumerable occasions upon crusiers and ships trading in the West Indies and on the West African coast. After 1850, steamships replaced the sailing ship, and later on iron replaced wood, and from that time yellow fever became rarer and rarer on board ship; but even to-day occasional outbreaks are recorded during the passage or on arrival at port.

It will be noticed that in many instances yellow fever broke out on the departure of ships from a northern port. This is explicable by the ship having previously come from some yellow fever port, such as from the West Indies. The infected *Stegomyia* could remain alive and infectious for several weeks, and perhaps months in the cabins and galleys.

In order to obviate the risk of infected *Stegomyia* getting on board ships, the latter should be anchored as far off wharves as possible, compatible with business requirements, and should remain as short a time as possible.

Those living on board should also be all provided with mosquito nets: some of the latest ships are provided with wire gauze screens to the ports, allyways, and ventilators. It is also proper to fumigate all parts of the ship before leaving the port, to make certain that all infected *Stegomyia* are killed.

Experience shows that neglect of this rule invariably sooner or later leads to the spread of yellow fever. I know of one instance where a Central American ship was fumigated, with the exception of the engine-room: the consequence was that yellow fever developed on board when the port of arrival was reached.

Railway carriages may also transport infected *Stegomyia*. It is well known how outbreaks of yellow fever are liable to spread to the towns along a railway line. Therefore if it is not possible to keep the carriages outside an infected town, they should be fumigated before starting on their journey.

Cargoes and passengers' baggage may harbour mosquitos. The danger is not great, but instances have been met with, so that there is just a possibility. Much, however, will depend upon the nature of the merchandise and where it was packed, for it stands to reason that it is not easy to entrap a *Stegomyia* in a bale of goods.

Lighters.—For *Stegomyia* in lighters, see under "Trade Routes and Yellow Fever."

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CHAPTER VII

HISTORY OF YELLOW FEVER ON THE WEST COAST OF AFRICA [1]

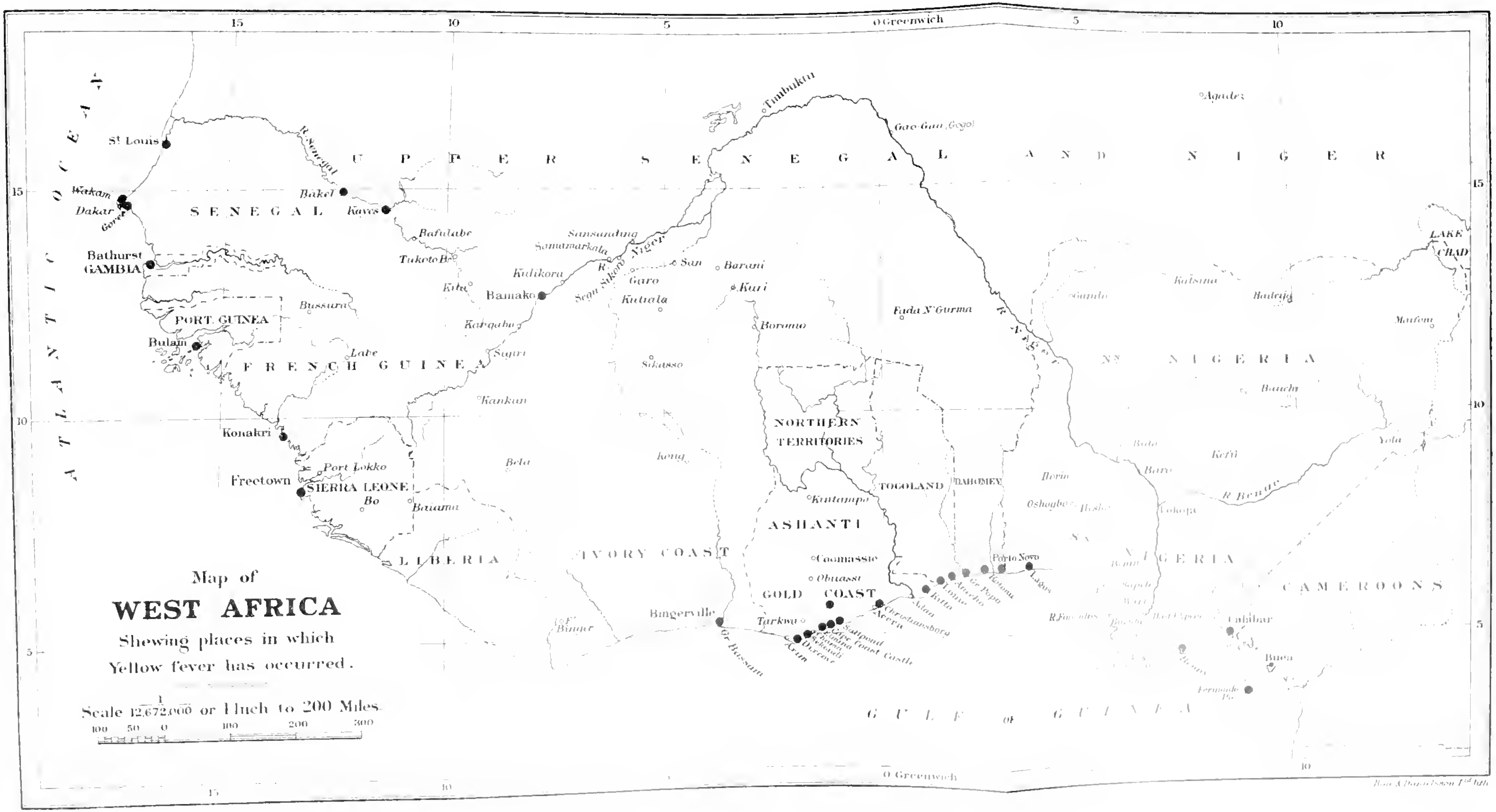
General. Yellow Fever in Sierra Leone. Yellow Fever in Southern Nigeria. Yellow Fever on the Gold Coast. Yellow Fever in the Gambia. Yellow Fever in Senegal. Yellow Fever in Dahomey. Yellow Fever in Togoland. Yellow Fever in Ascension and Boa Vista.

General

THE conclusion arrived at concerning the question whether yellow fever was first endemic in the West Indies or in Central and Southern America, was that yellow fever was endemic amongst the early inhabitants of both places. When the even more fragmentary history of yellow fever in West Africa is examined, we will, I think, come to a similar conclusion as regards the West African continent, viz., that yellow fever was in all probability a disease endemial to the native races of the coast. West Africa did not attract military, or missionary, or even commercial expeditions at so early a date as did the more attractive New World. It is not until the eighteenth century that information begins to filter home of the deadly African fevers of the coast, of "Bulam fever" (1793), of the "fever of Fernando Po," of the "fever of the Bight of Benin," etc. Amongst the earliest records are those relating to the presence of yellow fever in St Louis in 1778.

The fact that yellow fever has persisted in unbroken line from the eighteenth century to the present day appears to me to be the strongest evidence in favour of the essentially

[1] Reprinted by kind permission from the *British Medical Journal*, 1911.



Map of
WEST AFRICA
Shewing places in which
Yellow fever has occurred.
Scale 1/12,672,000 or 1 Inch to 200 Miles.
ATLANTIC OCEAN
UPPER SENEGAL AND NIGER
SENEGAL
GAMBIA
PORT. GUINEA
FRENCH GUINEA
SIERRA LEONE
LIBERIA
IVORY COAST
GOLD COAST
ASHANTI
NORTHERN TERRITORIES
TOGOLAND
DAHOMEY
NIGERIA
CAMEROONS
GULF of GUINEA
LAKE CHAD
St Louis
Dakar
Bathurst
Bulam
Konakri
Freetown
Bakel
Kayes
Bamako
Timbuktu
Bingerville
Tarkwa
Accra
Porto Novo
Lagos
Calabar
Buea
0 Greenwich

endemial character of the disease. Also many of the earlier military writers on yellow fever adopted this view.

In the case of the sister disease, malaria, we do not discuss whether it was imported into West Africa or whether it was endemic. We regard it as a disease essentially endemic to those peoples living amongst Anophelines; similarly, having regard to the very widespread distribution of the *Stegomyia* in Africa, we may reasonably assume that yellow fever has existed so long, that it may reasonably be regarded as endemic. As in the case of malaria so in yellow fever the infection may, of course, have been introduced, but introduced at such an early period that the question whether imported or endemic is beside the mark. We know that there still are countries, as the East Indies, in which the *Stegomyia* abounds, but in which the disease has so far not been signalled.

We also know that in the eighteenth century ships could have readily at any time introduced the disease into West Africa, for in those days the ships were exceedingly few which did not regularly carry infected *Stegomyia* and patients suffering from the fever. The tables were turned when Grenada accused Bulam, in West Africa, for having introduced the *nova pestis*, as they termed it, into the West Indies.

The story is instructive, because it shows that at that period great confusion had already arisen as to whether yellow fever was contagious or not; this same confusion has persisted to the present period in West Africa. Dr Chisholm maintained that up to 1793 yellow fever was a miasm fever, and, therefore, non-contagious; but that in that year the ship *Hankey* introduced "a new plague," as he called it, into Grenada for the first time, from whence it spread to the other islands, and from them to America and Europe.

In the eighteenth century the slave-ship was no doubt one of the most powerful factors in the distribution, not only of yellow fever but of all other racial and endemic diseases and of the insect carriers peculiar to them. Not only did the slave-

ship carry human beings in whose blood might have been the virus of yellow fever, malaria, sleeping sickness, relapsing fever, filariasis, plague, etc.—it equally well served as the means of transport of the various species of mosquito, fly, or flea. Some of these might have been taken on board infected; others we know could have developed on board ship; for, in all probability, every cask of water taken on board at a tropical station was already infected with the ova or larvæ of the *Stegomyia*. The "slaver" was a floating native village, in which the worst features of the native village were reproduced, white and blacks living jammed together in hot stifling quarters, providing the ideal conditions for the multiplication of the *Stegomyia* and the spread of yellow fever. The slave-ship justly earned its reputation of being the great cause of the dissemination of diseases, and now in the light of modern discoveries we more strongly than ever realise the truth of this statement.

The consideration of the following records of outbreaks of yellow fever in West Africa shows how often history repeats itself. If the early settlements in the West Indies and in Central America were hampered by yellow fever, so, precisely, on the West Coast of Africa, the foundation of missionary settlements, or the arrival of new regiments were heralded as a matter of course by outbreaks of what in every probability was the same disease.

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HISTORY OF YELLOW FEVER IN SIERRA LEONE

The foundation and settlement of Freetown appears from the commencement to have been impeded by outbreaks of yellow fever. References are constantly made by writers on yellow fever to an epidemic which occurred in 1815. According to Johnson, in that epidemic 26 out of 50 died, the symptoms being jaundice, pains in the loins, stupidity in the head, black discharge or black vomit, followed soon by death. He states further that the Nova Scotia settlers called it break bone fever. The fever was at the time attributed to a vessel which arrived in January 1815. There can be little doubt that the disease was genuine yellow fever.

This epidemic is also referred to by Staff-surgeons Barry and Fergusson.

French writers have taken it for granted that Sierra Leone was the home of yellow fever on the West Coast, on the very natural ground that it was more thickly peopled and had wider relations with the outside world.

Pym also contended that Sierra Leone was the home of yellow fever in West Africa.

Griesinger believed that it had early become endemic.

From Freetown the disease is supposed to have spread to the other parts of the coast.

1817.—Johnson refers to this year as an epidemic year, and states that 3 missionaries died after five days' illness.

In 1821 a missionary died from yellow fever accompanied by black vomit.

From 1822 to 1824 a considerable number of cases occurred. Staff-surgeon Barry, who described the outbreak, states that it broke out in December 1822; and that the first case was that

of a sailor; previously, however, the crews of several ships in the harbour had been attacked with what he describes as the endemial remittent fever of the locality. It was also a matter of much speculation, whether the disease was imported or endemic; some held that it was an imported contagious disease, others that it was local in origin, and that the endemial remittent fever of the place was another form of the disease; others, again, held that the endemial remittent and yellow fever had a common parent. One thing is clear, however, from the description of the symptoms and post-mortem appearance: that the disease was yellow fever.

The white population, and especially the sailors, suffered from the disease; out of a party of 12 schoolmasters, with their wives, 10 died.

It was noticed, however, that the blacks remained healthy, with the exception of the Croomen. During the outbreak the H.M.S. *Bann* arrived and her crew were attacked. She then sailed for Ascension, and, some affirm, introduced the disease for the first time into that island in 1823. (See Ascension Island.)

The mortality is given as follows:—

(Walker)—

		Deaths.
December	1822	7
January	1823	2
February	„	9
March	„	11
April	„	12
May	„	24
June	„	12
	Total	77

According to Burnett, a disease differing from the usual remittent and possessing the character of yellow fever occurred in 1823. It was not contagious and was not imported by the H.M.S. *Caroline*.

In 1825 an epidemic is described, with a total mortality of 263.

There was also a considerable outbreak in 1826, for out of a garrison of 535 soldiers, 115 died from 14th June to 24th August.

Major Crofts gives a table showing the mortality from remittent fever in July 1826. Fifty-three men and 3 officers are stated to have died. In all probability the disease was yellow fever.

In 1827 a typical case of black vomit was recorded (Fergusson).

In 1829 an epidemic broke out, and in the garrison of 130 whites 12 cases were reported. Eleven deaths occurred.

Boyle, in commenting upon the 1829 outbreak in Freetown, and refuting the idea that the fever was imported into Freetown by the H.M.S. *Eden*, states that occasional cases of yellow fever were met with every year in Freetown. Violent controversy also arose as to whether the disease was contagious or not. In consequence, a memorandum to the effect that yellow fever was not contagious, was drawn up in Freetown on 27th May 1829, and signed by J. Boyle (Colonial Surgeon), M. Sweeny, M.D., Deputy Inspector, and W. Fergusson, Surgeon, R.A.M.C.

In 1835 a case of black vomit is recorded.

The origin of the outbreak was ascribed to importation from Fernando Po, and by others to the town of Sangara, 400 miles distant. It was seriously proposed to build a high wall to keep out the pestilential breeze which, it was alleged, came from this town.

The next large epidemic occurred in the years 1836-1838. In December 1836, a malignant fever broke out amongst the crew of the barque *Mary* whilst in Freetown; 15 of the crew were attacked and 5 died. Cases then appeared on shore; one of those who died being a mulatto lady who had come from the United States; another was that of a young man who had only been one month in the colony. Five of the crew of another ship, *The Lady Douglas*, who had gone ashore and who

had lodged in the house previously occupied by the crew of the *Mary* were infected, and 4 died in January 1837.

Deaths occurred in March and April, and the fever was at its height in May and June. The last case was recorded in July, when, as the writer states, the yellow fever was succeeded by the common remittent fever of Freetown, and confidence was once more restored; but, adds the writer, to everyone's astonishment the fever broke out afresh before the end of the year. The first to succumb were the crews of three ships which had arrived during the autumn, and Staff-surgeon Fergusson attended 20 cases amongst the white residents between November 1837 and February 1838; 2 ended fatally.

In 1839, 6 officers of the garrison, 7 officers of the royal navy, and many soldiers and sailors died of yellow fever.

Table showing the number of cases treated by Fergusson from February 1837 to March 1838.

Occupation.	Cases.	Deaths.
Navy	43	25
Army	11	5
European resident merchants and seamen	272	107
Total . . .	326	137

During the prevalence of the epidemic, H.M.S. *Curlew* arrived and stayed one week in Freetown harbour and sailed for the Gambia. During her voyage the crew are stated to have suffered from an outbreak of the disease. Freetown was, in consequence, blamed for having introduced yellow fever into that colony for the first time.

As in the previous epidemic, so also in this one the relationship of the endemial remittent fever to yellow fever was much discussed. Fergusson put forward the opinion that yellow fever or malignant remittent fever was only the malignant form of the common "endemial remittent" fever, and he thought that

if cases of the simple endemial remittent type were transported by ship to other localities they might give rise to the malignant form. Fergusson firmly believed that "yellow fever was a product of the colony itself," but that both the Gambia and Ascension were infected from Sierra Leone.

In 1837 Burton described an outbreak of yellow fever in the Island of St Mary, in the Gambia, and to the north-west of Sierra Leone.

From 1837 to 1839 inclusive, the quarterly medical reports of the garrison and town show unquestionably fatal cases of yellow fever entered under the heading of malignant remittent fever.

In the period 1840-1845 there is a yearly record of the "endemial remittent fever," sometimes with severe and fatal symptoms. Ships' crews were often severely affected.

In 1845 a case of malignant remittent or yellow fever appeared; the fever broke out amongst the crews of the squadron at anchor on the Roquelle River.

The symptoms recorded are unmistakable, and are confirmed by the post-mortem accounts (Fergusson).

In this year also there appears some evidence that Bona Vista was infected from Freetown by H.M.S. *Eclair*.

It is also stated that in this year cases occurred frequently, and with the most severe symptoms amongst the newly arrived sailors, and least so amongst the native soldiers.

At this period also the expression "bilious remittent fever of the country" and "inflammatory fever" occurs—another name no doubt for the same disease.

In 1847 an epidemic is chronicled by Staff-surgeon Lawson. The resident white civilian population is put down at 100, and of these 12 died from yellow fever. There were also cases amongst the sailors in the port and on H.M.S. *Syren*, on which ship there were 17 cases, 3 of which developed black vomit ten days after embarkation.

In 1850 a case of fatal yellow fever was reported; it was said to have been introduced by a sailing-ship from Rio.

In 1859 an epidemic occurred in which 100 whites died of the disease.

In 1865 and 1866 cases were also observed.

After this period there is a lull, in which no doubt the disease persists amongst the permanent residents, but in a mild and undiagnosed form, but does not make itself obviously manifest, owing to altered conditions in the navy, mercantile marine, and in commercial enterprise.

In 1872 it broke out again, and 6 deaths were recorded. The colonial surgeon, writing in 1883, states we have had no cases of yellow fever since 1872. It is presumed to have been present in 1878. In 1884 a severe epidemic prevailed in Freetown; the cases appear to have been diagnosed either as yellow fever, severe, or pernicious remittent fever, and African fever, and a dispute arose as to the nature of the fever.

In the annual military returns 1 soldier is returned as having died of yellow fever, and the statement is made under date 25th June 1884, that yellow fever and a severe type of remittent fever prevailed in the town during this year.

There is no doubt that great confusion existed at the time between remittent fever and yellow fever.

The term "bilious remittent fever" has been employed in a great number of cases as another name for genuine yellow fever; some have, however, regarded it as a distinct disease. From the history of yellow fever on the West Coast, it certainly appears probable that a very large proportion of the bilious remittent fevers were cases of yellow fever. When the fever assumed a more severe type and became epidemic it was called yellow fever; as long as the cases remained mild they were put down as remittent fever.

The question, therefore, arises: Do the remittent fevers represent the mild forms of yellow fever? In this connection it is interesting to note that there is some evidence from the MS. notes of the period, that the "endemial remittent fever of Freetown" conferred a certain degree of immunity against

yellow fever, which would show that the remittent fever might be a mild form of yellow fever. Moreover, the fact that the natives escaped the severer form would also tend to show that they had had a milder form of the disease, probably the "endemial remittent." Lawson states that some medical men of the period considered that the natives did suffer from a mild form which was capable of inducing the malignant form in the whites. Lawson himself considered that both diseases had a common origin.

A Dr Davies, practising in Freetown, reported a case of yellow fever on 15th July 1884. Upon receipt of this report, the acting principal medical officer sent a letter to Dr Davies, asking him upon what grounds he had diagnosed a case of yellow fever, and if "he considered the cases of yellow fever, which he had had in his practice, to be contagious or not?" In a subsequent letter to the Governor, the principal medical officer stated that in his opinion Dr Davies had made a grievous mistake in his diagnosis of yellow fever. This correspondence is of great interest, as showing the attitude of mind at the time as regards the nature and diagnosis of yellow fever. Yellow fever in the same year appears to have broken out at Rufisque in another part of the colony.

In 1884 the evidence shows that there was considerable reluctance to notify the outbreak as yellow fever, and cases which terminated fatally and with the classical symptoms of black vomit were entered as "pernicious remittent fever," "African fever," "typhoid," and "enteric fever." The year 1884 showed a mortality of 50 amongst the whites; probably the majority of these were yellow fever.

In a report sent on 30th July 1884, to the then Secretary of State—the Earl of Derby—by Sir Arthur Havelock, the Governor of Sierra Leone, it is stated that a fever described as typho-malarial fever was prevalent during the months of May and June; recently arrived Europeans suffered most severely. The malignant symptoms of the disease became more marked

every day. On 27th June the disease was described as a "pernicious remittent fever on the borderland of yellow fever." At the same time a private practitioner had already concluded that the disease was yellow fever, and the military medical officer had actually reported a case of yellow fever. On 2nd June a European died of black vomit. On 28th July 2 Europeans died of yellow fever, diagnosed as such by the colonial surgeon. On 6th July another fatal case occurred. After this date a few more cases occurred, but of a less virulent type.

The final opinion, given on the 17th July by the colonial surgeon and the other practitioners in Freetown, was that the disease was a "mild type of yellow fever of a non-contagious nature." The Governor in his report states that a noticeable feature was that as the disease assumed a more virulent type, it became more and more restricted to Europeans. The natives seemed to have complete immunity from its attacks, there not being a single authenticated case amongst the negro population.

To Sir Arthur Havelock's covering despatch is appended the report of the special medical committee, consisting of the acting colonial surgeon, the senior military medical officer, and Dr Cole, a private practitioner.

From the report it appears that the epidemic was most fatal in Westmoreland, Rawdon, and Home Streets (the European quarter). The disease, they state, "resembled yellow fever or that type of pernicious remittent fever of a malignant destructive type, having as its characteristics yellowness of the skin and conjunctivæ, dark-coloured and very offensive alvine evacuations; dark-coloured urine containing blood casts and very obvious albumin, a quick pulse, and a persisting high temperature ranging from 102° to 105°; vomiting often persistent and very difficult to control, dark in colour, and containing a large quantity of bile, in some cases with distinctly black vomit."

Duration, 5-7 days; and, in malignant cases, 4-5 days.

The report describes the types of fever prevalent on the coast as intermittent fever or ague, remittent fever, enteric or typho-malarial fever, and pernicious malignant or yellow fever.

They state that the years 1807, 1809, 1812, 1815, and 1819 were marked by great sickness, the nature of which they do not state. They allude to the 1823 yellow fever epidemic, and state that in 1825, out of 902 persons attacked 263 died. Another epidemic occurred in 1829, when, out of 150 Europeans attacked 11 died.

In 1837 an outbreak also occurred in April, preceded by very suspicious cases in January. The epidemic, they state, died down, passing insensibly into the common endemic remittent fever. In 1838 yellow fever appeared in February and ended in March.

In 1839, 6 officers died of yellow fever and an appalling number of the troops; 7 officers of the royal navy and 13 seamen died.

In 1845 yellow fever broke out amongst the crew of Her Majesty's squadron at anchor in the Roquelle River.

In 1847 yellow fever was epidemic in Freetown during June, July, and August.

In 1859 also an epidemic broke out, when 100 Europeans died of yellow fever. Cases also occurred in 1865 and 1866.

In 1872 there was an epidemic, and 6 deaths occurred in December of yellow fever. The total mortality from this disease may have been 250.

Dr Lamprey, describing the cases of yellow fever in the 1884 epidemic in the *British Medical Journal*, 1885, states that the outbreak of yellow fever in Sierra Leone was sporadic in origin, the undoubted product of Freetown, and that all attempts to trace its origin to a non-sporadic source had failed.

He notes the difficulty of distinguishing between the bilious remittent fever and yellow fever. He states that in 1853 yellow fever was present, and that an epidemic occurred in 1825. He

states that also in the years 1829, 1837, 1838, 1839, 1845, 1865, 1866, and 1872 there were very many cases of yellow fever.

In the 1884 epidemic he notes that the total death-rate amongst the native population was 35 per 1000; and amongst the Europeans 6 per cent. per month. The epidemic commenced in May and lasted until August.

He then describes in detail the symptoms of a large number of cases. There can be no doubt, therefore, that there existed a severe yellow fever epidemic in 1884.

Examination this year, 1910, of the scanty medical reports of Freetown shows that in the year 1893-1894 deaths occurred from pernicious malarial fever.

In 1894 the statement is made that there were 16 deaths amongst the Europeans, of which 13 were stated to be due to bilious remittent hæmorrhagic fever and 1 from malignant malarial fever. Three of the cases died within thirty-six hours of their landing from the rivers.

Of course it is not now possible to be certain whether they were cases of blackwater fever, or whether some of them were not genuine yellow fever cases.

In 1899 there were 17 cases of remittent and 6 cases of bilious remittent fever entered in the hospital records.

In 1900 the statement is made in the annual report that there were 2 cases of yellow fever in the six years from 1885 to 1891.

In 1908 a Syrian died with symptoms, which the medical officer who was in attendance regarded at the time as a case of gastric ulcer. He now thinks that it might very well have been a case of yellow fever.

In 1909 a fatal case also occurred which, in the light of recent experience, the medical officers, who were in attendance, now conclude was yellow fever.

In 1910 some 10 cases of yellow fever have been reported, of which 8 proved fatal; but there may have been more cases and more deaths. The one fact which is certain is that the

disease was yellow fever, and that the Syrians were the first attacked, and amongst whom the greatest mortality occurred. The outbreak occurred in May and continued into September.

From the preceding history of epidemic and endemic disease in Freetown, the reader is, in my opinion, forced to but one conclusion, that, in the case of Freetown, as in the case of the towns in Central America, South America, and the West Indies, yellow fever in a mild or virulent form has been a disease common amongst those living in the *Stegomyia*-infected town of Freetown since its foundation to the present day.

In the preceding account yellow fever is diagnosed and carefully described by well-known military and naval surgeons, fully conversant with the disease in other parts of the world, and the descriptions which they have left in manuscript prove that their diagnosis was correct.

Again, there is the history of the almost annual occurrence of outbreaks of black vomit—a disease which all observers mention attacked newcomers in preference to the indigenous inhabitants. Indeed, authorities were agreed that the permanent black population did not get the severe yellow fever of the white man. Some authorities began to discuss the relationship between the common mild endemial remittent fever, or acclimatising fever of Freetown with yellow fever, and many concluded that the mild form could pass into the severe yellow fever. Others concluded that both had a common parent; others, again, that the remittent fever of the native could give rise to the yellow fever in the white man.

Another authority of the time concluded that the endemial remittent fever conferred a certain degree of immunity against yellow fever. All these are points which have, over and over again, been discussed in countries where yellow fever is endemic. They are all based on accurate observation; for, no doubt, the mild endemial remittent fever of the inhabitants of Freetown was in very many instances mild yellow fever, and it naturally conferred a certain degree of immunity, or perhaps complete

immunity to a subsequent attack of yellow fever. The endemial remittent fever of the native was a source from which the *Stegomyia* obtained its infection. The outbreak which has taken place this year, and in which the Syrians were early infected is, in my opinion, the final proof of the essentially endemic character of the disease in Freetown. The Syrians have, in recent years, increased in numbers in Freetown. They are engaged in a small trade, and live with and amongst the natives in the more crowded parts of Freetown. Their yards and those of their neighbours were infested with *Stegomyia.* It was but natural, therefore, that yellow fever should first manifest itself amongst them, as they were obviously most exposed to the infection. On the other hand, the merchants and officials living in better and less congested quarters suffered to a far less degree, whilst those completely segregated were not affected at all.

Sir William Pym, in commenting upon the origin of yellow fever in West Africa, states the great difficulty of bringing positive proof of its existence amongst the natives, because they have it in so mild a form that it does not kill.

He mentions how the Croomen escaped yellow fever in the West African squadron, when the white crews were suffering and dying from the disease. He concludes that it is reasonable to suppose that the Croomen had already had the disease in their native country.

Note.—The opinion of the acting principal medical officer —Dr Kennan—and of others was, that the epidemic of 1910 had a local origin and was not imported.

This opinion of the endemic origin of the epidemic of yellow fever was the view most frequently adopted by those who had been called upon to investigate outbreaks in the past. Investigation failed in 1910, as in past years, to prove importation.

I concur, as the result of examination of the facts on the spot, with the endemic origin of this outbreak.

Taking into account the very numerous outbreaks and sporadic cases of yellow fever which have been described in Sierra Leone

during the nineteenth century up to date, and bearing in mind that after no epidemic or sporadic case was any town fumigated to destroy the infected *Stegomyia*, we may be certain that, following the laws of yellow fever, cases of the disease occurred in intervening years. When these facts are borne in mind, and the same reasoning applied to the other colonies in West Africa, we are in possession of facts which, in my opinion, place it beyond a doubt that yellow fever is endemic on the west coast of Africa.

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Yellow Fever in the Gambia

It is most probable that yellow fever appeared in the Gambia at the same period as in the case of Sierra Leone and in Senegal, for epidemics in North-West Africa appear to have been on most occasions in the past widespread, sometimes starting in one colony, sometimes in another, but usually ending by affecting all three—Sierra Leone, the Gambia, and Senegal.

The period embraced by these epidemics and sporadic cases extended from the end of the eighteenth century, through the nineteenth, up to the present epoch.

Outbreaks have been recorded in 1768 and 1869, 1842, 1845, 1852, and in 1878, when the fever is stated to have been severe. During this period it will be remembered that the ships of the mercantile and royal navies were perpetually infected with yellow fever, and that, therefore, the disease could be imported in any year until it became endemic. Of course the disease might have been endemic from a much earlier period, but, as colonisation only set in in the eighteenth century, it is impossible to know what happened before this date, for here, as elsewhere, it was only by the arrival of non-immunes that the world was made aware of the existence of the disease.

We may reasonably suppose that the *Stegomyia* existed in the Gambia in early times just as it does to-day.

In 1837 Fergusson describes an outbreak of yellow fever in the Gambia, imported, so he believes, by the H.M.S. *Curlew*, which sailed from Freetown, where it had remained one week; and during an outbreak members of the crew contracted the disease, and when the ship arrived in the Gambia the sick were taken ashore and the disease spread amongst the residents. The colonial surgeon died from it. In the same year yellow fever was present in Senegal.

From the annual report of the West African station it appears that 4 cases of yellow fever occurred, of which 2 proved fatal, in the garrison in the period 1841-1842.

In 1878 a severe epidemic is stated to have occurred, but, so far, I have been unable to come across an account of it. It was also present in Senegal in this year.

In June 1900, a fatal case of yellow fever was reported officially from the Gambia. It was present in Senegal.

Bathurst was declared an infected port in June 1901, also Senegal.

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Yellow Fever in Senegal

The history of yellow fever in the French Colony is comparatively very complete, numerous French authorities of eminence having carefully described the various epidemics. As in the case of Sierra Leone, so in this colony the history of the disease is coincident with white colonisation and commercial advance.

Outbreaks of sporadic cases are recorded in 1768, 1769, 1778-1779, 1814, 1816, 1828, 1830, 1837, 1840, 1841, 1845, 1846, 1852, 1858, 1863, 1866-1867, and 1872. The symptomatology of very many of the outbreaks has been carefully recorded by Bérenger-Féraud, so that there is no doubt about the nature of the disease.

From the year 1830 yellow fever, however, attracted the attention of numerous observers, and, as in the case of the other West African colonies, the symptomatology is given with care, so that here again there is no doubt about the true nature of the disease. Schotte, in describing the great epidemic of 1778-1779, states that 59 whites died; that the deaths were most frequent amongst the whites, next in frequency amongst the mulattos, and least often amongst the blacks. The symptoms were: intense headache and lumbar pain; congestion of the eyes; obstinate vomiting or hiccough; black vomit (coffee ground-like material); urine greatly diminished or total suppression, delirium, coma, and death. Frequently the very characteristic and often fatal symptom of "feeling better" occurred towards the termination of the illness. From 1778 to 1814 no large outbreak occurred; then in 1816 the disease broke out at Cape Manuel. From 1817 to 1830 Schotte considers there were numerous sporadic cases. In 1830 a widespread epidemic occurred which affected Gorée, Saint Louis, and Dakar.

In 1837 the disease again broke out in the Island of Gorée, introduced, as it was thought by some, from Bathurst. Bathurst had meanwhile attributed their own outbreak to infection from Freetown (*see* Sierra Leone).

From 1837 to 1859 there is an apparent absence of recorded outbreaks. Then in 1858-1859 the disease again attracted attention. Again from 1864-1867 there are records of small outbreaks.

In 1872 a severe epidemic occurred, which spread from Gorée to Saint Louis, and then to Bakel in the Upper Senegal.

That is to say, the disease now showed its characteristic tendency to penetrate into the interior from the coast, following the trade routes. The writers of the period also state that up to this date yellow fever was usually regarded as imported from Freetown or the Gambia. But after 1872 authorities were disposed to admit its endemic origin.

In 1878 an outbreak is recorded, and it was stated also to be present in 1897.

From 1900 to 1906 there is an annual record of cases. In 1900 quarantine was officially declared, and Dakar, Gorée, and Conakry were declared infected ports. The 1900-1901 outbreak has been made the subject of more than one monograph. Kermorgant states that there were 146 cases and 225 deaths, and describes how it spread up into the interior and along the railroad from Kayes to Dioubeba in the Soudan. Bérenger-Féraud has also written the history of the epidemic. Both authorities mention how the disease was considered by many as endemic in Senegal. The possibility, however, of the ever-shifting Syrians having introduced the disease is also mentioned. In 1902 a recrudescence of the disease is recorded in the Soudan and Upper Senegal. In 1904 Dakar is declared to be an infected port. In 1906 yellow fever breaks out at Bamaku in Upper Senegal, and at points along the Kayes-Niger railway. The first case appeared to start in Segu in the Soudan.

An official communication from the British Consul-General in Dakar states that yellow fever was present in 1906 during the months of September, October, and November in the Upper Senegal and Niger Territories.

Thus, in Senegal, there has occurred an advance of yellow fever from the coast into the interior following the railroad and trade routes. This same phenomenon has repeatedly occurred in Central America (see my Honduranian Report), and will be repeated in Nigeria and in the Gold Coast colonies unless preventive measures are taken at once. This year the disease has penetrated to Saw Mills on the Gold Coast, and there is as yet not sufficient protective measures enforced to prevent it spreading to either the tin-mining centres in Nigeria, to Tarquah and Obuassie, or to the gold-mining centres on the Gold Coast.

The interior of a country offers no protection from yellow fever.

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Yellow Fever in Ascension, Boa Vista (Cape Verd Islands and Fernando Po)

The interest attaching to yellow fever in these islands is mainly from the historical point of view. They were ports of call for the West African ships in the eighteenth century, so that ships with yellow fever cases on board arrived regularly every year. The medical authorities of the period differed as to whether the yellow fever which did occasionally break out in epidemic form was imported or of local or endemic origin. Of course it would be difficult to settle that question now, as both views were readily possible, but at the time they gave rise to bitter controversy.

On the whole, the evidence is in favour of the disease having been endemic in the early part of the eighteenth century or even in the seventeenth century. Outbreaks have also been recorded in 1863 and 1868.

Ascension.—According to Staff-surgeon Barry, yellow fever was imported into Ascension in 1823 by the H.M.S. *Bann*, which became infected in Freetown during the yellow fever epidemic of 1823-1824. Yellow fever broke out upon the ship and 32 men died. The sick on arrival of the ship were sent ashore, and a few days afterwards fever appeared amongst the garrison, and of these 17 died. The *Bann* had a crew of 130, and of these 38 perished.

The same authority states yellow fever broke out in 1838, and was, as before, attributed to a ship which sailed from Freetown during an epidemic.

According to Malcolm, genuine yellow fever was present in 1818.

Boa Vista (*Cape Verd Islands*).—This island is stated on the authority of Pym to have been infected by the H.M.S. *Eclair*, which arrived from Freetown during the epidemic there of 1844-1845.

On the other hand, yellow fever is stated to have made its

appearance in these islands in the sixteenth century, and that they became an endemic centre from which the disease spread to the African coast.

Fernando Po.—In 1829 yellow fever is said to have been introduced in its malignant form by the H.M.S. *Eden*, but it has been also stated by medical chroniclers of the time that the disease might have been endemic (Pym).

Outbreaks are also recorded in 1839 and 1862.

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History of Yellow Fever in the Gold Coast Colony

There can be little doubt that yellow fever was as prevalent on the Gold Coast in the early part of the nineteenth century as in Sierra Leone.

From the report of Staff-surgeon Tidlie, quoted by Boyle, it appears that in 1819, out of 9 new arrivals, 3 died; in 1829, out of 4 new arrivals, 2 died; and in 1821, out of 7 new arrivals, 2 died, or two-thirds of all newcomers, within twelve months of arrival. In the same period 15 of the African Company had died. It is most probable that this fatal disease, which attacked the newcomers, was here, as elsewhere in the tropics—yellow fever.

In a report by Bell, upon the garrison at Cape Coast Castle for 1824, it is stated that 217 deaths occurred in the regiments, exclusive of women and children.

Of the first detachment of Europeans, numbering 128, which arrived in April 1823, only 1 survived; out of 109 women and children who had arrived since October 1823, 70 had died.

It is stated in the report that the cause of death was bilious

remittent fever, which usually terminated fatally on the third, fifth, or seventh day. Many of the cases were as "yellow as an orange." Boyle also mentions that an epidemic occurred in 1824, similar to that which took place in Freetown in 1823.

In 1824, out of a third detachment of 131 men disembarked in March 1824, the majority had died after a few months from "remittent fever and dysentery," and the same occurred with numerous subsequent detachments. Then comes the usual pause, commencing from the middle of the nineteenth century, probably due to the withdrawal of the garrison, and, therefore, of large bodies of non-immune new arrivals.

At a later date, following on the commercial development of the Colony and the growth of the coast towns, yellow fever again began to attract attention.

As had happened, however, in other British West African colonies, the disease was very frequently not recognised, and was more often wrongly diagnosed.

Examination of the case books of the European hospitals in the principal seaports are of very great interest, for they show in the first place, the difficulties which medical officers experienced in making a diagnosis, and, secondly, the unwillingness, as in Sierra Leone, on the part of the medical authority of the Colony to admit that yellow fever existed, in spite of the fact, as the careful records amply testify, of the patients presenting all the classical symptoms of fatal yellow fever. The stumbling block here, as in numerous other instances, being occasioned by the confusion brought about by the use of the word "contagious" as applied to yellow fever.

Examination of the medical reports of the Colony show that cases of yellow fever were recorded in the following years:— 1895 (several cases); 1897, 4 cases; 1902, 2 cases.

Whilst on the Gold Coast I examined the hospital case books of Cape Coast, Saltpond, Elmina, Axim, and Accra, and I find that yellow fever was diagnosed as such, and entered in the hospital case books at Cape Coast in 1897, 1902, and 1903;

at Saltpond in 1897 and 1902; at Elmina in 1895; and at Accra in 1899.

In 1899 Dr Elliott, who had had four years' experience in the Gold Coast, published in detail three cases of yellow fever which he attended at Saltpond.

He alludes to the reluctance prevalent against the diagnosis of yellow fever and the tendency to regard all fevers as malarial.

In 1901 Dr S. O. Browne published a case of yellow fever which he attended at Saltpond.

In addition to these well-marked cases which were diagnosed at the time by the physicians in charge as genuine yellow fever, the histories of the cases, in my opinion, also furnish unmistakable proof of the almost continuous occurrence of yellow fever for the past fifteen years.

The evidence is all the more trustworthy, for the medical officers, who recorded the symptoms, were not looking out for yellow fever.

The histories also show the difficulty which a medical officer experienced in interpreting the symptoms on the supposition that the disease might be malarial fever.

From my analyses of the case books, I conclude that yellow fever existed in addition to the above dates in the following places on the coast:—

(1) *Elmina. History of Cases examined since* 1895 *up to date*

In March 1895, a case was diagnosed as "hepatic fever," and ended fatally.

The symptoms were: great prostration, black vomit (coffee-like grounds), eyes and skin yellow, suppression of urine.

In April 1895, a case diagnosed as "malarial fever": it terminated fatally; the symptoms were: intense head and back ache, nausea and epigastric pain, black vomit; temperature 103·4°, pulse 92. A post-mortem examination showed skin yellow, stomach containing black vomit, congestion of the skin.

The doctor in charge of the case strongly suspected yellow fever, as also in the preceding case.

In January 1902, a case was diagnosed as "resembling yellow fever." The symptoms were: intense headache, albuminuria, intense vomiting, black vomit, black motions, death.

The case-book shows in addition numerous histories of cases which might well have been both mild and severe cases of yellow fever.

(2) *Quitta*

I examined the cases in the case-book from 1888-1900.

In 1894 a new arrival was taken ill, and a diagnosis of "malignant remittent fever" was made; temperature 104; conjunctivæ yellow; vomiting, urine black and bloody; delirium; albuminuria, followed by suppression of urine; convulsions; and death.

The medical officer states:—

"I cannot help being struck by the severe and persistent symptoms of this case, particularly the vomiting, which nothing seemed to affect. In fact, I can scarcely see the difference between such a case as the above and yellow fever."

In 1900 a suspicious case also occurred, the diagnosis being "remittent fever."

The medical officer stated the probability that the patient might be suffering from yellow fever suggested itself; but as the temperature showed no signs of subsiding, and there was no congestion of the face, it was decided that it would be better to wait and see, than to suggest the possibility to headquarters.

(3) *Saltpond. Examination of Cases from* 1889 *to* 1903

In 1895 a case occurred—diagnosed as "remittent fever"—terminating fatally. It was characterised by jaundice; temperature 105·8; pulse 60; black vomit; suppression of urine; hiccough; intense headache.

There were also about this time numerous other cases with a similar diagnosis presenting very suspicious symptoms.

In 1897 yellow fever is diagnosed.

In July 1897, a case was diagnosed as hæmoglobinuria and acute nephritis.

It was not diagnosed as yellow fever because of the absence of black vomit. Faget's sign, jaundice and hiccough, were, however, present, and hæmorrhage from gums as well as vomiting of dark bilious fluid is mentioned.

So, considering there were other cases in that year, it is not improbable that this was also a case of yellow fever.

In June 1898 a case occurred, ending fatally, diagnosed as "'acute' delirious malignant malarial fever."

The patient was so maniacal that he had to be handcuffed; temperature 105·4 and rose to 107·2; skin jaundiced, head and shoulders intensely congested, nausea, vomiting, coma, and death. Post-mortem: Cutaneous hæmorrhages, mucous membrane of stomach intensely congested, stomach contained dark green treacly mass, liver saffron yellow, with patches of congestion. This might well have been a case of yellow fever, in spite of the fact that it is stated "pigmented corpuscles were found in the blood."

In 1901 two cases were diagnosed as yellow fever; but in addition, in each year there occurred cases which, when we know that there were genuine cases of yellow fever, might equally well have been cases of yellow fever. The diagnoses made were usually "bilious remittent fever."

(4) *Axim. Examination of Case-Books from* 1906-1910

In 1905 the medical officer in charge of a case which occurred in that year at Axim, and which terminated fatally, is now of the opinion that the case was one of yellow fever.

In this year, 1910, a death occurred from yellow fever and was reported. The symptoms were, however, not well marked.

Later on in 1910 another fatal case occurred, which was

diagnosed as "remittent fever." The chief symptoms were: temperature 104·9; pulse rate from 110-120. Temperature did not yield to large doses of quinine.

Another case was diagnosed as "pernicious remittent fever," and proved fatal.

The symptoms were: intense headache; temperature 102·8; pulse 88; delirium, and finally coma.

The doctor in charge suspected yellow fever.

(5) *Cape Coast*

In 1903 Dr G. L. Barker wrote to the acting principal medical officer at Accra reporting a case of yellow fever, and he states that "this makes the fifth case this year."

The medical officer who diagnosed the case was Dr Rome Hall, and the principal medical officer, in writing a memorandum to the colonial secretary, states:—

"I regret to say that I am obliged to take the alarmist statement, made by Dr Rome Hall, with a large amount of doubt."

In 1895 a case of remittent fever is diagnosed, in which the characteristics are: congestion, intense headache, temperature 108·8, and pulse 52. The doctor in charge mentions that the curious point about the case was that "the pulse continued to get slower as the temperature got higher." The case, however, recovered.

Another case of remittent fever was diagnosed, which ended fatally; temperature 105; jaundice; great weakness, black motion, anorexia; delirium; patient then slowly rallied; this was followed by a relapse, coma, and death.

In this year there were several other very suspicious cases.

In 1901 a series of fatal cases, diagnosed as "remittent fever," occurred one after the other, in which there were characteristic symptoms: jaundice, delirium, great weakness, and coma; in one case the vomit contained bloody matter. In 1902 a case, diagnosed as "pernicious malaria," occurred, and

ended fatally; temperature 103; pulse 80; intense gastric symptoms; black vomit, delirium, hæmorrhage from the mouth, skin and conjunctivæ yellow; the physician notes that there was no black water, and that the pulse rate was not in proportion to the temperature.

Several other fatal cases also occurred in March and June 1902; in one, the diagnosis of "continued fever" was made. In this case there was black vomit and black motions.

In another case a diagnosis of "hyperpyrexial fever" was made. The skin and conjunctivæ were yellow, and the post-mortem showed the liver yellow, the mucous membrane of stomach and intestines dotted over with fine sub-mucous hæmorrhages.

Another case was diagnosed as "remittent fever." No parasites were, however, found in the blood. Vomiting was persistent; delirium, skin yellow, melena; post-mortem showed stomach and intestines intensely congested.

In another case a diagnosis of "malarial fever" was made; temperature 108·2; and it was noted that the pulse was very slow in comparison with the temperature. There was persistent black vomiting, melena, albuminous urine, conjunctivæ and skin yellow; post-mortem examination showed intense congestion of the stomach and kidneys. Then in July of the same year the doctor's suspicions were aroused, and he had no longer any hesitation in diagnosing "yellow fever," and two fatal cases are recorded.

It is therefore exceedingly probable that the other cases also were yellow fever.

In the commencement of 1903, Dr Rome Hall diagnosed a case of yellow fever, and he records minutely the symptoms. He states that he first considered the case to be one of black water, but the presence of albumin in the urine cast doubt upon the diagnosis. He states "that the post-mortem record appeared to be universally in favour of yellow fever. The writer then states that yellow fever has several times appeared

on the French Ivory Coast and in Senegambia. The Ivory Coast is only 150 miles, or forty-eight hours away from Cape Coast, and that at the present time quarantine is declared against the Ivory Coast." He then states that "the *Stegomyia* are swarming in the merchants' tanks, and in the tanks of the natives."

Dr Savage, of Cape Coast, who saw the fatal cases of 1902-1903, informs me that there was no question about the diagnosis of yellow fever.

(6) *Extracts from Case-books, Accra*

CASE *A. J. S.*, a new arrival, history of being bitten at Winnebah, arrived in Accra, April 1904.

19*th April.*—Patient felt unwell.

21*st April.*—Temperature 104°, pains over body.

22*nd April.*—Temperature 104-105°, urine scanty, albumin present.

23*rd April.*—Vomiting black coffee-ground material, stools, tarry.

25*th April.*—Vomiting persistent and violent, hiccough, tarry, stools, mind wandering, pulse 120, temperature 105°, coma followed by death. The medical officer regarded the disease as malarial.

CASE *R.*, April 1899.—Notes very meagre, but the diagnosis of yellow fever was made. The symptoms of black vomit, yellow blotchy skin, and intense headache were present, temperature 105°. On the other hand, there was no albuminuria.

CASE *A. W.*, patient died.—Diagnosis, "subacute rheumatism," June 1905. The symptoms were pains in joints; temperature 102·4°; scanty urine with trace of albumin; later on vomiting became a prominent symptom, accompanied by hiccough, mind wandering, pulse weakened, coma, and death.

Post-mortem examination showed deeply congested kidneys, and the note is made that immediately after death, the body assumed an intensely yellow colour; this is a sign which, in my opinion, is extremely characteristic of yellow fever.

CASE———, merchant, July 1905.—Diagnosis, "bilious remittent fever." The symptoms on 19th July were intense headache, temperature 101·8°, later in day 104°. Albuminuria, quinine little effect upon the temperature, vomiting became persistent, and patient became drowsy, urine much diminished, death on the 24th.

Post-mortem examination showed enlarged liver (colour not stated), and deeply congested kidneys.

CASE *Rev.* ———, February 1906.—Diagnosis, "remittent fever." Symptoms, 1st February: intense headache, temperature 103-104°. Gastric irritability, urine scanty with albumin. On 4th February commenced to have black vomit (coffee-ground material), collapse, death.

(7) *Tarquah District. Mantrain* (1902) *and Saw Mills* (1910)

In 1902, 8 men out of a staff of 14 died within about three weeks of one another. The doctor in charge also died; the others stampeded.

In a report which was sent from Tarquah on 14th June, 1903, to the principal medical officer at Accra, the following statement is made:—"I am informed that death in several cases occurred from hyperpyrexia, and that the administration of quinine, even in hypodermic injections in large doses, failed to reduce the temperature. In four of the cases death took place four days after the onset of the illness. Dr Macdonald examined his own blood during his illness, and, it is stated found the malarial parasites.

The writer formed the opinion that the disease was "pernicious malaria with hyperpyrexia," and he notes that no mosquito nets were used with two exceptions.

In this year (1910) a case of genuine yellow fever has occurred at Saw Mills, a small camp close to Tarquah, and I am, therefore, of opinion that it is not unreasonable to suppose that the fatal epidemic of Mantrain was genuine yellow fever, especially in view of the fact of the enormous numbers of *Stegomyia*.

Conclusions.—I am convinced from the preceding examination of the case-books that there were many more cases of mild yellow fever which were diagnosed as "remittent fever"; it was only when a case died, and when black vomit, slow pulse, and albuminuria became marked features, that the suspicions of the doctors in charge were aroused.

The mild cases passed unrecognised.

If additional proof were wanted to demonstrate the essentially endemic character of yellow fever in West Africa, it is furnished by the history and progress of the outbreak of yellow fever in the year 1910 on the Gold Coast.

In the first place, yellow fever broke out at three distinct points: Secondee, Saw Mills, and Axim. In the second place, it shows the difficulty which the medical officer had at first in deciding whether he was face to face with yellow fever or not.

Little time was, however, lost in arriving at a final diagnosis. I am convinced from analysis of many hospital case-books that in every probability a similar difficulty has presented itself to medical officers in the past upon many occasions, and that medical officers have refrained from giving the more serious, or, as it has been termed, "alarmist," diagnosis of yellow fever. In support of this contention the following memorandum is of the greatest interest:—

Outbreak of Yellow Fever, Secondee, 1910

The Colonial Hospital, Secondee,
9th May 1910.

"Sir,—I have the honour to report that recently there have been three cases of fever here, two of which came from the same bungalow, namely, Mr and Mrs C., and more recently Mr W., Supervisor of Customs, that present unusual symptoms.

"Mrs C. was removed into hospital with a temperature of 105°, persistent vomiting, and severe headache. She later became intensely jaundiced, but eventually made a good recovery, and has proceeded to England.

"Fifteen days later Mr C. was removed to hospital with a

temperature of 103°, intense headache, diarrhœa, injected eyes. He suddenly collapsed, and died three days later.

"Mr W. got a sudden attack of vomiting with a temperature 103·6° at night. He now has some diarrhœa, and his eyes are injected; jaundice seems coming on. I have treated each case with intramuscular injection of quinine until chinchonised. The first case had no albuminuria; in the second I was unable to procure a specimen, as he could only micturate when defæcating. Up to the present I have only been able to procure about two drams of Mr W.'s urine, which shows marked albumin with nitric acid (in the cold).

"These cases show a remarkable likeness to a series of cases in Accra, when I was there in 1905, all of which were fatal. I mentioned two cases to Dr Tweedy when he was here on his way to Tamele, who told me that he had met with similar in Cape Coast; also I had discussed them with Dr Slack. Without wishing to cause unnecessary alarm, I much fear yellow fever may be present here, and beyond Drs Slack and Tweedy, the only other person I have mentioned the matter to is the Acting Provincial Commissioner, Mr Furley.

"Will you inform me by telegram what steps, if any, I can take to prevent an epidemic, as *Stegomyia* are very prevalent here.

"C. H. D. RALPH."

The outbreak of yellow fever which took place in Secondee this year, 1910, differs in no essential respect from all previous ones in West Africa, or in the other parts of the tropical world. It is a matter for congratulation, however, that this disease was early recognised, and that the medical authority of the colony did not lose a day in taking preventive measures.

The outbreak, which started in March and ended in June, was a comparatively small one—about 13 cases—but as in all these outbreaks, so here it is more than probable that there were other mild cases which were not diagnosed until the severe cases drew attention to the nature of the disease; there were 10 cases amongst the white population and 3 amongst the native. The native population is large, and the white com-

paratively very small (about 120). Therefore, as in all previous epidemics, the comparative percentage mortality amongst the whites was very much greater than amongst the natives; in fact, there is no comparison. Of the whites who were attacked, it is significant that they had been in Secondee for periods varying from a few days to a few months immediately prior to the outbreak.

The mortality in proportion to the number of cases notified was very high. All the cases proved fatal with one exception. The high mortality rate is of common occurrence in countries where the medical authorities are not suspecting yellow fever.

The chief symptoms recorded are typical of yellow fever, viz., violent headache, yellow scleræ, jaundice, or sudden turning yellow immediately after death, albuminuria, suppression of urine, delirium, Faget's sign, black or black-speckled vomit, coma. Where quinine was administered it did not appear to have any effect.

Post-mortems showed :—

Boxwood liver, congested kidneys, stomach and intestines, otherwise organs normal.

In the opinion of the senior sanitary officer, Dr Rice, and of the acting principal medical officer, and also of the other medical officers who were associated with them in the investigation of the 1910 outbreak of yellow fever in Secondee, the disease was of endemic origin. The following are Dr Rice's conclusions, and with them I agree, after examination of all the circumstances on the spot :—

"That yellow fever had previously broken out in the Gold Coast is well shown in the extract which the acting principal medical officer has furnished from the annual reports.

"I am a comparatively recent importation to the Gold Coast, and hence am not in a position to state what were the reasons which led the authorities to call this disease, for so many years, by another name. A study of the case-books from the various

parts of the Gold Coast leave upon one's mind the impression that in many instances Europeans who have been returned as having died of malignant malaria, have really died of yellow fever. Sometimes the medical officers have discussed the possibility of certain cases being yellow fever, and in other instances they have called it so, but in the annual returns this disease has usually been conspicuous by its absence, and, apparently, whether by accident or design, it had never been given official recognition.

"In reply to telegrams from headquarters as to the source of infection, I replied that I considered yellow fever to be endemic among the native population of West Africa, and I am still of that opinion. There is no other hypothesis that to my mind can explain this and previous outbreaks. The precise conditions that are necessary for the disease to be communicated to the non-immune white population are as yet unknown, but two conditions are always present when non-immunes are attacked, viz. :—

"1. They live in native towns unsegregated from natives.

"2. Under conditions which favour the breeding of the enormous numbers of *Stegomyia.*

"Dr W. C. Gorgas makes an interesting statement on this subject in the Proceedings of the Canal Zone Medical Association, 1908, viz. :—'That a certain proportion of *Stegomyia* must be present in a locality for the spread of yellow fever.'

"It is customary when an outbreak of yellow fever occurs to look for the ship that is always supposed to have brought the infection, in spite of the fact that to bring yellow fever to West Africa is equivalent to carrying coals to Newcastle.

"Two ships, the barques *Montgomery* and *Cosmos*, had brought cargoes to the port from America. The *Montgomery* from Gulf Port, Mississippi, arrived in Sekondi on the 17th of January, and left on the 7th of March. The voyage lasted seventy-five days, and the ship had a clean bill of health.

"The *Cosmos*, from Mobile, United States of America, arrived at Sekondi on the 5th of March, and left on the 10th of April, having been forty-five days on the voyage, with a clean bill of health, so these ships may be regarded without suspicion."

Extracts from the Annual Medical and Sanitary Report, 1895, Gold Coast Colony

"*General Health of the Colony.*—The general health of the colony was extremely bad during the period, the endemic fever assuming a pseudo-epidemic form of a malignant type closely approaching in its clinical manifestations the vomito negro or yellow fever of the West Indies. The death-rate was enormous among Europeans, and the excitement induced thereby amounted almost to a panic and served to intensify the fatal tendencies of the prevailing fever in the latter part of the year.

"At the beginning of the month the general health of the town of Accra was bad, and some cases of the malignant type of fever which during the first six months of the year prevailed on other parts of the coast appeared. Three deaths occurred from it amongst the officials of the African Direct Telegraph Company in connection with this disease (which, in my opinion, is of the same type as has during the last few years appeared occasionally at Sierra Leone, Bonny, Lagos, and other places on the west coast of Africa)."

Cape Coast.—During the first quarter the health of the European residents was extremely bad, and judging from the death-rate, worse than it has been for some years. "The prevalent diseases were remittent fever and diarrhœa. The former seems to have been of a very pernicious nature, with a tendency to hyperpyrexia, suppression of urine, and amaurosis" (Dr Lyons). Four deaths occurred among the non-official Europeans.

During the second period the same condition of things persisted, and the mortality was great; among officials 2 died, and among non-officials 6.

Elmina.—The first quarter was marked by an unusual prevalence of malarial fever. Out of a grand total of 10 officials, 8 suffered from attacks of fever more or less violent, and of these

3 died and 2 were invalided. "The non-official Europeans did not suffer so severely: out of a grand total of 14, 1 was invalided and died three days later at sea. The prominent features of the various fatal cases were blood destruction, suppression of urine, hyperpyrexia, and heart failure" (Dr Elliott). During the second quarter "the unhealthiness which characterised the previous quarter was continued into the early part of May, when the onset of the wet and cold season inaugurated a general change for the better."

"*Saltpond* (first quarter).—The general health of the Europeans was most unsatisfactory, the prevalent diseases being remittent and intermittent fevers and dysentery. One official died from remittent fever, complicated with suppression of urine (Mr Trigg).

"*Saltpond.*—In 1895 I was stationed for some time in Saltpond, during which period an outbreak of a very malignant type occurred amongst the limited European community. Amongst the cases were Father Riche, of the Catholic Mission; Mr Trigg, of the Public Works Department; Mr Moran, a trader; and another merchant. One of these cases, Mr Moran, recovered. The cases presented the features of rapid onset, high fever, which gradually subsided, jaundice, and albuminuria in the 3 fatal cases, black vomit, suppression of urine, coma with convulsions and death, and at no time was hæmoglobinuric urine passed in any case. A prominent fact which lasted for ever in my memory was the existence of a peculiarly offensive odour and the passage of peculiar tarry motions, the immediate circumference of which in a bed-pan or chamber pot presented a look of altered blood. All these fatal cases presented a peculiar delirious restlessness before absolute coma set in. Although I was inclined to regard these as cases of yellow fever, I was in possession of such information that I knew such a declaration would be useless on my part, and in this connection I invite attention to paragraphs 12, 14, and 31 of the *Annual Medical Report* for the year 1895. Mr Trigg lived in hired

quarters in a native house, and the other Europeans lived in the town. The second Catholic Father was invalided very soon after the death of Father Riche. I closed their house, and condemned it. Some time after, when Bishop Albert and Father Wade arrived in Saltpond, I did not allow them to occupy the quarters, and told them what I thought the cause of the sickness was. At this time the mosquitos were plentiful at Saltpond in the native quarters, but I was not able to identify species.

" In the case of Mr Trigg I recollect wishing to try pilocarpine, owing to the suppression and to the fact that the skin remained dry. I also recollect improvising a vapour bath in the hope of starting diaphoresis.

" There had been much sickness and mortality prior to this at Elmina and Cape Coast, and my leave was twice put off, owing to the death of the medical officer deputed to relieve me, one, Dr Lyons, being invalided and dying. Soon after, the other, Dr Conran, died at Cape Coast. At this time much difference of opinion existed as regards the sickness, but as far as my recollection goes the majority of the medical officers who saw cases believed they were dealing with yellow fever, and I heard that Drs Lyons and Conran had succumbed to this disease." (From report furnished by Dr Garland.)

Extract from Report for 1896

The general health of the colony during the year under review shows little or no improvement on that of the previous year. This year there were amongst the European population 11 deaths of officials and 30 non-officials, as against 15 and 23 respectively during 1895, the number invalided being 23 officials and 35 non-officials, against 26 and 32 in 1895.

This high rate is to be attributed to the fact that the epidemic of the malignant type of fever which prevailed during the last half of 1895 continued during the first four months of this year, the period during which the greater number of deaths occurred.

In Accra there was a good deal of sickness during the first half of the year, but the last half was exceptionally healthy, more especially amongst the European officials; this, no doubt, being due to the fact that the majority now live outside and at a considerable distance from the native town.

Extract from Report, 1897

During the second quarter of the year Cape Coast and Saltpond suffered from an outbreak of fever, affecting, so far as I am aware, Europeans only, of the same nature as that which scourged Elmina and Cape Coast early in the year 1895, and which later in the year appeared in Accra, to which the chief medical officer alluded in paragraph 1, Report for 1895.

Health of Europeans at the Various Stations

Saltpond.—No deaths among officials. There were 6 deaths among non-officials, equal to a ratio of 67·4 of the total strength of Europeans in the station for the year. No officials were invalided, but 3 non-officials were. As regards the causes of death, 1 was a case of cerebral hæmorrhage in an elderly man; 2 were acute nephritis complicating malarial fever, and 3 were cases presenting the classical symptoms of yellow fever. The health of the officials was good, that of the non-officials exceedingly bad. The non-official Europeans, that is, the agents of the European mercantile firms, live in houses which are grouped round the lagoon, whereas the officials' quarters are situated at some distance from the lagoon and close to the beach. This is sufficient to account for the immunity enjoyed by the officials, while the European employees of the mercantile firms were suffering from all the varieties of fever that are met with on the coast.

Cape Coast.—There were 12 deaths among non-officials, equal to a rate of 50 per 1000; there were no deaths among the officials. Causes of death were chiefly bilious remittent fever. There was 1 from yellow fever.

A. Swanzy died at Accra, 25th April 1904, case being clinically similar to yellow fever. He was nursed by Miss Fraser, who went on leave 27th June, died on 3rd July at sea; symptoms alleged to be like yellow fever.

Note furnished by Dr Garland, Acting Principal Medical Officer

"I concur with the opinion of Sir Rubert Boyce that yellow fever existed for a considerable time and was not officially recognised. However, there were occasions in 1894 and 1895, and subsequently, when many of the medical officers of this colony openly stated they were convinced that they were dealing with this disease; and that the matter was one actually under controversy, is proved by the following extracts by the chief medical officer from the Medical Report for 1895:—

"Three cases in Accra occurred in one house. During this year, 1895, I had 4 cases of yellow fever at Saltpond, 3 of whom died. I wrote to some of my colleagues at Cape Coast, as I know that determination had been shown in favour of the disease being declared a non-infectious bilious remittent fever. I only learned that more than one of the members of our staff considered they were dealing with yellow fever, but that the diagnosis would not be accepted.

"I am inclined to believe that this outbreak originated at Elmina in 1894, where several Europeans died and which had previously borne the reputation of being a most healthy station. After a considerable lapse of time an outbreak of a fatal type of fever occurred at Accra in 1896, and I understand that there was a severe outbreak at Little Popo some time after this; but there is no opportunity of arriving at the facts with regard to the latter.

"In the year 1897 a case of yellow fever was officially returned by the medical officer at Cape Coast.

"In the year 1897 a series of deaths occurred at Saltpond and Cape Coast, and the disease there presented the clinical manifestations of yellow fever.

"In 1898, 4 deaths occurred in the first six months amongst the Europeans at Saltpond, and were classed as pernicious remittent fever.

"In 1902, 2 cases of yellow fever were recorded at Cape Coast by the late Dr W. Murray.

"In 1903 a highly suspicious outbreak occurred at Maintraim, and ended in 6 deaths, including the doctor, whose death unfortunately precluded the possibility of any medical history being rendered.

"Subsequent to this the only occasion on which cases presented a suspicious nature was in 1904, when Mr A. J. Swanzy died after a few days' illness, having presented the clinical symptoms of this disease.

"In June 1905, Mr Wrenn was admitted, and died within a few days, after showing many of the symptoms of this disease.

"Mr Lulwer died in the hospital on 24th July, after displaying many of the symptoms of yellow fever.

"In February 1906, Mr Jubb, a Wesleyan missionary, died; the symptoms pointed to yellow fever.

"On 14th March 1906, a European clerk named Bailey died from fever, showing symptoms of yellow fever."

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Yellow Fever on the Ivory Coast

From 1899-1905 this colony shows a practically unbroken record of genuine malignant yellow fever. So bad had it become at one time, that it was seriously proposed to abandon Grand Bassam, and, according to consular and medical reports,

the greatest hindrance to commercial advance in the past has been yellow fever.

In 1899-1900 there was a very severe epidemic.

In 1903, during a period of four months, no steamers entered the port, and all trade was at a standstill. In 1904 the disease was again prominent, and the port was again stated to be infected in 1905. In fact, from 1900-1905, a year has not passed without the report of cases, and "gazette" quarantine notices have appeared in regular succession. During one of the epidemics, 50 per cent. of the native population left Bassam. In 1903 the European population was about 60, and the death-rate amongst these was 50 per cent.

In recent years drainage and *specific anti-mosquito* precautionary measures have been taken, and have replaced the old-time process of disinfection and the burning of houses. It is to the awakening-up to the importance of specific *anti-Stegomyia* measures on the Ivory Coast, and in Senegal, and in Togoland, that must be ascribed the immunity from yellow fever which these colonies have experienced since 1905.

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Yellow Fever in Togoland

The German colony of Togoland, since it has been opened up to commercial enterprise, has been the seat of frequent outbreaks of yellow fever. Thus, in 1896, 40 cases were reported in Klein Popo.

After this period there appears a lull, that is to say, there was no obvious outbreak until 1905, when some cases were recorded at Anecho and Lomé. In 1906, 5 deaths were also reported at Lomé, and 1 case at Wydah. Krueger states that 30 cases occurred in Togoland from April to May 1906. As the

symptomatology and post-mortems of many cases are given, there is no doubt of the identity of the disease. According to the official gazettes, quarantine was declared against Togoland and Lomé on 22nd April 1905; Agone and Grand Popo in May 1905; and against all ports in Togoland, June 1906.

Dr Otto was strongly of opinion that the disease was endemic, and that the virus had been kept up by mild cases and recurrences. Those who have studied yellow fever in recent years regard this as the most rational explanation of outbreaks or sporadic cases of yellow fever.

Otto also draws attention to the fact that many of the cases of yellow fever were diagnosed as "pernicious fever" with "inflammation of the kidney," "febris malaria continua perniciosa nephritica," "fever and heart disease," etc. Thus, as in British colonies, so in the German, there can be no question that yellow fever has passed unnoticed under a great variety of names. Probably no disease has been mistaken for so many other diseases. In my opinion this has in large measure arisen from the universal reluctance to admit the existence of the presence of yellow fever in any colony. No doubt commercial interest is the real cause. There were, however, excusable reasons, such as the lack of any miscroscopic test, as in malaria, and the fact that a large proportion of cases bear a considerable general resemblance to malaria. It is for these reasons that the best guide to diagnosis in parts of the world where undoubted large outbreaks of the disease have occurred is the presence of the *Stegomyia* in abundance. This is the case in German, as well as in the British and French West African, colonies.

Vigorous anti-stagnant water ordinances have been introduced and enforced in Togoland.

Yellow Fever in Dahomey

Within recent years Dahomey has been the seat of numerous recorded outbreaks. In 1905, cases occurred at Lomé and at Agone, where several deaths took place.

In 1906 an outbreak occurred at Grand Popo. In the same year yellow fever was present in Porto Novo and Koonu, and 11 deaths are said to have taken place.

It is very generally recognised amongst merchants that the coast and river towns in Dahomey and Togoland are very liable to yellow fever.

Considering that these towns are close to Lagos, it would be exceedingly strange if Lagos should have been free of yellow fever in this period, even admitting that yellow fever was not endemic in that city. It was obviously liable to be imported at any time.

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Yellow Fever in Southern Nigeria

It would be strange, in view of the reasons which I have put forward for regarding yellow fever as endemic in West Africa, if a colony like Southern Nigeria, where the prevailing town mosquito is the *Stegomyia calopus*, and close around which the existence of yellow fever has been officially chronicled during the past fifteen years, should be exempt from endemic yellow fever. I am of opinion, however, that yellow fever is endemic, and that, in all probability, as in other colonies, the disease has long been overlooked and mistaken for other diseases. Unfortunately, the medical records which have been kept in the hospitals in the past have been of a very scanty nature, and there are no careful manuscript records of the diseases prevalent amongst the troops and sailors similar to those which have been preserved in Freetown.

There has been, however, a strong opinion expressed by many of the experienced traders that the disease which has proved rapidly fatal to the white man on several occasions, and

which had at the time been attributed to a malignant form of malaria, was, in all probability, yellow fever. This view is much strengthened by the outbreak of yellow fever which took place in Bonny in 1899, and by the undoubted cases, in my opinion, of yellow fever which occurred in Lagos in 1893-1894 and again in 1905 and in 1906.

I will now briefly record the outbreaks and supposed outbreaks of the disease in some of the chief towns of Southern Nigeria, in order that the student may realise why I consider yellow fever endemic in Southern Nigeria at the present time :—

Yellow Fever in Southern Nigeria

Warri.—The medical officer reports that a tradition exists that there was a yellow fever outbreak at the European factories about the years 1860-1870.

Calabar.—An undoubted case occurred in the years 1890-1891.

Bonny.—1873: Several deaths were recorded which might have been due to yellow fever.

In 1883 it is stated that an outbreak of yellow fever occurred due to importation from Freetown.

In 1890-1891 a very serious outbreak is recorded of what was unquestionably yellow fever. The medical officer, Dr Parker, was certain that it was genuine yellow fever. The description of the symptoms are such as to leave no doubt as to the nature of the disease. The disease was thought by some to be malignant malaria, and was attributed to the pulling down of an old factory; others maintained that it was introduced in the straw and litter on a ship coming from South America.

The natives were not affected. There were 11 cases and 9 deaths amongst the population of 15 white men.

Dr A. J. Brown, who was in Bonny at the time, informs me that he attended two of the cases. The symptoms were headache, pain, great prostration, albuminuria, yellowness, persistent black vomiting.

Dr Parker, who was the medical officer at the time, stated that he was convinced that the disease was yellow fever.

He subsequently contracted the disease and died.

Dr MacDonald, Bonny, has furnished me with notes of a suspicious case which occurred in 1909, six weeks after arrival in Bonny. The diagnosis at the time was "gastritis and jaundice." No malarial parasites were, however, found in the specimens of blood which were repeatedly examined.

From numerous inquiries which I have made there can be no doubt that yellow fever occurred in Lagos in a virulent form from 1894-1895. Thus, I have it on reliable authority that 17 members of a mission arrived from England in Lagos on 13th December 1893. Of these, 5 had died by the end of January 1894. In addition, a resident missionary, and the young child of another missionary, had also died. The cases presented the classical symptoms of yellow fever. According to Ott, cases were reported at Lagos in 1896. This is confirmed by Dr Hopkins who saw 2 cases in that year.

At Sapele, in 1898-1899, there were a considerable number of suspicious deaths; and in 1907 an outbreak of a severe type of fever was recorded at Widah.

Examination of the medical notes in the hospital at Lagos convinces me that genuine well-marked cases of yellow fever occurred in the years 1902-1905; there were also many mild cases.

The symptoms recorded are violent headache and body pains, high temperature, slow pulse, suppression of urine, black vomit, and coma, terminating fatally. In 1905 the symptoms recorded could hardly be those of any other disease but yellow fever, yet the diagnosis made at the time included "fever and gastritis" and "fever and morbus cordis"—a diagnosis which Ott also states was made in Togoland. I am therefore strongly of opinion that in Lagos one of the causes of mortality in the past has undoubtedly been yellew fever. When it is recollected how little is known of the fevers amongst the 60,000 native

inhabitants of Lagos, and when it is understood that by far the most abundant mosquito is the *Stegomyia*, it is not unreasonable to assume that the natives in all probability suffer from a mild type of yellow fever, and that, therefore, yellow fever is endemic.

Further evidence in favour of this contention is furnished by the admitted frequent presence of yellow fever in Dahomey and Togoland close by (see these colonies). It must also be recollected that, just as in the case of Sierra Leone, the infected *Stegomyia* were not destroyed by fumigation, but were left to propagate the disease.

Summary

I think I have brought forward sufficient evidence based upon accurate records, clinical and historical, written by men of admitted ability and experience in yellow fever, to conclusively prove that yellow fever has been of far more frequent occurrence than is usually supposed on the West Coast. That, in fact, it has appeared annually over a very large number of years practically, as my figures show for the last hundred years.

A few gaps of a few years have occurred, such as between 1852 and 1858, 1868 and 1872, 1873 and 1878, 1878 and 1883, 1884 and 1890.

But from 1890 to the present date I am of opinion from the data which I have examined that there is an unbroken line.

During the whole hundred years there is no large interval which would make it reasonable to suppose that yellow fever had completely died out on the coast.

In my opinion this evidence is so strong that we are obliged to assume that the disease is endemic upon the West African coast for at least the last hundred years.

The question now therefore arises, by whom has the virus been kept up?

In the first place, we know positively that the transmitting agent, the *Stegomyia*, is present in overwhelming quantity. It

only remains to prove how a continuous source of infection has been maintained.

To those who would adopt the theory of importation, it would mean a continuous importation from, say, the West Indies, Central, or South America, and there is no history of such importation.

Therefore, in my opinion, the most reasonable explanation is the one which has proved correct in the West Indies, Central, and South America; and is adopted by the most recent English, French, and German investigators in yellow fever, namely, that the disease exists amongst the natives in a mild form; in other words, that it is endemic.

A little consideration will show that the whites have not been the source of the continuous infection for the reason that the total number of whites on the whole West African coast has never been large enough to admit of continuous keeping up of the virus; the whites are in the very small minority.

Therefore, precisely as in the case of the sister disease, malaria, the continuous or endemic source of infection is the comparatively dense native population of the West Coast.

The evidence which I have brought forward also conclusively points both in English, French, and German colonies to a vast amount of *mistaken diagnosis*. Yellow fever was not suspected in its mild form, and it was not found out, it was only discovered when fatal cases made their appearance, and, as my evidence shows, these cases were as often as not mistaken for other diseases. These same mistakes in diagnosis have occurred over and over again in yellow fever countries, especially in the commencement.

I therefore conclude from the evidence that a comparatively large number of deaths and mild cases have occurred from yellow fever in the past and which have been attributed to malaria, chiefly the "bilious remittent fever." Most authorities upon yellow fever are agreed that in a very large number of

instances "bilious remittent fever" is another name for yellow fever.

It must be recollected that it is only in this year that effective sulphur fumigation of the infected *Stegomyia* has been attempted on the West Coast after outbreaks.

Therefore infected *Stegomyia* were left in the past to live on and to carry infection into a succeeding year.

Finally:—

(1) The historical record of outbreaks and sporadic cases, as recorded above;

(2) Mistaken diagnosis; and

(3) The absence of any destruction of infected *Stegomyia* in the past, is evidence overwhelmingly in favour of the disease being endemic on the coast, and of its having been repeatedly mistaken for other diseases or entirely overlooked.

Years in which Yellow Fever has appeared in Sporadic or Epidemic Form in West Africa

Note.—These figures are based upon documents, official reports, and published memoranda carefully examined by the author.

1807	1826	...	...	...	1894
1809	1827	1844	...	1878	1895
...	1828	1845	1862	...	1896
...	1829	1846	1863	...	1897
1812	1830	1847	1864	...	1898
...	...	...	1865	...	1899
1814	...	...	1866	1883	1900
...	...	1850	1867	1884	1901
1816	...	...	1868	...	1902
1817	1835	1852	...	...	1903
1818	1836	...	...	...	1904
1819	1837	...	...	...	1905
1820	1838	...	1872	...	1906
1821	1839	1856	1873	1890	...
1822	1840	...	...	1891	1908
1823	1841	1858	...	...	1909
1824	1842	1859	...	1893	1910
1825					

PART II

SYMPTOMATOLOGY AND TREATMENT

CHAPTER VIII

EXPERIMENTAL YELLOW FEVER AND YELLOW FEVER TYPES

In this chapter I desire to draw attention to the various types of yellow fever, those forms, more especially, which often pass unrecognised because of the mildness of their symptoms; but which nevertheless are the means of keeping up a constant infection, and are the source of the more typical severe forms of fever which attack non-immunes and new arrivals.

The study of infectious diseases teaches us that until we are in possession of some simple scientific clinical test, such, for example, as the finding of a protozoon or bacterium in the blood, or in a tissue, or until we possess a serum reaction as in the case of typhoid, there must exist immense difficulty as to the diagnosis of the true nature of any disease in its early stages.

The young medical officer will experience this difficulty in deciding what is and what is not yellow fever. The same difficulty crops up in every disease. Were we in a position to diagnose early the very mild forms of typhoid, scarlet fever, diphtheria, etc., the prevalence of these diseases in Europe to-day would be far different.

In the majority of cases a diagnosis is not made until the well-marked, severe, or as they are termed "classical" signs of the disease in question have declared themselves. So it is with yellow fever.

It is for these reasons that it is very essential to study what are the uncomplicated symptoms produced by the bite of the

Stegomyia, and then to analyse and see if in nature there are diseases which present similar or closely similar symptoms.

I have endeavoured to bring out these points in the following account. It will serve as an introduction to those more severe symptoms met with in yellow fever, which are regarded as "typical" of that disease.

It is a golden rule in analysing any disease, to, where possible, direct in the first instance attention to experiments or direct observations. Unfortunately a large number of tropical diseases are still based upon a most hazy and inaccurate pathological foundation.

In yellow fever, however, thanks to the experimental work of Finlay, Reed, Carroll, and other American observers, we have a very definite foundation to build upon.

I.—Experimental Yellow Fever

The reason why the Reed Commission did not hesitate to make direct *Stegomyia* inoculations in man, was because as they state, Finlay had shown as the result of 90 inoculations that little danger resulted from the bites of infected *Stegomyia*. In other words, Finlay found that in 18 per cent. of his inoculation experiments there resulted *a very benign form of yellow fever*. The American Commission made a series of very carefully recorded observations, and in 12 cases were able to produce yellow fever in its milder type. The following are some typical examples:—

Case V.—Infected 19th January. On the 23rd took to bed with feeling of lassitude and headache; temperature 99·2°; pulse 78. Later in the day these symptoms increased, and were severe headache, chill, eyes and face flushed; temperature 100°; pulse 104; still later headache increased with marked backache, vomited once; temperature 103·6°; pulse 110.

At the end of 42 hours albumin appeared in the urine. The scleræ became jaundiced on the second day, and by the fourth day this had extended to other parts of the body.

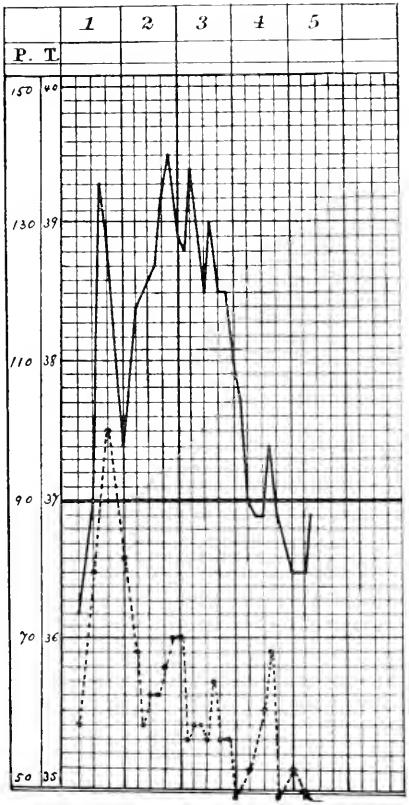

FIG. 6.—Temperature and pulse curve (dotted lower curve) from an experimental case of infection by the *Stegomyia*. There is an early remission in the temperature curve on the second day. The pulse falls from the end of the first day, and does not follow the temperature.—GUITERAS.

[*To face p.* 100.

The symptoms did not increase, and on the seventh day the temperature was normal.

Diagnosis.—Yellow fever of moderate severity.

CASE VI.—C. W., American, non-immune, aged twenty-seven, with his full consent was at 9.30 A.M., 31st January, bitten by two of the three mosquitos that had been applied to the foregoing Case V. The interval that had elapsed since their contamination was therefore 51 days. The subject remained well until 12 o'clock noon, 3rd February, when he complained of heaviness in his legs and some supra-orbital pain. His temperature at this hour was 99° F. and pulse 70. At 1.30 P.M., it had risen to 100° F., and at 5 P.M. to 100·6° F., and pulse to 84. The primary rise of fever, which was somewhat fluctuating in character, did not reach its height until at the end of 24 hours, noon, 4th February, when temperature was 102·4° and the pulse 92. The facies was now suggestive of yellow fever. Remission occurred at the end of 45 hours and lasted for about one day. The secondary rise was slight in character, the temperature falling to normal on the morning of the sixth day. The case was very mild in character. Albumin appeared at the end of 75 hours (beginning of fourth day); it never amounted to more than a distinct trace and disappeared on the eighth day. There was no occular jaundice, and although the gums were injected and swollen, there was no hæmorrhage at any time. The patient perspired freely throughout the attack. Convalescence was rapid. The subject had been in quarantine for the period of 6 days prior to inoculation.

CASE VII.—J. H., American, non-immune, aged twenty-six, with his full consent was bitten at 11 A.M., 6th February 1901, by the same two mosquitos that had 6 days previously bitten Case VI. Fifty-seven days had therefore elapsed since the insects had been contaminated by biting a case of yellow fever. He remained well until 12 o'clock noon, 9th February, when he experienced slight chilly sensations, accompanied by yawning. At this hour his temperature was 100° F. and pulse 72. At 3 P.M., temperature 98·8°, pulse 72. Says that he feels "out of sorts" but has no headache. At 5.30 P.M. his temperature was

100·6° F., pulse 78. He was not seen until 7.30 P.M., when he complained of backache and severe general headache, more intense through the frontal region. Eyes much injected, photophobia very marked, face flushed. He was stretching and yawning constantly, complained of nausea, and vomited a small quantity of partially digested food. The height of the primary fever was reached at 3 P.M., 10th February, *i.e.* 22 hours after the commencement of the attack, when the temperature was 102·8° and the pulse 98. Remission of the fever to 99·4°, and the pulse to 74, occurred at 6 A.M., 11th February, making the duration of the primary paroxysm 36 hours. Twenty-four hours after the remission had occurred, the temperature had risen to 102·4° with a pulse of 70. The fever continued to steadily increase until midnight of 12th February, when a temperature of 105° F. was recorded, with a pulse of 90. The subsequent course was that of a case of severe yellow fever. Slight oozing of blood from the gums occurred as early as the third day. Ocular jaundice, beginning on the third day became later very distinct, and was associated with general jaundice. Albumin, however, did not appear until the sixth day. The fever subsided on the ninth day, and was followed by a slow convalescence. The subject had been in strict quarantine for a period of 78 days prior to inoculation.

Cases of Yellow Fever Produced by the Injection of Blood
(Reed, Carroll, and Agramonte)

CASE I.—W. J., American, non-immune, aged twenty-seven —in quarantine since 20th December 1900—with his full consent at 11 A.M., 4th January 1901, was injected subcutaneously with 2 c.c. of blood taken from the general circulation of a case of mild yellow fever at the beginning of the second day of the disease and having a temperature of 100·8° F. The subject, who had been in strict quarantine at the station for a period of 45 days, remained in his usual health until the early morning of 8th January, when he complained of slight frontal headache. At 6 A.M. his temperature was 98·2° F., and pulse 70; 9 A.M., temperature 98·8° F., pulse 95; frontal headache increased, with slight, chilly sensations in the feet and lower extremities. There was some congestion of the eyes, and his usual florid

complexion was heightened in colour. At 10.15 A.M. temperature 100·6° F., pulse 97; complains of some pain in the lumbar region. At 11.20 A.M., temperature 101·4° F., pulse 99. The height of the febrile paroxysm was reached at 3 P.M. the same day when the temperature was 103·4° F., and pulse 108. The facial expression was now characteristic of yellow fever. The eyes were deeply injected and watery and the face much suffused. Photophobia moderate, frontal headache and backache severe. The skin was moist. The remission occurred at the end of 24 hours—9 A.M., 9th January—when the temperature had fallen to 99·4° F. and the pulse 86. The subsequent history was that of a case of yellow fever of moderate severity. Albumin was found in the urine at the end of the sixty-first hour. There was some bleeding from the gums on the third day and moderate ocular jaundice on the fourth day. Fever disappeared on the morning of the seventh day.

CASE II.—W. O., American, non-immune, aged twenty-eight, in quarantine since 20th December 1900. On 8th January 1901 at 9 P.M., with his full consent, he was given, by subcutaneous injection, 1·5 c.c. of blood taken from the median cephalic vein of Case I. just 12 hours after the beginning of the attack, and when the temperature was 102·4° F.—that is, just after the first febrile paroxysm began to decline. The subject remained in his usual condition the following two days.

11th January 1901.—Six A.M. his temperature was 98·9° F. and pulse 70. He complained of being disturbed by dreams during the night and had some frontal headache. At 9 A.M. temperature 100° F., pulse 77. At 10.15 A.M. temperature 101·4° F., pulse 76. Eyes decidedly congested and face moderately suffused. At 12 o'clock noon, when the temperature had risen to 103·2° F. and the pulse 102, the height of the primary paroxysm had been reached. Headache and backache were now much complained of. The facial expression was characteristic. The remission occurred at the end of 24 hours, lasted one day, and was followed by a very moderate secondary fever. A distinct trace of albumin was found in the urine passed at 2 A.M., 12th January, 17 hours after the attack began. A few hyaline casts were also present. Slight bleeding from the gums

occurred on the second and third days of sickness. The character of the attack in this case was very mild. The albumin, which at no time amounted to more than a distinct trace, did not disappear, however, until 24th January.

CASE III.—W. F., American, non-immune, aged twenty-three, was, with his full consent, at 1 P.M., 22nd January 1901, injected subcutaneously with 0·5 c.c. of blood taken on the second day from the general circulation of a severe case of yellow fever, which was fatal on the seventh day of the disease. The patient's temperature when the blood was withdrawn was 103° F. and pulse 90°. The subject remained well during the following day, 24th January ; at 6 A.M. his temperature was 98·4° F. and pulse 78. He partook of a hearty breakfast at 6.30 A.M. which he vomited soon afterwards. At 7 A.M. he complained of dizziness and general lassitude; temperature 98·4° F., pulse 78. At 9 A.M. chilliness complained of, but there is no record of temperature or pulse. At 9.30 A.M. temperature 100·6° F., pulse 82. Frontal headache well marked. Eyes already injected and face slightly suffused. At 10.30 A.M. temperature 101·2° F. and pulse 86. An hour later his temperature was 102·6° F. and pulse 82. The height of the primary paroxysm was reached at 1 P.M. when the temperature was 102·8° F. and pulse 98. At this hour photophobia was well marked, and constant complaint made of severe frontal headache and backache, together with pains in the lower extremities. The skin was moist. The remission occurred at the end of 36 hours. The subsequent course was that of a case of yellow fever of moderate severity. With the return of the secondary fever there was present sharp backache and headache. Albumin appeared in the urine at the end of 57½ hours. Ocular jaundice was present on the third day and thereafter until convalescence. The gums did not bleed, although they were swollen and injected. Fever subsided on the sixth day, and albumin disappeared on the eighth day.

CASE IV.—J. H. A., American, non-immune, aged twenty-two, with his full consent, received subcutaneously, at 12.15 P.M., 25th January 1901, 1 c.c. of blood taken from the median cephalic vein of Case III. just 27¼ hours after the commencement of the latter's attack of yellow fever; temperature

100·6° F. The subject remained in his usual condition during 26th and 27th January, except that on the afternoon of the last mentioned date he complained of occipital headache. This was present on the following morning, 28th January; otherwise he felt well. His temperature at noon was 98·6° F. and pulse 68. Occipital headache continued. He partook of dinner with fair appetite. He was not seen again until 3 P.M. In the meanwhile, at 1.15 P.M., the patient states that, while sitting alone in his tent, he began to feel cold, and that this was quickly followed by a decided chill with increase of headache. He noted the hour in writing at the time. At 3 P.M. his temperature was 103·6° F. and pulse 120. The eyes were intensely congested and face deeply suffused. The patient was very restless, and complained bitterly of occipital headache and backache. Photophobia was very marked. He vomited several times within the next 2 hours. Skin hot and dry. The height of the primary paroxysm was reached at 3.30 P.M., at which hour the temperature was 104·2° F. and pulse 120. The subsequent history was one of severe yellow fever. There was no remission of the fever until the fourth day, when the temperature fell to 101·2° F. Now, for the first time, the patient ceased to complain of occipital headache and backache. Albumin appeared at the end of 18¼ hours (7.30 A.M., 29th January). A few hyaline casts were also present at this time. The specimen of urine passed at 6.40 A.M., 30th January, contained albumin one-twentieth by volume, and many fine and coarse granular, bile-tinted casts. Ocular jaundice appeared on the third day. The skin of the face and of the anterior part of the neck and thorax was tinted on the fourth day. This rapidly became intensified and general. The secondary fever lasted about 30 hours, the temperature falling to 97·2° F. at 12 o'clock (midnight) of the fifth day. Marked fluctuations of temperature continued until the eleventh day of illness. Recovery was slow and much delayed by the development of a carbuncle in the left sacral region. A trace of albumin was still present on 1st March, 32 days after the attack had begun.

As the result of the preceding and of further experiments, it is proved that during the first three days of illness, the blood of

the person suffering from yellow fever contains the virus, whatever its nature may be; that the *Stegomyia* may become infected, and may transmit the virus after an incubation period of at least twelve days to a non-immune. Similarly when during the three first days of illness blood was withdrawn and injected into a non-immune, fever also resulted, as is shown by the preceding experiments of Reed, Carroll, and Agramonte.

These observers also showed that whatever the nature of the virus it could pass through a Berkefeld filter, for the filtrate when injected produced an attack, and the blood of such a case when inoculated into another person, also produced a reaction.

It has also been shown that ·1 c.c. of blood-serum taken on the first day of illness from a case of yellow fever has when inoculated subcutaneously induced yellow fever.

References

Experimental Yellow Fever

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Report of Commission of Medical Officers, "The Cause of Yellow Fever," Marine Hospital Service, Washington, 1899.

Carroll (J.)—"History, Cause, and Mode of Transmission of Yellow Fever," *Journal of the Association of Military Surgeons*, 1903.

In all the preceding experimental cases the Cuban board of yellow fever experts had no doubt about the nature of the disease produced. Yet we must agree that the symptoms are very mild, and not very specific. Headache, backache, chill, lassitude, a rising temperature, the pulse not following, a trace

or slight albumin in the urine, occasional vomiting, jaundiced scleræ, flushing, gums spongy, and showing a tendency to bleed, a remission, and then a second attack.

These are certainly not symptoms which a medical officer would pronounce to be those of yellow fever unless he was looking out for the disease.

The great authority, Carroll, has stated that their experiments show that genuine yellow fever may be so mild in character that no man, no matter how extensive his experience may have been, would dare to diagnose it as such, unless he knew the disease to be prevailing at the time.

Such cases appearing at the beginning of an outbreak, would render it extremely difficult or impossible to trace the origin of the severer cases occurring later.

In the light of the above precise experimental data we may then, I maintain, be prepared to find that yellow fever has masqueraded under many euphonisms, as the following shows :—

II.—Local, "Endemial" or "Acclimatising" Fevers of Tropical Towns

Inflammatory Fever, Fièvre Inflammatoire

The "inflammatory fever" of the West Indies has been long recognised as an acclimatising or seasoning fever which newcomers were expected to get. It was another name for some local or endemial malady, about which no one knew anything precise, except that all were expected to get it at one time or another.

The older writers wrote much about it, and held that it did not bear any strict affinity with the dreaded yellow fever. The symptoms recorded were lassitude, chill, injected eyes, flushing, fever, pains, frontal headache, lumbar pain, nausea, vomiting, epigastric tenderness, urine scanty and high coloured. These symptoms might become more marked, headache more violent, also the epigastric tenderness, persistent vomiting, delirium,

elevation of temperature. Later, again, marked yellowness of the skin might set in. In still more severe attacks persistent black vomit and hæmorrhages, tarry stools, violent delirium, coma, and death occurred. In this picture of the inflammatory fever of the West Indies the reader will agree there are the various types of yellow fever—the mildest to the most severe forms.

More recent writers have drawn attention to this disease, because it explained the recent outbreak, 1909, of yellow fever in Martinique and Guadeloupe. Most of our knowledge is due to Simond, Aubert, and Noc, who have been engaged on more than one yellow fever investigation, and who were in the West Indies when I was there last year on yellow fever inquiries. These investigators show that the symptoms of inflammatory fever are identical with those of experimental yellow fever. They give cases where a mild attack of inflammatory fever passes into the severe form of yellow fever. The symptoms can be so mild as to produce only an "embarras gastrique."

The symptoms are usually slight fever, vomiting, albuminuria, jaundice on the fourth or fifth day, a remission, then a rise. The disease is not fatal, nor does it confer absolute immunity. It may occur quite independently of any case of severe yellow fever and may occur in epidemic form; it often precedes an outbreak of malignant yellow fever, but it may not do so.

They conclude that under inflammatory fever may sometimes be concluded either mild cases of yellow fever or cases of malaria. They also state that yellow fever is characterised more by the milder symptoms than by the malignant.

References

Inflammatory Fever

Simond, Aubert, et Noc—"Contributions a l'étude de l'épidémiologie amarile," *Annales de l'Institut Pasteur*, 1909.

Dickinson (Nodes) — *Observations upon the Inflammatory Endemic Incidental to Strangers in the West Indies, commonly called the Yellow Fever*, London, 1819.

As in the case of experimental yellow fever and in that of inflammatory fever, the patients are as capable of giving infection to the *Stegomyia* as virulent cases of yellow fever.

There can be no doubt, therefore, both from the evidence of old clinical investigators, as well as of modern experimental investigators that this endemial acclimatising or seasoning fever is in reality very often genuine yellow fever, and is the source from which *Stegomyia* becomes infected. Like experimental yellow fever, it would be overlooked did not severe types of yellow fever occur from time to time.

The continuous existence of inflammatory fever is as significant as the existence of severe yellow fever, and proves that yellow fever is endemic. In West Africa attention has more than once been directed to inflammatory fever.

III.—Bilious Remittent Fever

This fever takes the place to a large extent in West Africa of the inflammatory fever of the West Indies. It is a very old name, and typified the "acclimatising," "seasoning" or "endemial fever" of any locality which newcomers were expected to get. Volumes have been written upon it. Some of the older writers held that just as in inflammatory fever, it could pass into severe yellow fever; others held that it was a specific entity.

"*Borras*" *and* "*febre biliosa*" *of Brazil.*—The majority of large tropical towns, in Central and South America and in the West Indies have each had their own special local or *acclimatising* fever. It is most significant that in these same centres of population, *yellow fever* was the disease which sooner or later attacked the newcomer. We further know that the *Stegomyia* was the common mosquito of these places. In Cuba the local fever was called "Borras." In Brazil, "febre remittente bilioso dos pezos quentes" or "febre amarelle dos acclimatados." The evidence is in favour of these being mild types of yellow fever, common amongst the native inhabitants, and giving them the

necessary immunity against the severe or fatal type. Therefore, in epidemic times they escaped, whilst the mortality rate amongst the new arrivals was appalling. Examples of this have been recorded at Rio, Santos, Vera Cruz, Para, Havanna. Now, however, thanks to anti-*Stegomyia* measures, natives and newcomers are on the same footing, both are now non-immunes, and equally susceptible.

Some of the older observers held that bilious remittent fever, carried from one locality, could burst out into the malignant yellow fever on arrival in another country. All these doubts and theories pointed to the fact that the older clinicians were observing one and the same disease, but of different types of severity. From the investigation of careful records of outbreaks of yellow fever, there can be no other conclusion, than that by the name bilious remittent fever has passed a very large number of cases of genuine yellow fever.

Bilious remittent fever may also and does, just as inflammatory fever, include a proportion of malaria cases, but it undoubtedly has very frequently been used as another name for yellow fever.

For example, Colonel Birt in a recent paper, published in the *Journal of the Royal Army Medical Corps*, refers to the outbreaks of yellow fever in Malta, and how in 1881 there are entered 69 cases of bilious remittent fever with 6 deaths. He points out that primary malaria is exceedingly rare in Malta; and is limited to one remote valley. He concludes that the term "bilious remittent" was only a euphonism for yellow fever.

Carroll refers also to this disease in the following decisive terms.: "We know, as a matter of fact, that here in the United States, yellow fever has been time and time again called 'bilious remittent fever,' until the occurrence of a number of fatal cases with black vomit proclaimed the true nature of the disease. There is good reason to believe that with the approaching complete control of yellow fever, the old time bilious remittent fever will become much less frequent in the

United States." Rush, who had unique experience in yellow fever epidemics in the United States, regarded bilious remittent as yellow fever. Another American authority in describing the great epidemic of yellow fever at Baltimore, mentions how it commenced with a long series of bilious remittent cases. From Horton's description of bilious remittent fever in his work on the tropical diseases of warm climates, there can be no doubt that he is describing yellow fever.

Carroll further states:—"Recent experiments have shown that genuine yellow fever may be so mild in character that no man, no matter how extensive his experience may have been, would dare to diagnose it as such, unless he knew the disease to be prevailing at the time." Elsewhere he states:—"Bilious remittent fevers, epidemic in character and accompanied by jaundice, of short duration, occurring on vessels, in seaport towns, and along the lines of travel from them, are closely related to and probably identical with yellow fever."

I am informed that as a matter of fact bilious remittent fever has largely disappeared from the Gulf Ports of the United States.

In an official report, drawn up for the Marine Hospital Service in the United States in 1898, the following statement occurs:—"The bilious remittent fever, that in our old text books of medicine occupied such a conspicuous place in tables of differential diagnosis with yellow fever, has practically disappeared from the Southern Sea border, since yellow fever ceased to be an endemic there. It was in fact the yellow fever of the natives and of places in the interior." This is a most significant statement, for it shows that bilious remittent fever has in places diminished or disappeared *pari passu* with yellow fever.

Of course the malarial forms of bilious remittent may, and do continue to exist.

Pipe-borne water supplies appear to have affected bilious remittent fever, precisely as they have done yellow fever.

Let us now examine bilious remittent fever in the light of old

and recent observations, more especially as it affects West Africa. One of the best accounts of this disease will be found in the first volume of the *British Medical Journal*, then the *Provincial Medical Journal*, 1842, by E. J. Burton, Surgeon to the Royal African Corps.

He classifies the fevers of West Africa into (1) bilious remittent fever; (2) simple bilious fever; (3) "intermittent fevers"; (4) yellow fever.

With regard to yellow fever, he states that it generally appears in epidemic form every seven years in Sierra Leone. He mentions that its origin had given rise to much debate: some regarded it as imported, but the majority were of opinion that it had a local origin. Burton believed in the local origin, and the great majority of writers both before and after Burton's time have held a similar opinion. He then goes on to state that "yellow fever has been considered by many as an aggravated form of remittent fever, and the idea is supported by the fact that all the symptoms are similar, only much more severe in the former. This error, however, for error it must be considered, originates, I am led to think, in mistaking the severer types of bilious remittent for yellow fever, and it must still be considered that the latter is a specific disease—one, in fact, *sui generis*."

Burton describes how bilious remittent fever or "country fever" proves fatal every year to a large number of people, and how every year it appears in an endemic form. It can never be said to be wholly absent, and not unfrequently *appears in the dry season*. He further points out how Boyle, a well-known authority on the fevers of West Africa, proposed to divide the endemic bilious remittent into:—

(1) *Local bilious remittent; and*

(2) *Climatorial bilious remittent.*

It is most significant to note that Boyle found this division necessary, for how otherwise could he explain the occurrence of bilious remittent in ships.

So, therefore, he proposed the name "climatorial bilious remittent" for those cases developing on ship board who, not having set foot on land, nevertheless developed the fever. This is to my mind the most striking evidence in support of my contention that this fever is none other than yellow fever.

It was as common on ships in the nineteenth century as yellow fever. Naturally it was because it always has been present wherever the *Stegomyia* is found.

Burton gives the symptoms as follows:—

"The symptoms are extremely varied, and scarcely ever appear in the same order in any two persons attacked. There is, however, a sufficiently well-marked similarity in all cases to lead, after a little experience, to an immediate and correct diagnosis.

"Sometimes, indeed in the majority of cases, the disease is ushered in by a sudden chill, generally referred by the patient to the small of the back, in some cases extending along the whole course of the spine, and likened to the trickling of cold water down the back. The cold feeling just described sometimes amounts to a complete rigor, but in the greater number of cases the chill is only momentary, and at other times no sensation of cold whatever is felt. The rigor, or momentary feeling of cold, as the case may be, is soon followed by reaction; at first, alternate flushes of heat and cold are perceived; by degrees the skin becomes either moderately or excessively hot; if the latter takes place, the patient feels as if surrounded by furnaces, and tosses about in the most restless manner. There is usually a dizzy feel of the head from the beginning; but as the hot fit advances, headache, sometimes of the most excruciating description, sets in; there is usually a feeling of stupor during the afternoon, at which time the fever is at its height.

"The disease sometimes commences with vomiting, generally of bile, either in a moderate or excessive degree; but there is at other times merely a degree of nausea, and in some cases neither of these symptoms appear. At the beginning there is

usually a ringing sound heard in the ears; the eyes are dull, and more or less sunken; the conjunctivæ are red, and appear injected; the countenance is expressive of suffering and anxiety; the tongue puts on different appearances in most cases, and usually forms a good indication as to whether the disease is likely to prove severe or otherwise; if the latter, it is at the beginning of the fever white in the middle, and as the disease advances it becomes coated with a brown fur; but if the tongue is red, or coated with a white tenacious fur, with extremely red edges, then a severe and dangerous attack may be apprehended. Sometimes, during the progress of a dangerous attack, the tongue becomes dry and black. No symptoms portending worse consequences than this can show itself; but, happily, even under such discouraging circumstances, the case is not to be considered as necessarily fatal.

"There is usually an unpleasant feeling at the pit of the stomach; sometimes a burning sensation is complained of; in other cases it amounts to a feeling of pain, especially on pressure. The bowels are nearly always extremely costive, though the disease is now and then ushered in by bilious dejections. The urine is high-coloured and scanty; in some rare cases there is frequent and painful micturition, evidently depending on irritation or subacute inflammation of the mucous membrane of the bladder.

"The thirst is in most cases excessive; some patients will swallow gallons of fluid in the course of the twenty-four hours. Pains in the different muscles are often felt; but few cases are unaccompanied by great pain in the joints, and those of the knees seem to suffer most.

"Sometimes delirium appears in a few hours after the commencement of the attack. There are, however, few patients who pass through the disease without being more or less affected with this disagreeable symptom at some period of the fever, especially during the night.

"When it occurs only at night, it is a combination of delirium

and unpleasant dreams, extremely annoying and fatiguing to the sufferer. If delirium appears during the day it is fierce, and attended with much greater excitement and mental aberration.

"In some patients the disease sets in suddenly and violently; sometimes the actual period of invasion cannot be ascertained with any degree of precision. In these latter cases the person feels for a day or two a certain degree of langour and lassitude, with feebleness of the lower extremities, especially from the knees downwards, and partial loss of appetite. These symptoms become gradually increased, and finally so aggravated that the patient is obliged to keep his bed, and a regular attack of fever sets in.

"In the beginning of the disease there appears very little alteration in the pulse; but when the febrile symptoms become fully developed, it is either quick, full, and bounding, or exceedingly quick and hard; at other times it is only accelerated and full. The state of the pulse, however, depends more on the constitution and temperament of the person attacked than on the comparative severity or mildness of the case."

The symptoms described may appear either in a mild form at first, and afterwards become partially or greatly aggravated, or they may from the first set in with the utmost severity.

In some cases the irritability of the stomach increases to such a degree, that matter, strongly resembling the "black vomit" in yellow fever, is thrown up, and frequently a yellow tinge of the skin, chiefly about the neck and chest, is perceived. It is necessary therefore to bear in mind that there are two ways in which this fever commences.

The first is where it sets in with violent symptoms, accompanied by great heat of skin, headache, delirium, etc.: this may be denominated the tangible form, and is certainly the least dangerous, if promptly and energetically treated.

The second is when the symptoms are all more or less obscure, there is but slight heat of the skin; when the hand is applied there is a feeling of dryness rather than heat. There

is a constant dull pain in the head, scarcely amounting to headache; no delirium is present, but there is slight confusion of ideas during the night. The tongue is usually, in this form of the fever, red, or the edges are very red, and the middle of the tongue is white; in fact, all the symptoms of fever are present, but in so low and obscure a form that the nature of the disease might be mistaken until too late. This is decidedly the most dangerous kind of fever, and may be called the obscure form.

With regard to the remissions, he states that a remission may occur or the type may be continuous. If it does occur, it appears generally on the third day, when the diminution of symptoms is such that the patient feels well enough to rise from bed.

Burton lays stress upon a very significant fact in connection with bilious remittent fever, namely, the generally accepted doctrine, with which he agrees, that one attack of the fever confers immunity against a second attack. This is of course of great importance, because it points to a disease other than malaria, and is in harmony with our knowledge of experimental yellow fever, inflammatory fever, and yellow fever in all its types.

Burton thus describes "simple bilious fever."

He first mentions that some might regard it as a milder form of the bilious remittent; he, however, was inclined to regard it as a specific entity. The disease is ushered in with headache, vomiting of bile, fever; it attacks suddenly, usually with vomiting, retching, and nausea. In the majority of cases pain in the head, sometimes very severe. Delirium is not common, pulse quick and full. Tongue coated, urine high-coloured, scleræ slightly jaundiced.

Returning to bilious remittent fever, Burton states that it is the endemial remittent fever of all tropical countries, occasionally occurring in northern climates during an *unusually* hot autumn.

He describes it as met with in the West Indies, in a severe form, in the East Indies in a mild form. It causes much mortality in British Guiana and British Honduras, and in the southern parts of the United States; it has appeared in an extremely severe form in some of the Spanish provinces.

In other words, with the single exception of the West Indies, bilious remittent fever has been common wherever yellow fever has been present.

He then states that of all countries, it is most severe in West Africa, that a more treacherous fever cannot be conceived. He then appends the following tables, drawn up by Fergusson, for the troops of the Royal African Corps for 1825 :—

Stations.	Strength.	Treated.	Died.	Deaths to Cases Treated.	Deaths to Strength.
First Quarter.					
Sierra Leone. .	138	...	...	...	...
Gambia. . .	...	...	...	...	...
Isles de Los . .	108	...	...	...	...
Second Quarter.					
Sierra Leone. .	289	46	13	1 to 3·61	1 to 22·2
Gambia. . .	140	3	...	...	...
Isles de Los . .	106	5	2	1 to 2·5	1 to 53
Third Quarter.					
Sierra Leone. .	585	386	161	1 to 2·39	1 to 3·63
Gambia . . .	108	92	74	1 ,, 1·24	1 ,, 1·45
Isles de Los . .	103	99	23	1 ,, 4·3	1 ,, 4·47
Fourth Quarter.					
Sierra Leone . .	522	146	37	1 to 3·94	1 to 14·1
Gambia. . .	112	89	60	1 ,, 1·47	1 ,, 1·86
Isles de Los . .	75	120	7	1 ,, 17·1	1 ,, 10·7

From the above table, drawn up by Surgeon Fergusson of the Royal African Corps, it will appear that in a regiment never exceeding 796 men, there were treated in one year 986 cases of fever, and of these cases 377 died, giving a mortality of nearly one-half the total strength from one disease alone.

Although authorities might be multiplied in proof of the extremely dangerous and fatal nature of the fever in question,

one extract only, from the report of a medical officer of talent and high standing in the service, will be given.

These reports were made about sixteen years ago, but recent events upon the coast fully prove that the climate remains the same, and that Europeans cannot approach these regions of malaria without exposing themselves to the greatest danger.

"The arrival of twelve sergeants," says this officer, "forms a striking feature in the events now under consideration, and affords a gloomy illustration of this deleterious climate. They were selected from the detachments at the Isle of Wight; men of good character; their conduct, at least, when on this coast, was as exemplary as could be expected from men of their class in society. Some irregularities, of course, occurred; but had they been sinners above all sinners, we could not have expected so desolating a retribution.

"The whole of them were attacked with fever, and within a few months after their arrival, eight paid the debt of nature, and only one at present (the sergeant-major) appears fit for duty. The constitutions of the other three are in such a shattered state that plainly indicates an advanced stage of visceral disease. The sickness and mortality amongst their wives and children are nearly in the same proportion.

"This affords a very fair specimen of what may be expected from the effects of the 'bilious remittent fever' of Western Africa, without taking into consideration the many other diseases which attack the European residents.

"In fact, this is nothing but the process of 'seasoning,' through which every stranger must pass in a few months after his arrival in the country."

Impartially reviewing Burton's descriptions of bilious remittent fever in the light of experimental yellow fever and inflammatory fever, and bearing in mind that Burton's observations were long before the *Stegomyia* doctrine, or the discovery of the parasite in malaria, one is forced to the conclusion that by this name is described some of the typical types of yellow fever.

In order, however, to still further bring home the fact that, in my opinion, under the term "bilious remittent fever," yellow fever is very frequently included, I adduce the following further evidence.

Boyle, a well-known authority on tropical fevers, and Naval and also Colonial Surgeon of Freetown, devotes a whole chapter to climatorial bilious remittent fever.

By this term he meant a fever which broke out on ships of the type of bilious remittent fever. This is very significant, because it shows the great difficulty which Boyle had in conceiving why bilious remittent fever should occur spontaneously on board a ship, the crews of which had not been ashore for any length of time.

Naturally it is the old story of yellow fever over again; how to explain the occurrence of yellow fever on ships, likewise how to explain bilious remittent fever on ships. It would be quite impossible to explain either, unless on the doctrine of the presence of the *Stegomyia*.

But let us analyse some of Boyle's cases, abstracted from his journal on board the H.M.S. *Cyrene* in 1822-1823.

CASE I.—J. J., seaman, æt. thirty-five, had been ashore fourteen days previous to sailing; taken ill with headache and pains in body, fever; pulse 90; later delirium. Ended fatally.

CASE II.—J. A., at sea. Symptoms:—Intense headache, lassitude, thirst, fever, pains in body, nausea. Recovery.

CASE III.—S. S., at sea. Severe pain in head, back and loins, weakness, griping and purging, fever. Next day pulse reduced in frequency. Recovery.

CASE IV.—W. H., on board a schooner at Sierra Leone. Symptoms:—Intense pain in head, back, and loins, fever, pulse 130, vomiting, great thirst. Later, severe vomiting and epigastric tenderness developed, stupidity, pulse and temperature reduced. Post-mortem:—The stomach was deeply congested towards pylorus.

Amongst the numerous cases of bilious remittent fever which developed on the *Cyrene*, there occurred one case of fatal yellow fever, characterised by black vomit and jaundice.

Surely this case gives the clue to the origin of the cases of so-called climatorial bilious remittent which occurred on board. Very many other cases are given, together with the post-mortem findings, and there cannot be a doubt that they were genuine malignant yellow fever cases as well as milder types. These cases occurring side by side with fatal cases, which were either diagnosed at the time as yellow fever, or which, from the description of the post-mortems, I have no doubt were yellow fever, is unmistakable evidence of the yellow fever nature of the climatorial bilious remittent fever.

Boyle then devotes a chapter to his local bilious remittent fever, and here again it is abundantly evident that he is dealing with one of the common mild types of yellow fever.

He describes it as the acclimatising fever of the locality, which every newcomer had to get. He lays particular stress upon the fact of the exclusive predisposition of the English and Northern races to the disease. The symptoms are chill, malaise, pain in head and body, fever, bilious vomiting, congested eyes, yellowness of skin and eyes. In some cases tarry stools and dark vomit, delirium.

He then notes how sporadic cases of yellow fever break out, and he calls them aggravated cases of the endemic bilious remittent. So here again it is clear that we are dealing with one and the same disease, viz., yellow fever.

Boyle gives pages of description to this endemial remittent fever, and it is clear that it passes insensibly into the epidemic form or yellow fever.

The evidence of Surgeons Barry, Lawson, Fergusson, Tidlie, Burnett and many others, given either in books or in MS. records, all points to the one conclusion: that cases of bilious remittent fever and yellow fever occurred together. When the fever appeared in epidemic form or accompanied by black

vomit, it was called yellow fever, otherwise it remained simply climatorial or endemic remittent fever — a repetition of the picture of "inflammatory fever" in the West Indies: all degrees of severity from "embarras gastrique" to yellow fever.

All authorities were agreed that the native very rarely suffered from the severe black vomit type, and they also observed that the bilious remittent gave a certain degree of immunity against yellow fever. Now to turn to the recent evidence furnished by the 1910 epidemics.

In the first place, at the commencement of the outbreak of yellow fever in Secondee, came a stout denial on the part of the Press and some of the medical men, that yellow fever existed or even could exist on the coast.

They seriously declared that the disease was only the malignant form of bilious remittent fever with which they were well acquainted, and which usually attacked persons towards the rainy season. This statement is quite true, if it is accepted that the bilious remittent fever is true yellow fever and has, as the newspapers state, been no doubt present every year.

I have carefully examined a large number of case-books and other records at Secondee, Accra, Cape Coast, Saltpond, Elmina, Axim, Quittah, Lagos, and Freetown, and I have found that amongst the cases returned as yellow fever, there were others diagnosed as bilious remittent fever or simply remittent fever.

At Lagos I was particularly struck by the very large number of remittent cases. Of course a certain proportion of these are in all probability malarial, but with equal certainty we can say that a proportion are mild cases of yellow fever.

It is beyond dispute, then, that during outbreaks of well-marked black vomit, there have occurred in the present year and in past years numerous cases of "remittent" and "bilious remittent fever." This is not a new observation peculiar to the West Coast: the same fact has been observed wherever yellow fever has been present.

I will now give an example of one of the cases of yellow fever which occurred in Secondee during the recent outbreak.

The history and symptoms of the case were :—

Mr D. had been in the colony a few months on this occasion, but had been on the coast before. Taken suddenly ill, 19th May ; 21st May, vomited, severe headache, temperature 101°, jaundice, no albuminuria ; 22nd May, temperature 102°, jaundice deepened, conjunctivæ congested, urine scanty, no albumin ; 23rd May, temperature 102°, albumin present in the urine, vomited material like beef-tea dregs ; 24th May, temperature 103·4°, urine scanty, very albuminous, jaundice, pulse 70, vomits speckled with black spots ; 25th May, temperature 101°, restless, urine scanty ; 26th May, temperature 98·8°, pulse 80, urine loaded with albumin, scanty, vomited, petechiæ present on belly, jaundice deep, hiccough ; 27th May, temperature 98·8°, pulse feeble, urine suppressed, hæmorrhage from mouth, death.

Post-mortem.—Surface of body yellow, petechiæ, liver yellow and congested, recalling the appearance of nutmeg liver, neck of the gall bladder deeply congested. Stomach contained a copious quantity of black gramous material, also the intestines, "stomach and intestines stained dark." There can be no doubt about this case belonging to the severe type of yellow fever. It was one of several that occurred one after the other, but nevertheless a native medical man comes forward and states as follows :—

"The case of the late Mr D., whose autopsy I witnessed yesterday, was one of *malignant bilious remittent fever*, and not one of the true pestilential yellow fever. That, therefore, all the preceding cases which were on all fours with Mr D.'s case were malignant bilious remittent fever and not yellow fever."

This is the comment of a medical man who is not an exception ; he is an example of many who go about encouraging the belief that yellow fever does not exist. It is of course clearly obvious that this attitude proves that malignant bilious remittent fever is only yellow fever under another name.

We may conclude, therefore, that the bilious remittent fever

of West Africa is, in a *very large number* of cases, genuine yellow fever in its various degrees.

Additional note on remittent fever.—Careful examination of the account of fevers in West Africa for the past hundred years up to the present day, shows the extremely ill-defined position which bilious remittent and remittent fevers occupy. To this day they are regarded as local in origin, the product of the coast, in other words endemic.

Some medical men would say that they are the result of infection by anophelines, that is that they are malarial; but others again would point out that very frequently no malarial parasites are found in the blood, and that therefore they must have another origin.

But there are very few who take into consideration the possibility of some of the cases being due to infection by the *Stegomyia*, that is that they may be in fact mild forms of yellow fever. If the view is correct, it accounts for the difficulty which the older clinicians experienced in differentiating between remittent and yellow fever.

The history of an epidemic shows that very often an outbreak of yellow fever was preceded by an outbreak of remittent and bilious remittent fevers. Medical officers, moreover, often described remittent fever ending fatally with symptoms of typical black vomit. In every outbreak of yellow fever they discuss the relationship of the one disease to the other, and even go so far as to point out that an attack of remittent fever confers a certain degree of immunity against yellow fever (Burton).

In 1837 Fergusson describes how in 1836 the barque *Mary* arrived at Freetown. In fifteen days the whole of the crew were attacked with endemial remittent fever. Of the 15 attacked 10 recovered and 5 died. Fergusson adds that the disease was not accompanied by black vomit; but shortly afterwards another ship arrived, the *Lady Douglas*, and several of the crew died with the severe symptoms of yellow fever. About the same time the H.M.S. *Curlew* sailed from Freetown for the

Gambia, and her crew went down with malignant remittent fever. On arrival at Bathurst the disease developed in its typical malignant yellow fever form.

Fergusson states how the yellow fever outbreak in 1837 in Freetown died down and "was succeeded by the common remittent fever"; but to everyone's astonishment, the malignant symptoms again made their appearance after a few months' apparent cessation, and again the whole of a ship's crew is reported to have died of malignant remittent.

In subsequent years the military reports contain frequent references to remittent fever ending fatally with all the clinical symptoms of yellow fever; it is also mentioned how closely they resembled yellow fever, and how frequently they occurred in ships' companies. In other cases, again, half the cases are diagnosed as remittent and the remainder as yellow fever cases.

Staff-surgeon Lawson mentions how in 1847, 12 whites died from yellow fever and 5 from remittent fever, and how the disease first started as the ordinary local remittent form and these developed into the malignant.

It is a well-known observation that frequency of the occurrence side by side of bilious remittent or remittent fevers and yellow fever both on land and on ships. Remittent fever was a "ship's fever," as much as yellow fever. Furthermore, the remittent fevers have appeared at places where there was no endemic malaria there.

Blair draws attention in his tables to this prevalence of remittent fever side by side with yellow fever in Barbados, where we know there are no anophelines.

Thus out of a garrison of 2310, in the year 1816, there occurred 470 cases of remittent and 705 cases of yellow fever, whereas only 36 cases of intermittent fever are recorded, which latter were in all probability imported cases.

In West Africa the case is similar, whilst no doubt a considerable certain number of cases of remittent fever are malarial.

On the other hand, the evidence is very strong that a certain number of them are genuine cases of *Stegomyia* fever, *i.e.*, yellow fever, and that both whites and blacks are liable to it, but especially the former.

Remittent fever on the Gold Coast.—A return of diseases under the heading of malaria furnished by the hospitals at Accra, Cape Coast, Elmina, Axim, Kwitta, Secondee, and Comassi furnishes the following results:—

Year.	Remittent Fevers.	Intermittent Fevers.
1904	241	235
1905	231	161
1906	273	225

A return on the same lines from the Gold Coast Colony, Ashanti, and the northern territories gives in 1907 a total of 1104 cases of remittent fever and 1223 cases of intermittent fevers treated.

Remittent fever in Sierra Leone.—Examination of the cases admitted into the Missionary Home at Freetown (for whites only) shows a very considerable number of cases diagnosed as remittent fever, and one or two cases of bilious remittent fever each year. The figures for remittent fever are:—

1902	16
1903	29
1904	34
1905	32
1906	21

Remittent fever in Southern Nigeria.—Here also examination of hospital case-books shows very large entries under the heading of remittent fever. This is especially noticeable at the Lagos Hospital.

In view of what has already been said concerning remittent fevers, the question remains: Are all these cases of remittent fever malarial? Or may not a considerable portion of them be

due to the *Stegomyia?* For it must always be remembered that the *Stegomyia* is the most common mosquito, and that precisely similar returns of remittent fever were furnished by places where there were no anophelines, and where therefore the remittent fever must have been due to the *Stegomyia*, which we know was abundant.

It is now incumbent upon medical officers to bear these facts in mind, and to endeavour to divide the remittent and bilious remittent fevers into those which are genuine cases of malaria, and those which by exclusion may be classed as *Stegomyia* fevers.

Another interesting point to observe will be: whether a reduction in bilious remittent and remittent fevers will follow the putting into practice of rigorous anti-*Stegomyia* measures.

On going over records of the health of our troops in the West Indies and other stations, it is very clear that remittent fevers and yellow fever occurred and disappeared together.

The great authority upon yellow fever, Bancroft, held strongly the view that remittent fever and yellow fever had a common origin.

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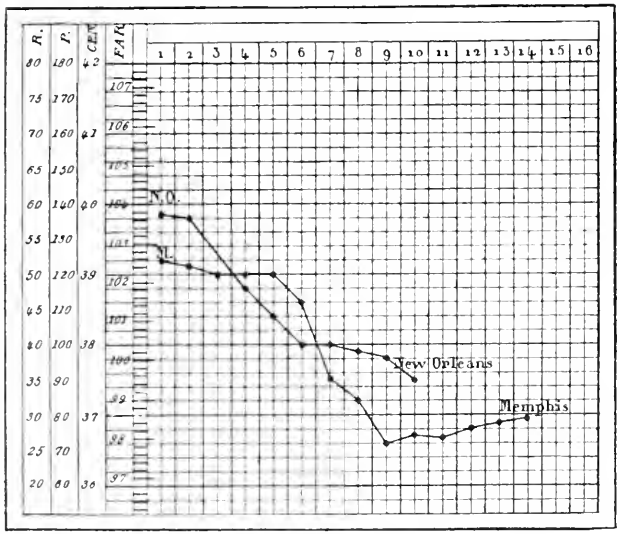

FIG. 7.—Average temperature curves for the yellow fever epidemics of New Orleans and Memphis (1870-73), based upon a very large number of records. Observe that the temperature falls after the first two or five days respectively.—FAGET.

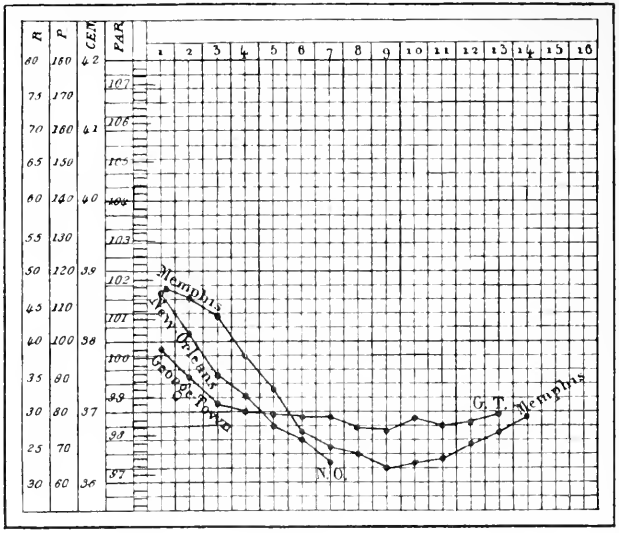

FIG. 8.—Characteristic pulse curves, based upon a very large number of observations in the yellow fever epidemics of New Orleans, Georgetown, and Memphis (1870-73). Observe that the pulse falls from the initial rise.—FAGET.

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CHAPTER IX

THE CLINICAL HISTORY OF VARIOUS EPIDEMICS OF YELLOW FEVER

Symptomatology of Yellow Fever: General

UNDER experimental yellow fever, inflammatory fever, and bilious remittent fever, I have already described very many of the symptoms of yellow fever. The point to bear in mind in yellow fever is the wide range of symptoms, from an "embarras gastrique" and feeling of lassitude to the severe symptoms culminating in black vomit and suppression of urine.

The symptoms usually commence with a chill and feeling of lassitude, headache, lumbar and joint pains, which become more intense. The temperature is above normal, and continues to rise from twenty-four to forty-eight hours, reaching 104·5° or even higher, but not usually. The temperature may continue for four or five days more, with slight variations, or as may happen, there is a remission, or the temperature curve may present several remissions. The pulse is that of any fever, it is quickened 100 to begin with, but as the fever progresses the pulse slows down to 90, 80, 70, or 65, or even lower, whilst the temperature may stand at 100°, 102°, or 104°.

This want of accord between pulse and temperature, although by no means constant, is exceedingly characteristic, and serves to stamp the disease as very suspicious of yellow fever. It is known as Faget's sign. The tongue is coated in the centre and the edges and tip are red. The gums are sore, and often

present a fine red line of congestion where the lead line is usually found.

Nausea and vomiting may commence very early. At first the contents of the stomach, then thin mucus, or bile-stained mucus, then on the fourth or fifth day, or even earlier, small black specks, are observed in the vomit. Subsequently the typical black vomit sets in, or blood may be vomited. The stools are now often tarry.

The urine is a febrile one to commence with, then after the end of forty-eight hours or later a trace of albumin is usually found. This increases, and at the same time the urine diminishes in quantity, and finally may end in total suppression.

Yellow discoloration usually sets in about the third day, and is first noticed in the scleræ, then it spreads to the rest of the body. It is characteristic to observe the deepening of the colour as the disease progresses. Or the yellow colour may not appear till after death, or during the death agony. When once seen this symptom can never be forgotten.

Congestion of the eyes and of the upper part of the body is often seen early and petechiæ may appear. Hæmorrhages are common from the nose, mouth, and other orifices.

The remission, referred to above, may last for several hours, and very often gives a false sense of security. I have noticed it many times, and it is a characteristic and suspicious sign. The patient feels well, wants to get up and take a meal. It is succeeded by a relapse and the temperature rises. This is sometimes known as the second paroxysm, or the reactionary fever.

To sum up—yellow fever may be described as a continuous fever of one paroxysm, lasting more than twenty-four hours. The onset is sudden and accompanied by violent headache, pain over the eyes; eyeballs tender to pressure; violent pain in the back (*coup de barre*); epigastric tenderness; anorexia; nausea and vomiting. This is sometimes described as the *first stage.*

In addition the pulse rate is increased, the conjunctivæ are

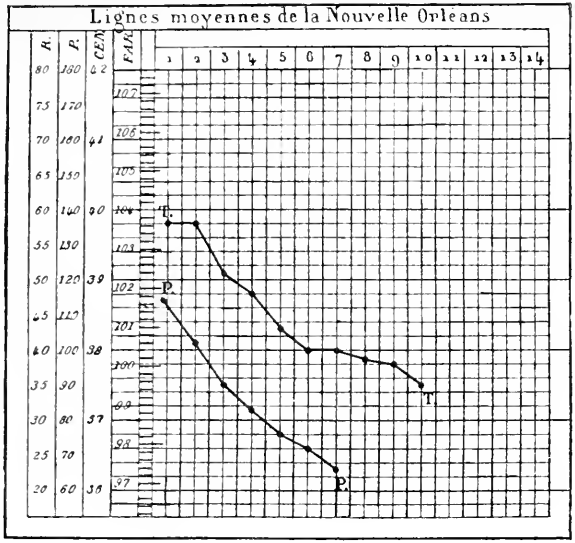

FIG. 9.—Average temperature and pulse curves in the non-fatal cases in the New Orleans 1870 epidemic of yellow fever. The temperature falls from the second day, the pulse immediately after the initial rise.—FAGET.

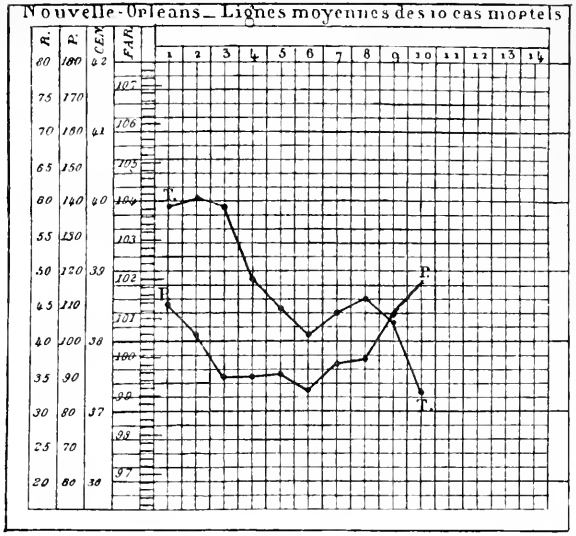

FIG. 10.—Average temperature and pulse curves in ten fatal cases of yellow fever in the New Orleans 1870 epidemic. The temperature falls from the third day. There is a relapse on the eighth day, then the fatal drop. The pulse falls from the initial onset, but rises again when the temperature begins to fall.—FAGET.

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congested; the eyes often brilliant; the urine high coloured, but no albumin; the face and upper part of the trunk may be flushed; there is restlessness. This stage is in fact one of stimulation, the virus is commencing to take effect on the tissues and organs: it is succeeded by the second stage or stage of depression, in which the organs show that their metabolism is now profoundly affected by the virus, the symptoms develop from day to day.

The headache and pains continue and increase; the restlessness becomes more marked and often passes into delirium of a violent character; the gastric symptoms intensify; the vomiting is more severe; minute specks of blood begin to appear in it for the first time; later the whole vomit becomes dark, like "coffee grounds," owing to the escape of blood into it derived from the intensely congested stomach wall. This constitutes the "black vomit": it is essentially a sanguinolent, serous discharge from the congested mucous membrane, comparable to that which takes place from the nasal cavity, or the bronchioles, or the intestines, when the mucous membranes of these tracts are the seat of intense congestion.

Flakes of lymph are found in the vomit, and these together with the mucous and red corpuscles constitute the coffee-ground deposit. Sometimes the vomit is clearer and more serous-like, sometimes darker: this, we can readily understand, depends upon the degree of inflammation of the mucous membrane. In some cases the hæmorrhagic appearance of the vomit is marked, just as in hæmorrhagic pneumonia: in this case the vessels bleed into the stomach.

In connection with the mechanism of vomiting, it is often noted that the expulsion of the contents of the stomach takes place suddenly without any effort. I have myself seen this many times. The vomit wells up and gushes out over the bed clothes or on to the floor; hiccough often now becomes very troublesome and intractable.

Sometimes the black vomit stage is not reached, and it is

only at the post-mortem examination that the black vomit is found in the stomach.

Black vomit is usually compared to coffee grounds; sometimes it is described as bog water or porter like. It has been mistaken for altered red wine, and the unfortunate patient has been picked up and lodged in jail as drunken.

The congestion of the mucous membrane of the stomach may extend to the gut, and dark tarry stools may take the place of the intensely fetid stool of the first stage. No doubt some of the black matter present in the stools is derived from the stomach.

The tendency to congestion is not confined to the alimentary tract; the conjunctivæ and skin may be congested, and this may markedly increase; petechiæ or small subcutaneous hæmorrhages develop, especially in dependent and injured parts; the gums, nose, and eyes may also begin to bleed.

The effect of the virus begins also in the second stage to take effect on the vascular system, the liver, and kidneys. The temperature is rising or remaining constant, but the pulse does not quicken to correspond with it; on the contrary, it becomes slower. This characteristic slowing of the pulse may be due to the markedly altered metabolism in the liver, whereby the bile salts are absorbed and lead to the slowing of the pulse.

Jaundice may now appear in the scleræ, and may extend and deepen from day to day until the whole surface is yellow, the colour of the urine proceeds *pari passu*. Sometimes, as stated, the yellow coloration only appears during the death agony or just after death.

The progressive character of the lesions is also observed in connection with the kidneys.

The urine may remain free from albumin for the first three or four days, and then the albumin may begin to show itself, at first as a trace, gradually increasing in amount; it is essential therefore to test the urine frequently throughout the day. The quantity of urine passed may begin to fall off and be suppressed

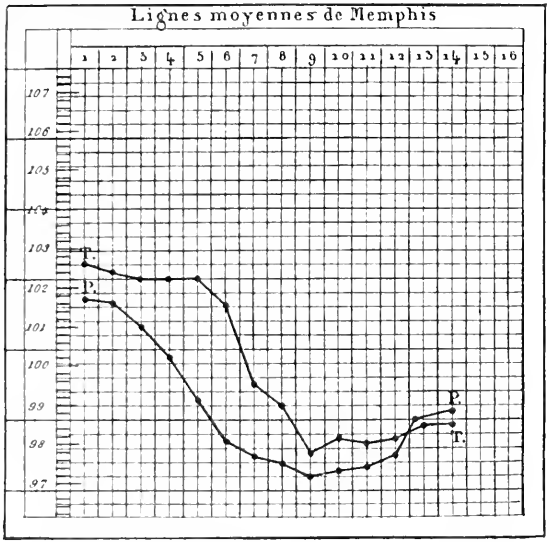

FIG. 11.—Average temperature and pulse curves in the non-fatal cases in the Memphis epidemic of yellow fever, 1873. The temperature descends after the fifth day; the pulse after the initial rise.—FAGET.

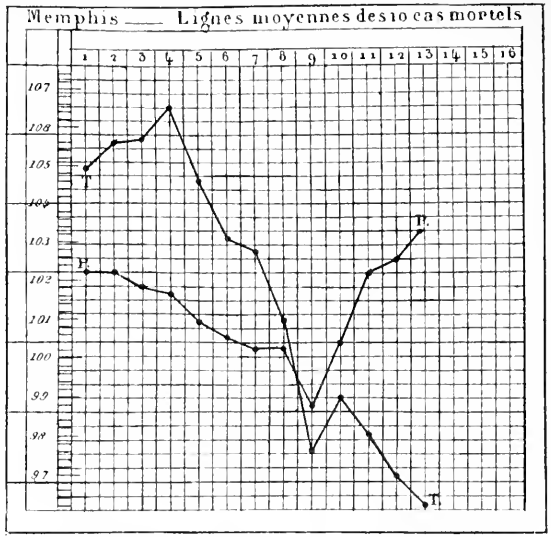

FIG. 12.—Average temperature and pulse curves of ten fatal cases of yellow fever in the Memphis epidemic of 1873. In the temperature curve the fall occurs after the maximum rise on the fourth day. On the ninth day the fall is very marked. The pulse curve is the opposite; there is a fall from the first initial rise, and a marked rise on the ninth day.—FAGET.

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for one or more days. If this occurs, the mental symptoms begin to gain in severity, the delirium becomes maniacal, and is usually followed by coma and death.

In the following pages I have grouped together the symptoms which have been observed in cases of yellow fever which have occurred in Cuba, Barbados, British Honduras, New Orleans, and in West Africa. Their study will afford an exceedingly accurate picture of the disease.

The account of the symptoms of the cases seen in West Africa in the years 1823, 1837, and 1847, are taken directly from the reports of Staff-surgeons Barry, Fergusson, and Lawson respectively.

These reports, which for neatness and powers of observation are of a very high order, are preserved in the medical military reports of the garrison of Freetown, and for permission to see them I am indebted to Colonel Sutton, R.A.M.C., and the General in command. This is, I believe, the first time that the reports have been reproduced.

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Symptomatology of Yellow Fever in the West Indies and Central America

Guiteras has recently published a clinical résumé of 275 cases of yellow fever, as well as the findings of a life's experience in yellow fever countries. In the following paragraphs I have

embodied the observations of Guiteras, and have added the results of my own experience in Barbados and British Honduras.

Guiteras defines yellow fever as a fever of from two to seven days, characterised by sudden onset, a fastigium of from one to four days, followed by an irregular lysis, sometimes interrupted by a secondary exacerbation.

A steady fall of the pulse, commencing in the fastigium; vomiting, jaundice, albuminuria (tending to blood stasis), and finally hæmorrhages. His clinical picture of the disease is as follows:—

"During the early hours of the morning the patient awakes with a slight rigor, and, on moving, experiences vertigo and numbness, and heaviness of the lower extremities. This is followed by nausea, and in some instances by vomiting of the remains of the last meal; the temperature rises rapidly; frontal headache, rachialgia, and pains in the limbs develop, and the pulse becomes frequent. The face assumes an injected, turgid appearance; the eyes are red and moist. The patient looks like a person who had indulged in an alcoholic debauch.

"During the day the fever continues to rise, and the patient complains of discomfort, pain or burning in the epigastrium, with sensitiveness to pressure. The temperature rises between 102° and 103° F. and the pulse to 100 or 110. After six or nine in the evening of the first day, the temperature may fall somewhat. This may amount to a distinct remission.

"After the initial elevation of the temperature, jaundice will develop sooner or later, and the course of the disease may vary according to three different types: the *descending or mild type*, the *continued type*, and the *remitting*, complicated or *secondary fever type*.

"The vaso-motor erethism will begin to subside, together with the painful symptoms, after the diurnal elevation of temperature of the second day, and is replaced either by the evidences of a gradual return to the normal, or by the signs of blood stasis with hæmorrhages from the mucous membranes, or with the syndrome of a malignant icterus.

"The urine becomes albuminous on the second or third day

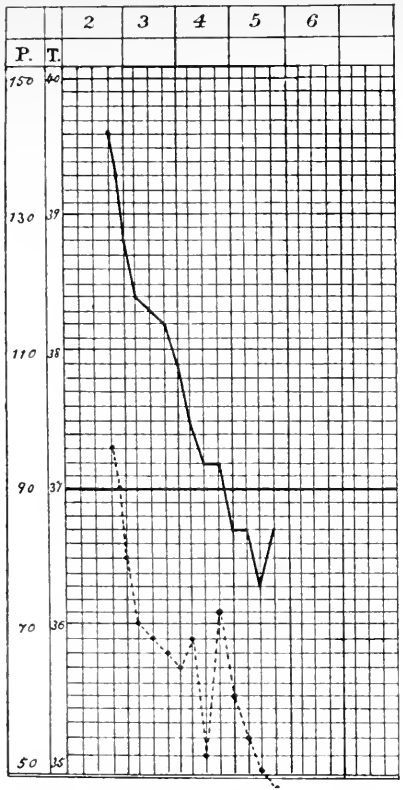

FIG. 13.—Temperature and pulse curves of the mild or abortive type (acclimatising or inflammatory fever, etc.).—GUITERAS.

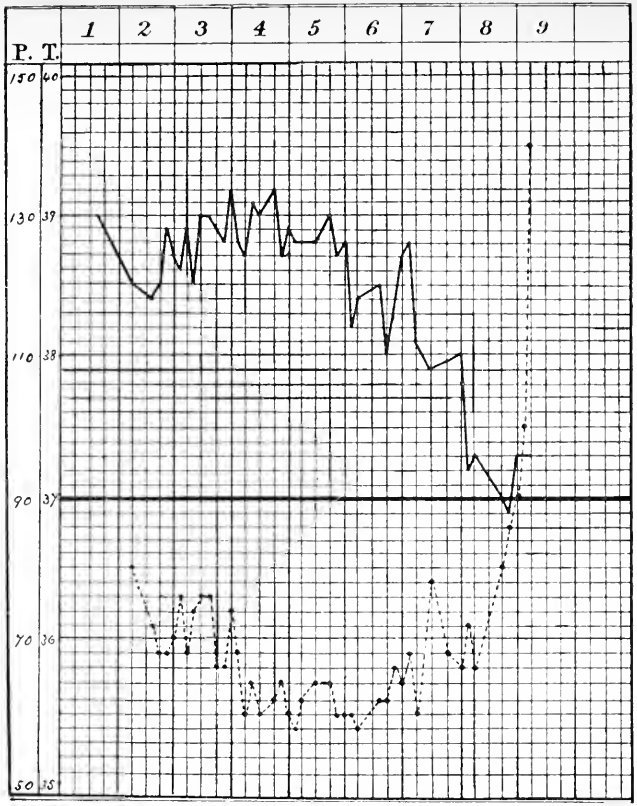

FIG. 14.—Temperature and pulse curve in a fatal case of yellow fever of the "continuous type." Observe the complete divergence of the pulse, and then its rapid rise during the fatal termination, and when the temperature is falling.—GUITERAS.

[*To face p.* 132.

of the disease. The mental attitude is usually one of alertness. Even when the patient is delirious the expression of the face is apt to be attentive, though the mind be utterly confused and the speech wild. In some cases there is somnolence.

"The pulse begins to fall on the second day, and continues to fall even though the temperature may rise. Recovery is usually rapid, and sequelæ are rare. Independently of the course of the temperature, we may recognise certain types, such as the uncomplicated, the hæmorrhagic, the icteric, the ataxic, and, as rarer forms, the anuric, the dystolic, and the fulminating. The various combinations of these, however, are of too frequent occurrence to give these types any practical value."

Guiteras points out that the old classical description of yellow fever, as a fever of two paroxysms separated by an interval of calm, is really based upon clinical observation, and not upon the use of the thermometer.

The temperature chart is, in fact, like that of scarlet, or of the other specific fevers. In other words, it is a single paroxysm fever; but there is a *remitting type*, which shows a fall of temperature on the third, fourth, or fifth day. In this case the second exacerbation may be due to hæmorrhages or to secondary infection.

As in all fevers, however, so in yellow fever, there are oscillations, and there is nothing absolutely diagnostic in the chart.

The *descending type* is mild, and corresponds to, no doubt, the acclimatising or endemic fever common to yellow fever countries. The temperature reaches its maximum on the evening of the first or second day, and then falls with oscillations. It is a mild attack, and, no doubt, confers a certain degree of immunity. Sometimes these cases end fatally.

In the *continued type* the high temperature is protracted; this form is usually fatal, and the delirium, albuminuria, and suppression of severe yellow fever may be present.

Guiteras regards the want of correlation between pulse and

temperature as one of the most constant features of yellow fever. He points out, however, that a sudden fall of temperature accompanied by a rise of pulse rate is obviously a very serious symptom.

Guiteras also emphasised a fact which is brought out in some of the clinical histories in the following pages—namely, the sense of false security which an apparently good, full, and regular pulse of 70 will induce in those who are not on their guard.

The same observer lays stress upon the characteristic facies of yellow fever, the ferrity eyes, and the constant presence of some shade of yellow in the skin or in the scleræ.

"The dead body is always yellow in yellow fever." Vomiting is by no means a constant symptom, persistent vomiting is a grave symptom: the first signs of the black vomit are the appearances of the "fly specks." Black vomit may be absent in severe and even fatal cases, but in these cases the black fluid will certainly be found in the stomach at the autopsy.

Some Cases seen in Barbados, 1909

CASE I.—A. L., black native, æt. twenty-one.

27th February.—Felt unwell.

4th March.—Admitted into hospital; temperature 101°; pulse 100; backache; headache; epigastric tenderness; vomiting; slight icteric tint; suppression of urine; a small quantity was withdrawn by the catheter: it contained blood and much albumin. Later in the day black vomit appeared and a tarry stool passed.

5th March.—Urine withdrawn by catheter: it contains blood; great restlessness; temperature 103°; pulse 104, and weak.

7th March.—Urine passed in bed; temperature 105°; pulse 96.

8th March.—Urine containing blood withdrawn by catheter; temperature falling; pulse rapid, 120; death.

This case shows a diminishing pulse with rising temperature: there was a temperature of 105° with a pulse of 96. This was

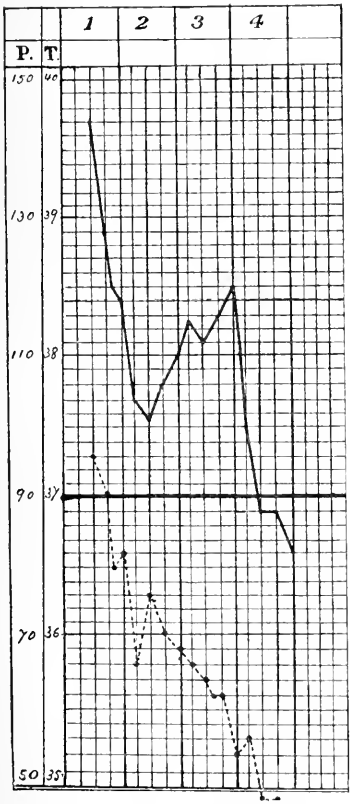

FIG. 15. — Temperature and pulse curves in the "remitting type." The remission occurs in this case at end of first day. Note the divergence of the pulse.—GUITERAS.

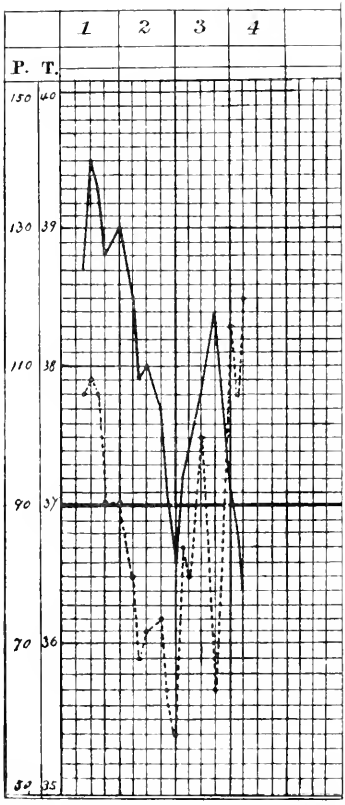

FIG. 16. — Temperature and pulse curves in a fatal case of the "remitting type." The remission took place on the second day. Note the fall of the temperature and rise of the pulse at the fatal termination.—GUITERAS.

[*To face p.* 131.

soon followed by the reverse action, the temperature fell rapidly and the pulse rate quickened : these were very serious symptoms. In addition the kidneys were severely affected, as is shown by the blood and albumin and the suppression. The chart is very characteristic.

Post-mortem examination made by me three hours after death showed :—Subcutaneous tissues distinctly stained yellow ; liver congested and of a yellow shade of colour ; on section it presented a typical boxwood colour. Kidneys congested. Stomach contents consisted of dark bloody fluid ; the mucous membrane deeply congested ; pericardial fluid slightly bile-stained ; substance of left ventricle soft. Spleen normal in size and firm.

The above case, both from the clinical as well as the post-mortem findings, is a typical case of yellow fever, and moreover of yellow fever in the black subject. The case emphasises the fact that the black is not necessarily immune to yellow fever.

Case II.—Mrs G., a native resident.

27th February.—Patient taken ill with chill, followed by headache ; epigastric pain ; nausea ; examined by the doctor who found temperature 102·5° ; pulse 80 ; albumin present in the urine.

3rd March.—Chocolate-coloured vomit ; temperature 101·5° ; pulse 64 ; much nausea and epigastric pain ; albuminuria.

Case III., *27th February.*—Taken ill with rigor, pain in the loins ; headache ; epigastric tenderness ; no vomiting. Later the scleræ became jaundiced and albumin made its appearance in the urine ; temperature 103·4° ; pulse 88. The pulse and temperature rate whilst the patient was under observation was as follows :—

Temperature 100·4° ; pulse 80.

Temperature 103° ; pulse 80. Temperature 102° ; pulse 60.

Some Cases observed in British Honduras, 1905

Case I.—C. R. D., æt. thirty-eight.

14th May.—Taken suddenly ill with chill ; temperature 103·4° ; pulse 120 ; headache.

16*th May.*—Temperature 103°; pulse 80; tongue furred, edges red.

18*th May.*—Temperature 101°, then rose to 102·4°; pulse 72 to 74. Scleræ yellow, no albuminuria. Later temperature fell to normal, and patient recovered.

CASE. II.—J. W. C., æt. thirty-two, recent arrival in Belize, but many years on East African coast.

11*th February.*—Woke up with chill; temperature 103·6.° pulse 72; eyes congested and scleræ yellow; urine dark; trace of albumin; headache; great thirst.

12*th February.*—Fair night; got up; scleræ deeply jaundiced.

13*th February.*—Feels better, but headache persists; no albuminuria.

14*th February.*—Patient delirious; temperature 102°; urine diminished; much albumin.

15*th February.*—Vomiting of food; drowsy, followed later by convulsions and death.

CASE III.—Miss B., recent arrival.

4*th May.*—Taken ill; temperature 103°; pulse 120; gastric irritability; much prostration; no headache or backache.

7*th*, 8*th*, 9*th May.*—*Pulse almost normal*, and patient appeared to have recovered.

10*th May.*—Temperature 103°, and remained almost stationary till death.

14*th May.*—Vomited an "enormous" quantity of brown fluid, and passed a large quantity of same fluid material; the pulse rate rapid.

In this case the "period of calm" was on the 7th, 8th, and 9th, and no doubt the almost normal pulse put the medical attendant off his guard.

CASE IV.—Rev. C.

16*th May.*—Taken suddenly ill with rigor; temperature 104°.

19*th May.*—Temperature 102°.

20*th May.*—Temperature 104°; *pulse* 48; black vomit

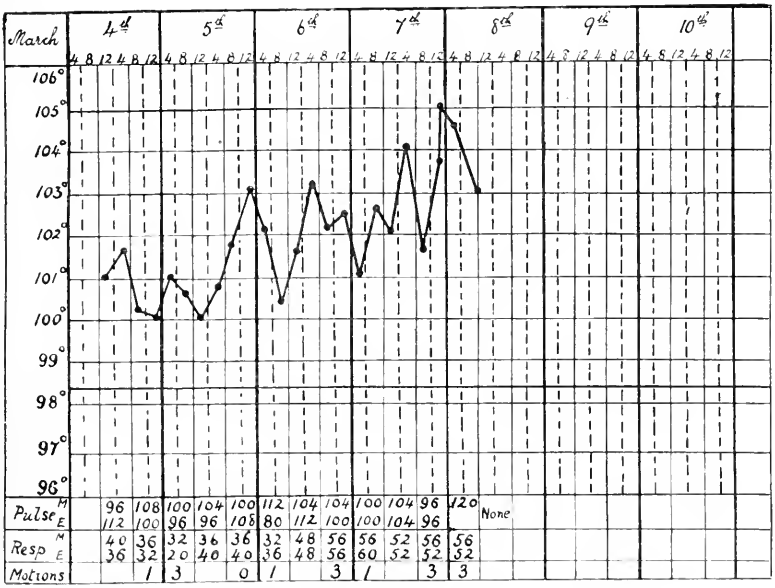

FIG. 17.—Temperature Chart of Case I. A.L. Barbados.

[*To face p.* 136.

albuminuria; petechiæ on palate; conjunctivæ congested and yellow.

27*th May*.—Death. *Post-mortem*.—Stomach contained a large quantity of black vomit; liver fatty, said to have presented a typical appearance.

CASE V.—F. B., new arrival.

18*th May*.—Taken ill with headache; sleeplessness and constipation; temperature 101°; pulse 100.

19*th May*.—Feels better.

21*st May*.—Temperature 103·4°; pulse 76; no albumin.

22*nd May*.—Temperature 101·8°; pulse 76; albuminuria.

23*rd May*.—Temperature 100·2°; pulse 74; albuminuria.

24*th May*.—Temperature dropped to subnormal; pulse 89.

25*th May*.—Temperature rose to 102·4°; pulse 90; delirium; death.

CASE VI.—Miss N. S. E., æt. twenty, recent arrival.

22*nd May*.—Taken ill; feeling bilious; headache; face flushed; eyes congested; tongue furred; temperature 103°; pulse 110. Later scleræ jaundiced and orescia.

24*th May*.—Vomiting commenced.

25*th May*.—Incessant vomiting of dark flocculent matter.

26*th May*.—Vomiting worse.

27*th May*.—Vomit completely black; urine decreased; albumin present; delirium.

28*th May*.—Suppression of urine; death.

CASE VII.—Major B., æt. forty-one.

24*th May*.—Taken suddenly ill with chill; temperature 103·4°; pulse 102.

25*th May*.—Remission occurred; temperature 99°.

28*th May*.—Temperature rose to 103·4°; pulse 100; gastric hæmorrhage set in.

31*st May*.—That is the eighth day of illness; black vomit commenced; albumin increased in quantity in the urine, followed by suppression and death on 4th June; that is the eleventh day of illness.

The following are a few cases described by Dr Durham during his investigations in Para, Brazil. Dr Durham and his companion, Dr Myers, also themselves at the same time became infected, Dr Myers fatally.

CASE I.—Robust middle-aged woman; arrived from France, 1st September 1900; has not used a mosquito net.

11th September.—Quite well at 9 A.M., taken with some shivering about midnight; vomited bilious matter; severe headache.

12th September.—Severe, intense frontal headache; no pain elsewhere; facies, nil; chest, nil; abdomen, nil; tongue flabby; skin, moist, sweating slightly; temperature, 38·7°; pulse, 118. Four P.M. headache still severe; pain and tenderness at epigastrium; pain in loins and calves; face a little flushed, but no marked facies or thoracic injection; rather intense photophobia; tongue, furred; skin, hardly moist; vomit, yellowish, with a good deal of mucus; no ague parasites could be found in blood films; temperature 37·6°; pulse 108.

13th September.—Headache less; skin moist; no icterus; no conjunctival injection; no pain; no vomit; complains of weakness; temperature, 37°; pulse 84. Evening, temperature 37°; pulse 98.

14th September.—Some headache; no icterus; tongue slight fur, edges red; papillac swollen; tenderness and pain at epigastric angle; no albumin (boil and nitric); temperature 37°; pulse 98.

15th September.—No icterus; no fever; no albumin; gums rather swollen. Recovered.

CASE II.—Lives in a chalet which is very full of mosquitos, partly *S. fasciata*, chiefly bred in the ant guards of the pillars supporting the house, and partly with *C. fatigans*, probably from the stables. Arrived in Para from France, 14th July 1900. Sleeps with inefficient mosquito net.

15th September.—Taken suddenly ill in evening; at 8 P.M. temperature 40°.

16*th September.*—Face and eyes injected; skin flushed; no headache or pains; no vomit; skin dry; tongue, central fur and very red edges; no icterus; no albumin; gums not swollen; slight injection of fauces.

17*th September.*—Still flushed; hardly conscious; some albuminuria; no icterus; spleen and liver, nil.

18*th September.*—Still flushed; temperature falling; fair quantity of albumin in urine; no icterus.

19*th September.*—Feels better; no icterus; gums not swollen.

20*th September.*—Some epistaxis; gums slightly swollen; (?) slight conjunctival icterus.

21*st September.*—Some epistaxis; gums much swollen.

22*nd September.*—No epistaxis; bleeding now from gums.

23*rd September.*—Distinct conjunctival icterus, gums bleeding; albuminuria.

26*th September.*—No icterus; feels weak.

2*nd October.*—No albumin; appetite returned.

CASE III.—Middle-aged man, arrived with wife from France.

1*st October.*—Taken ill in the afternoon; temperature 39°.

2*nd October.*—Complains of frontal headache and lumbar pain; temperature normal; no sweating; examination of blood for ague parasites negative; spleen and liver, nil; eyes rather injected.

3*rd October.*—Eyes injected; epigastric pain and frontal headache; tongue, much moist white fur; no icterus; much bilious vomiting; no albuminuria.

4*th October.*—Headache persists; no icterus; bilious vomiting; returns all ingesta; abdomen not tender; tongue, much white fur; acute epigastric pain relieved by vomiting; much albumin in urine; blood examination negative.

5*th October.*—Less pain; abdomen rather tender; insomnia; much albumin; no vomiting; no icterus.

6*th October.*—Pain slight; generally better; albumin moderate; no icterus; weak and emaciated; further history, no icterus; no hæmorrhage; gums, nil.

Staff-surgeon Barry on the Symptomatology of the 1823 *Epidemic in Freetown, West Africa*

"In the 1823 epidemic in Freetown, the attack was generally ushered in with pain in the loins, limbs, but more particularly in the calves of the legs; sometimes distressing pain in the head, and at others with great uneasiness about the præcordia, with occasional vomiting and irritability of stomach, the tongue in general, white and tremulous, in some cases red and clean, and in a few an ingrained blackness was perceptible; but perfectly different from that collection of sordes which forms a characteristic of the typhus gravior, no symptom of which was apparent during any stage of the disease.

"There was no great degree of heat of the surface of the body, but on grasping the limbs or body firmly, a very peculiar sensation of stinging heat was communicated to the hand, which is retained for a considerable time. The pulse hurried and sometimes full, but seldom indicating any considerable degree of inflammatory action. No remarkable appearance of the urine; the eyes were generally suffused, and in most cases there was considerable anxiety during the first stage of the disease.

"This train of symptoms generally lasted for the first thirty-eight or forty hours, by which time the aperient medicines which had been administered had freely operated, but in vain were healthy bilious evacuations sought for, the disease now became evidently manifest in the thin dark fluid which was passed downwards in considerable quantities and rendered particularly characteristic by innumerable small floating flocculi which had very much the appearance of the washed and broken down fibrin of the blood.

"The patient about this period felt much relieved, and appeared unconscious of his danger, and this delusive state often gave sanguine hopes to the attendants. This calm was followed by a morbid torpidity, or sometimes by low delirium; this state continued until the fourth day, when the inevitable forerunner of a fatal termination, the "Black Vomit," made its appearance, at first in small quantities and mixed with the ingesta, but afterwards in amazing volumes, and ejected from the stomach with a most extraordinary spasmodic force.

"The fluid has the appearance of broken down and diluted coagulum of the blood, and frequently with portions of coagulated lymph assuming very much the appearance of the inner coat of the stomach. The fluid gave a dark stain to the linen not easily removed, and had a raw, unpleasant odour, so perfectly peculiar that on entering the chamber the state of affairs become immediately manifest. In some cases a troublesome hiccough occurred, and in those the matter vomited was less abundant.

"The state of the patient's mind was most peculiar in this latter stage, and generally expressed himself as being much better, until, the vital flame gradually receding from the surface and extremities, dissolution took place, which in some cases was preceded by violent straining of the eyeballs, incoherent expressions, or by some convulsive motions; sometimes prior to this a dingy yellow appearance took place on the body, particularly on the neck and chest."

Staff-surgeon Fergusson on the Symptomatology of the 1837
Yellow Fever Epidemic, Freetown, West Africa

"Premonitory symptoms did not always occur, but when they did occur they were of short duration. The person about to be attacked having been in the enjoyment of ordinary good health, awoke some morning with a sensation as if all was not right with him. He was not absolutely sick, nor in fever, but had a sort of confused recollection that he had not passed a comfortable night—perhaps had dreamed more than usual—he felt languid, and yawned occasionally; tongue furred but not thickly so.

"This state was soon followed by a sense of dryness of skin, headache, pains in the back and loins, sometimes extending to the thighs, knees, and legs; he became alternately cold and hot; sometimes there was a rigor; at this early stage the eyes became suffused, and an increased number of small red points were developed on the conjunctivæ; the pulse rose rapidly to 96, 100, or 120; the temperature of the surface became rapidly and permanently increased; this stage was also in some cases accompanied by a sense of nausea, in others by copious vomiting of tea, lemonade, toast, water, or other ingesta, which

an unusual degree of thirst had induced the patient to swallow; the tongue now became more densely furred, and all the symptoms increased in intensity until what was mere malaise in the morning had by mid-day merged into an unequivocal paroxysm of fever; towards evening the symptoms became more aggravated, skin dry, parched, and hot; the carotids throbbed violently, the headache was also more severe, but this symptom, though severe, was usually pretty tolerable so long as the patient kept in the horizontal posture; but when he attempted to get up, the forcible closing of the eyes and the general appearance of the countenance bore sufficient evidence of the agony that was endured; pressure with the finger on the eyeball caused great pain; moving the eye upwards and outwards also caused pain in the eyeball. Besides these symptoms there was a sense of general distress wholly distinct from pain, which it is not easy to describe. There was seldom any tenderness or pain on pressure of the epigastrium or hypochondrium, and even when that symptom did exist, it was in a very trivial degree. The first night was always one of great suffering. As the night advanced the headache became excessive, and all the symptoms still more aggravated. In some cases the patient slept; in others he was utterly sleepless and restless, tossing about in the vain search for a cool place to lay his head on. But whether he slept or not, still the night was one of great suffering. Such sleep as that by which the patient was visited was anything but repose, for no sooner were the eyes closed than a thousand incongruous fancies flitted before the imagination. The patient in this stage is quite conscious that his fancies are delusive and incongruous, but no effort of reason can divest his mind of the certainty of their actual presence, and the constant and unavailing effort to rectify his perceptions is productive of the utmost distress. There is a consciousness of the incongruity of ideas, and a desire to exert the rational faculties, though these are not subservient to the will. One grade beyond this in the severity of the disease, and this last ray of consciousness is lost in delirium.

"Towards morning, or about 5 or 6 A.M., the patient generally falls into a deep sleep, sound and without dreaming. This may continue from one to three hours, and when he awakes, the whole surface is covered with a gentle moisture; the headache,

pain of limbs, and general distress are all alleviated; the pulse has subsided to 78 or 76; the tongue, however, retains its coating, and the eyes are probably more suffused.

"During the first twenty-four hours a large quantity of semifluid fæcal matter of a dark green colour is evacuated; succeeding evacuations become less fæcal and more watery until at length the dejections consist solely of a dark watery fetid substance, interspersed with whitish floculi; about this time the patient's countenance assumed a darker hue than usual, and the conjunctiva betrays a slight tinge of yellow. He is, however, on the whole, much better, and is more comfortable in all his feelings: he has not the slightest appetite nor desire for food, the thirst is moderate, and, in fact, he would be altogether well were it not for a very uncomfortable feeling of giddiness in his head when he attempts to rise. In some cases there is a second febrile paroxysm similar to the one described; in other cases there is even a third, but very commonly there is no decided second paroxysm. This is more especially apt to be the case in those instances where the disease terminates fatally at an early period.

"The second stage of the disease is of a most delusive nature: the skin is cool, in many cases it is moist; the pulse is commonly enlarged in volume, and *from* 68 *to* 76, rarely exceeding the latter; the thirst is moderate; the tongue in many cases moist, and not much loaded, with a scarcely perceptible redness around the edges and at the tip; it is in many cases remarkably tremulous when protruded; in fact a stranger to the insiduous nature of the disease would at once pronounce the patient in this state to be a safe case, and at the point of convalescence. Although there is little suffering and little complaint, yet when the patient endeavours to rise, his gait is unsteady, he staggers towards the closet stool as if he were intoxicated; the dejections continue dark, watery, fetid, and flocculent; the eyes are much suffused, and the colour of the countenance now deepens into the swarthy appearance of a dark Spaniard or Italian, or of a person deeply sunburnt. If there has been no second paroxysm the patient is now pretty comfortable during the day. His nights are, however, very distressing: he is restless or sleepless as before, or his sleep is again disturbed by incongruous, visionary fancies,

till, towards morning on the second or third day, a distressing irritability of stomach sets in. The pulse is not usually, however, affected by it either in strength, or volume, or frequency. The substance thrown off consists at first merely of the ingesta, mixed with a little froth or perhaps green or yellow bile. Careful and minute inspection will frequently at an early period detect among the substance vomited the incipient state of that which afterwards forms the "black vomit." It is difficult to describe this appearance in its early stage so as to make it intelligible, but a near approach to it may be seen in the boiled coagula which float in soup made of meat from which the blood had not been well washed, minute shreds of a similar looking substance from the incipient stage of "black vomit." The patient may continue in the state here described for one, two, three, or more days, the irritability of stomach and vomiting continuing with more or less severity until the full development of the "black vomit" throws a shade over the countenance of both patient and medical attendant. The patient now sinks gradually. Sometimes petechiæ appear.

"The tongue may be dry, hard, horny, the papillæ communicating, when touched by the finger, a sensation as if the finger grated on sandpaper; at other times it is clean and smooth, divested of even the smallest appearance of papillæ; again it is black and dry; again it is red like a piece of raw beef, enlarged in volume or shrivelled in size. Large quantities of coffee-ground-looking substance are thrown off the stomach; there is great anxiety of countenance; the breathing becomes oppressed and laborious; the whole surface is covered with cold, clammy perspiration; the pulse ceases at the wrist. The patient at this stage has usually been for some hours comatose, before the scene is closed in death.

"The above is merely an outline of the prominent and more common features of the disease, but innumerable varieties of detail are met with, not only in individual cases but also in the general character of the epidemic at different times of its appearance. Thus in 1829, the invasion of the disease was ushered in by greater severity of pain in the back, loins, thighs, knees, and legs than what accompanied its early stages either in the early part or towards the close of 1837.

"In one case there was most painful and protracted cramps of the calves of the legs, during which the gastrocnemii muscles were drawn up hard and round like a ball. In another case death was preceded by most painful spasms of the hands and arms. Nothing of the sort appeared in 1837.

"In the second visitation of 1837 there was for some time in nearly every case an exudation of blood, even in the early part of the disease, from the mouth, nose, and arms. The appearance of petechiæ also set in earlier and was much more common; whereas in the visitation of 1829 and in the primary one of 1837, I did not meet with a single case accompanied by sanguineous exudation.

"In the latter part of 1837, there occurred several cases in which the patient was immersed in perspiration and covered with petechiæ from the very commencement of the disease. In these cases the blood exuded from the mouth, but flowed from the nose in a stream, and that sometimes to an alarming extent. In many cases the urinary secretion was more or less suppressed. In some a period of three days and nights elapsed without a drop of urine having been voided; this state was not accompanied by any symptoms of retention.

"In many cases the nervous system was affected in an extraordinary degree: the staggering gait of the patient on an attempt to rise has been noticed, as also the remarkable tremulousness of the tongue when that member was protruded; a similar tremulous affection of the hands and arms was not uncommon. In several cases not only the conjunctiva, but the face and trunk also were deeply jaundiced; but in the generality of cases the colour did not deepen beyond the dark, swarthy appearance above noted.

"To enumerate all the varieties that have been met with would be an endless task. I shall therefore sum up this part of these observations with a statement of the symptoms which I consider as being pathognomonic of the disease, not that they are by any means of uniform or constant occurrence in every case, but because they have been less frequently absent than any of the others, viz.:—

"(1) The slow and languid pulse which succeeds the cessation of the first or second pyrexial paroxysm.

"(2) The delusive absence of obvious pyrexial symptoms which accompanies this state.

"(3) Concomitant with these the suffusion of eyes.

"(4) The 'black vomit.'

"The pathological appearance observed on dissection were few in number, but these were of such constancy and uniformity as might, *apriori*, be considered almost incompatible with the great variety in the symptoms during life as exhibited in individual cases. The head was rarely examined as, in those cases in which it was examined, nothing pathologically referrable to the disease could be detected. The leading feature in the post-mortem inspection was an obvious and unequivocal appearance of inflammatory action over the external as well as the internal surface of the stomach and intestinal canals. This was repeatedly noticed in every grade, from a minute arborescence of small vessels to a dark livid state, in which the coats of intestine were easily lacerable; in short to a state bordering on sphacelus. I met with but one exception to this condition of increased vascularity. The livid appearance when present was never observed on the stomach, rarely on the large intestine, and was most frequently found on portions of ileum. The coats of the intestine at these parts were very much thickened; the omentum was extremely vascular. The stomach in all cases was found to contain more or less of the dark, grumous substance called 'black vomit.' This substance adhered tenaciously to the hands, and was not easily washed off. After having submitted the inner coats of the stomach to repeated washings, a thick glairy mucus was found adherent to it, through which red patches were observed, varying greatly in size and shape, but generally more abundant towards the pyloric than the cardiac orifice. When the mucus was scraped off and those patches viewed through a magnifier, they appeared to consist of a congeries of minute red points. They displayed no appearance of arborescence, or of continuity of canal, and I could not help regarding them as so many minute orifices from which the blood in a state of fluidity was exuded, which afterwards constituted the substance called 'black vomit.' The contents of the intestinal canal throughout were of a dark colour, and the coats, both externally and internally, bore marks of vascularity

less equivocally indicative of precedent inflammatory action than the congeries of minute points of which the red patches in the stomach were formed. The liver was commonly of a pale colour, sometimes mottled. The gall-bladder not so frequently distended as in the ordinary remittent fever; its contents dark, viscid, and tar-like. The spleen rarely altered either in size or structure, and that never to a great extent. In one case there was a large flat sanguineous clot found under the dura mater, and in the same case a similar clot was found within the sheath of the rectus abdominis muscle."

Staff-surgeon Lawson on the Symptomatology of the 1847 *Yellow Fever Outbreak.*

"John Ogden, marine, of H.M.S. *Growler*, was attacked with remittent fever on 15th March 1847, and on examination was found to be labouring under the following symptoms on the 16th :—Considerable frontal headache, flushed countenance, skin hot and dry; tongue covered with a yellow fur in the centre, being slightly red and dry in the tip and edges; constant nausea, occasionally vomiting a bilious-looking fluid, accompanied by epigastric tenderness. Pulse quick and full; urine scanty and turbid, depositing a sediment on cooling. Bowels confined; occasional attacks of shivering, accompanied by pain along the spine. A sample of calomel was administered immediately, followed in three hours afterwards by a solution of sulphate of magnesia in peppermint water. A sinapism was applied to the epigastrium, and the body sponged with lime juice and water.

"In the evening a remission took place, and he felt himself much better in every respect; the bowels having been freely acted on by the medicines, producing several stools, the evacuations being fetid and of a dark brown colour.

"18*th March*.—Three days after the accession of the disease the symptoms daily decreased in severity from this period, and on the morning of the 21st he stated that he had slept well during the night, that he felt quite well, and wished for something to eat. He was allowed 2 oz. of arrowroot for dinner, which he enjoyed very much, and at the evening visit was quite free from any febrile symptom. About 11 o'clock the same

night he suddenly screamed in his sleep, started out of bed, and attempted to jump out of the window. On visiting him immediately, I found him labouring under great mental excitement—his face flushed, his eyes brilliant, skin dry and burning, with slight tremor of the hands. Pulse rapid, but soft and easily compressed. He spoke incoherently, and had to be restrained in bed. He vomited a considerable quantity of a dark coloured fluid, resembling *coffee grounds*. Notwithstanding the active remedial measures employed, symptoms of coma came on in a few hours afterwards, and he died at 9 o'clock on the following night—the 22nd."

Post-mortem Appearances

Cranium.—"The sinuses and central veins slightly congested membranes of brain perfectly healthy. There was about two teaspoonfuls of serum in the lateral ventricles."

Thorax.—"Lungs and heart healthy."

Abdomen.—"Stomach was found half filled with a dark coloured fluid, similar in appearance to that vomited, having some resemblance to the 'black vomit'; but on minutely examining it, it was found to consist of *shreds of lymph*, mixed with the secretion of the stomach and the medicines administered. The membrane covering the cardiac extremity was soft and easily separated from the cellular coat, and presented vascular patches throughout. The remainder of the abdominal viscera were perfectly healthy."

Comment.—The above case is of interest, because it shows in the first place that it was diagnosed as remittent fever, although there can now be no doubt that it was yellow fever. In the second place, it is clinically of interest because of the relapse or "feeling better stage." In this stage the fatal error of giving food was made; this invariably precipitates an attack of black vomit.

The History of the 1884 *Yellow Fever Epidemic in Freetown, West Africa*

The following report of the 1884 epidemic of yellow fever in Freetown is very valuable from many points of view. The epidemic to commence with affects both the blacks and the

whites, but the severe cases which develop are amongst the whites. There is not one fatal case amongst the black population, with the exception of a black *West Indian* soldier.

In my opinion this is conclusive evidence that the black population of Freetown had been protected by previous attacks of the disease, the Europeans, especially the newcomers, were not so protected, and naturally developed the severe and fatal type of yellow fever.

The West Indian soldier likewise was no doubt a non-immune, just as in 1909 in Barbados I found that the mortality was very high amongst the native black population, for they had been protected from the disease for many years, and naturally became non-immune. It is also most noteworthy that the Medical Board concluded that the epidemic was of local origin.

Thirdly, the Board, like many others, experienced the difficulty of distinguishing the endemial local remittent fever from yellow fever. They mention two cases of remittent fever which ended in black vomit. In other words, they did not realise that in the cases of remittent fever they very often had yellow fever in its less severe types.

Report by Sir Arthur Havelock and of his Medical Board to the then Secretary of State for the Colonies, Earl Derby, upon the 1884 *Epidemic of Yellow Fever in Freetown*

This report is most interesting, because it shows that the observers could not differentiate between bilious remittent fever and yellow fever.

"During the month of May last and the early part of June, a form of fever which was described by the acting colonial surgeon as *typho-malarial fever* became prevalent. Europeans, and especially the Europeans who had recently arrived at Sierra Leone, appeared to be more subject than others to the attacks of this disease. It proved fatal in many cases. The malignant symptoms of this fever became more marked from day to day.

"On the 27th of June, the acting colonial surgeon described it as *a pernicious remittent fever on the borderland of yellow fever.* At the same time, one of the private practitioners in Freetown expressed his opinion that the fever in question had already assumed the form of *yellow fever of a mild type;* and the senior military medical officer reported the death from *yellow fever* of a soldier of the Second *West India* Regiment. On 28th June, a European died of a disease described by the acting colonial surgeon as '*black vomit.*' On 2nd July, two deaths of Europeans were attributed by the same officer to *yellow fever.* On 6th July, another European died of yellow fever.

"The disease seems then to have begun to assume a less virulent type. Several cases of yellow fever were reported, but recovery was made in all instances except one, which ended fatally on the 16th instant. On 12th July the acting colonial surgeon informed me that all cases of fever seemed to be becoming amenable to treatment.

"The final opinion with regard to the nature of the disease, which was given on the 17th instant by the acting colonial surgeon on his own behalf, and on that of the other practitioners in Freetown was, that it was a *mild type of yellow fever of a non-contagious nature.*

"A remarkable feature in the course of the progress of this disease is that, as it assumed a more virulent form its prevalence became more and more restricted to persons of European birth, till at the point at which it reached its worst stage and was admitted to be yellow fever, the natives seemed to have complete immunity from its attacks.

"With the exception of the case of a soldier of the regiment in the garrison here, a West Indian negro, the cause of whose death is, as I have already mentioned, stated to have been yellow fever, there has not been a single authenticated case of that disease among the negro population.

"A considerable number of cases of serious illness, resulting in death in the case of four Europeans, having occurred during the latter part of May and the first fortnight of June, within a narrow area in the central and best quarter of Freetown, I appointed a Medical Board, composed of the acting colonial surgeon, the senior military medical officer, and Dr Cole, a

private practitioner, to inquire into the causes of the special unhealthiness of the quarter in question, and of the general prevalence of fever of a dangerous type. I enclose a copy of the report."

Medical Report

The proceedings of a Medical Board ordered by His Excellency Sir Arthur Havelock, K.C.M.G., to assemble for the purpose of Investigating and Reporting upon the causes which have originated the malignant fever now so fatal in Freetown, particularly that part of it known as Westmorland Street, Rawdon Street, and Howe Street, and other localities. The Board having assembled beg to lay the following report before His Excellency the Governor-in-Chief for the information of the Secretary of State for the Colonies.

Definition.—"The character of the fever which has caused such extensive sickness and mortality amongst the natives and Europeans living in Freetown, and more especially in that limited area known as Westmorland, Rawdon, and Howe streets, resembles yellow fever or that form of pernicious remittent fever of a malignant destructive type having as its characteristics yellowness of the skin and conjunctivæ with dark coloured and very offensive alvine evacuations—dark coloured urine containing blood casts and obvious albumin—a quick pulse and a persistently high temperature ranging from 102° to 105° F. Vomiting often persistent and very difficult to control, dark in colour, and containing a large quantity of bile in some cases with distinctly black vomit. Duration of this form of pernicious remittent fever may be said to be from five to seven days, but in malignant cases four or five days.

"On every occasion that typho-malaria, enteric, or pernicious remittent, or yellow fever has appeared in Freetown epidemically, it has nearly always been of sporadic origin, the undoubted product of Freetown itself, as all attempts to trace it to a non-sporadic origin have totally failed, except when brought here by a sailing-vessel once from Rio Janeiro in 1872.

"The prevalence of this severe form of typho-malarial fever or yellow fever now so fatal among the European and native residents, and many still ill with fever in the town, undrained

insanitary areas of the town may be a warning of the approach of its more deadly sister—malignant, remittent, paludal, or yellow fever."

History.—"There may be stated to be four forms of febrile disease usually met with on the west coast of Africa, viz., intermittent fever or ague, remittent fever, enteric or typho-malarial fever, and pernicious malignant or yellow fever.

"Although the characters of these fevers when fully developed have been freely and frequently described, and show a distinctness of type one from the other, yet so numerous are the connecting links which bind them together that much experience and careful investigation is required before absolutely and positively declaring the type to be of the yellow fever character or that modified form of it, viz., typho-malarial fever. This typho-malarial or fæcal-malarial fever has always had seasons of exacerbation: during some years assuming a mild form, at others a most severe, the mortality increasing with the severity of the type.

"The earlier years of the existence of this colony have been marked by seasons of extreme unhealthiness, especially so in 1807, 1809, 1812, 1815, and 1819. In 1823, yellow fever was epidemic, commencing in the earlier part of the year, the so-called 'healthy or dry season,' and running on through the early rains, and ending with the 'heavy rains.'

"In 1825, yellow fever again became epidemic, and out of a known 902 persons attacked with that fever, 263 succumbed.

"In 1829, during the months of April and May, Sierra Leone was again visited, and yellow fever then confined itself principally (as in the present instance May and June 1884) to the lower levels of the town. This epidemic was, however, stated to be most prevalent during the blowing of the westerly winds and the falling of the heavy rain. It is recorded that out of 150 Europeans attacked with this fever, 11 perished.

"In 1837, yellow fever commenced amongst the Europeans in the month of April, but many very suspicious cases of endemic, remittent, or the so-called African fever occurred during the month of January, and 2 cases died having distinct black vomit. In March, yellow fever declared itself amongst the Europeans in Freetown, and the first case occurring

amongst the troops was on 11th May 1837. The disease is distinctly stated to have declined with the maturity of the rains, and gradually decreased with the saturation of the ground and the atmosphere with moisture until it finally ceased by almost imperceptible and indefinable lines merging again with the ordinary endemic remittent, from which nearly all cases recovered. In October the disease broke out, but not in so malignant a form, and finally disappeared in December.

"In 1838, yellow fever appeared in February and ended in March.

"In 1839, a severe form of remittent fever caused the death of 6 officers at Tower Hill. During the months of July, August, and September, every man of the Royal African Corps suffered, and the mortality amongst that corps is stated to have been appalling. There were 7 officers of the Royal Navy and 13 seamen attacked with yellow fever, and all died.

"In 1845, yellow fever got amongst the crews of H.M. squadron at anchor in the Roquelle River. The *Eclair* sailed from here on the 23rd July and 90 of her crew perished from yellow fever. One case died in September from malignant remittent fever.

"In 1847, yellow fever was epidemic in Freetown during June, July, and August, and the 'rainy season' was noted for great heat with little rain, only 38.45 inches falling, followed during rains by days of extreme heat.

"In 1859, there was no rain until April, May, June, which was then recorded as very slight. Yellow fever was epidemic in Freetown, and carried off 160 Europeans during this year.

"In 1865, yellow fever again appeared, and was epidemic in Freetown.

"In 1866, yellow fever was again prevalent. During the first quarter of the year there was no rain. Between the month of April and the 2nd of October 100 Europeans had died of yellow fever.

"In 1872, the fevers appeared to have been of a malignant type during the months commencing May and ending in December. In December there were 9 persons attacked with yellow fever—6 died. The average death-rate for this year has been rated at 250 per 1000."

Mortality.—The following are the names of the fatal cases taken from the report, occurring amongst Europeans and West Indian soldiers who died from pernicious remittent enteric or typho-malarial fever, and yellow fever, residing in Rawdon Street, Westmorland Street, and Howe Street, also in other localities, with a brief history of their illness during the months of May and June:—

"The first case amongst the Europeans was that of Mons. Loire, æt. twenty-four. He was taken ill on 6th May and died on 16th May. His case was one of the typho-malarial type. He had some abdominal tenderness and frequent vomiting of dark coloured slimy matter; flushed face and bright eyes; his temperature never went below 103° F.

"Our colleague, Dr Hart, residing in Westmorland Street, came from the western district on Sunday, 11th May, ill, after an absence of three days. His temperature up to the day he left for England kept constantly at 104° F. He had some vomiting resembling coffee grounds, pain, and diarrhœa. He was delirious at times, and when he left we had but small hopes of his recovery. He died at sea, on board the s.s. *Calabar*, on 29th May 1884, off Madeira. His case was one of the enteric or typho-malarial type. He was a strong man, and this may account for the long time he was ill. From evidence we have since received, the doctor had convulsive fits, twenty-nine in number, after all fever had left him. The purser of the ship died from fever, and was buried the same day. It is reported that he was in chronic ill-health and consumption.

"Mr Walter Maxwell, an agent of Messrs Lothian, Williams, and Company, residing also in Westmorland Street, was taken ill on 10th May, and died on 20th May. He vomited incessantly, but had no diarrhœa. He ultimately had congestion of the brain and paralysis of his vocal organs. He refused all medicines, food, and stimulants. His cry was 'I am going to die.' He was a hard drinker and from the beginning he caved in.

"Mr Donevein, a clerk, living in the same house as Mr Maxwell, residing also in Westmorland Street. His tempera-

ture ranged from 102° to 104·6°. Taken ill on 7th and died on 11th June.

"Mr Lambert was the sixth case. He was a clerk of the Matacond and North Western Trading Company, and I have never seen a man sick who was neglected as this poor fellow. His illness was only three days, and his temperature during the whole time never went below 104° F.

"Mr Moran, a man who was dismissed from the Matacond and North Western Trading Company was admitted into the Colonial Hospital on the afternoon of 17th June, and died on the 18th at 10.40 P.M. This case was one of delirium tremens and fever distinctly of the yellow fever type."

CASE VIII.—Brief extract of the case of Lance-corporal W. Wood, 2nd West India Regiment, æt. twenty-two years, a coloured man admitted into the Station Hospital, Sierra Leone, on 27th May, suffering from enteric fever or typho-malarial, and who died on 25th June 1884. On admission he had all the symptoms of ordinary African remittent fever, complained of pains in the back, head, and limbs, with a temperature of 102 to 103° F. For several days his temperature seldom fell lower than 101° F. and rose to 105·4° on the evening of the fourth day in hospital and the fifth of his illness. He now appeared gradually to improve, and the temperature shortly fell to normal. On the evening of 5th June, he had a sudden rise of temperature to 102°. The tongue was very red at the tip and edges, and he complained of gripping pains in his bowels. The eyes were bright, the face flushed and anxious looking, and he stated his head ached badly, and his condition was such that enteric fever was now diagnosed. He was watched with great care and everything done for him, and for a few days he appeared to improve. On 14th June he had a relapse, and the temperature rose in the evening to 105·2°, and remained in that condition during the night, until the early morning visit, when it was again found to be 105·2°, but fell during the morning to 102·4.° His condition now became serious. On the morning the temperature was 102·4° and fell again in the evening to 101·4°. His sleep during the night was much broken and disturbed. He became very restless, wandering in his mind, and attempted frequently to

get out of bed. His bowels were twice freely moved, the evacuations being very offensive, and the urine was voided in small quantity and contained traces of albumin. On the 25th he showed distinct symptoms of failure of the heart's action. He had twitchings of the fingers, and picked the bed-clothes; there was muttering delirium, and he became quite unconscious after a short attack of convulsions, with squinting, and twitching of fingers and lips. He expired on 20th June. The post-mortem showed disorganisation of the liver, kidneys, heart, and spleen, with an ulcerated condition of the bowels, enlargement of the mesenteric glands, and deep red well-defined ulcers of the Peyers patches and the solitary glands. The colon was not ulcerated. Cause of death: remittent fever, enteric or typho-malarial fever.

Typical case of yellow fever in a native of the West Indies. *A black man.*

CASE IX.—Report on the case of R. Codrington, No. 2544, Private 2nd West India Regiment. Died of yellow fever on the 25th June 1884 at Sierra Leone.

"This man reported sick at the Station Hospital on the 23rd June 1884, and stated that he was suffering from 'fever.' His temperature was 102° and his bowels were confined. He was ordered Sulph. Quinine gr. xv, and a diaphoretic, also a purgative of Calomel gr. v, Pulv. Jalap, Co. gr. xxx, and detained for the night. He stated on this occasion that he had suffered from 'fever' every evening for some days previous to his reporting sick.

"On the morning of the 24th was seen at 7.30 A.M. He complained of feeling very weak. His temperature was 100°. His tongue was covered with a white fur, and was red at the tip and edges. His conjunctivæ were tinged with yellow. He also suffered from frontal or orbital pain and flying pains over the abdomen. During the night, about 12 P.M., his bowels had been well moved, he stated. When I first saw him he said he had a great pain in his head. I prescribed a poultice to loins, together with hot fomentation to abdomen, and suitable diet. Next saw him at 10 A.M. the same day, and on going to his bedside I noticed in the chamber pot by his bed some black

fluid which he said he had just vomited. I now drew the attention of P.M.O. Surgeon J. J. Lamprey, A.M.D., to the case, as I suspected *yellow fever*, and he agreed with me. It was a most suspicious case. He had passed some urine before I came into the ward, but unfortunately it had been thrown away. I now ordered his urine to be carefully kept until I saw him later on in the day. I gave him a diaphoretic and a purgative of sulphate of soda, in lukewarm water. I also ordered the fomentation to be continued. At 3 P.M. I again visited him, and noted that he was suffering from a peculiar nervous twitching of the fingers, and that the conjunctivæ were of an intensely yellow colour, and the eyes looked very brilliant. I found he had not passed any water since my last visit, so I passed a No. 9 gum elastic catheter, and drew off about 10 oz. of high-coloured offensive urine.

"On examination of this fluid it gave an acid reaction, and on testing the specific gravity, it only registered ·1014. On the addition of pure nitric acid I got a most distinct ring of albumin, and on heat and nitric acid being applied, the urine became almost solid. His bowels had not been moved. I continued the fomentations, and also ordered him an enema to which was added ol. Terebinthinæ, this treatment having been recommended by Professor Aitken and all other authorities on the subject. I gave him also during the day weak brandy and water with dram doses of ol. Terebinth. He appeared to be better at 7 P.M. from this treatment and inclined to sleep. He subsequently fell asleep at 5 A.M. on the 25th day. He suddenly awoke and jumped out of bed. He was got into bed by the orderly in charge, and then became comatose. I saw him at 7 when he was in this condition; breathing was stertorous and heart's action very feeble. He could not be got to swallow anything. At 8.30 he had another black vomit, and almost immediately breathed his last.

"In conclusion I may mention that this man was in the habit of visiting parts of the town which I believe is called *Soldiers Town*, and where the most insanitary state of things exist. In my humble opinion (and that of my brother medical officers in the garrison also) I may state that I am convinced that it was a true case of yellow fever, and the appearance of the eyes, the

pain in frontal and orbital regions, coupled with the intense pain in the region of the kidneys, the presence of albumin in the urine, and the pain over the liver, all tend to bear out my diagnosis.

"I may mention that at 7.30 A.M. on the 25th (to-day) there was suppression of urine, and the bowels had not acted after the enema given on the evening of the 24th, that is to say, the fluid injected came away unchanged."

A. HICKMAN MORGAN,
Surgeon, A.M.D.

Certified true copy,

J. J. LAMPREY, A.M.D.,
Senior Medical Officer,
West Africa.

CASE X.—A brief extract of the case of Sister Theonite (Barbe Tuntag), æt. thirty years, a resident nun in the convent and school-house in the Roman Catholic Mission in Howe Street, where she contracted fever early in the month of May.

"Some bright rose-coloured spots appeared on her skin, and she complained of pain in the head, back, and limbs. There was very little fever, and she attended to her duties. On 18th June she went to bed and complained of great pain in the region of the stomach. She did not improve and her condition became seriously worse. She discharged from the bowels on 26th June, a large quantity of blood. She suffered very great pain all over the abdomen. She became delirious and died on Friday morning, 27th June 1884.

"The death of Barbe Tuntag was due to enteric fever, or that form of it complicated with malaria known as typho-malarial fever.

"The sanitary surroundings at Howe Street and in the Roman Catholic school-house and convent are highly defective, and the system of sewage disposal is very bad.

"The other members of this mission have suffered during the present season from repeated attacks of remittent fever, and two are at present suffering from a low form of malarial fever, contracted in these infected localities."

CASE XI.—Mrs Hagar Palmer, aged twenty years, a native of Sierra Leone, residing at Lumley Street, died on 25th June of yellow fever as reported by Dr Davies.

CASE XII.—Mr Charles James Ryder, aged nineteen years, a European clerk at Messrs Pickering & Berthoud, residing at Howe Street, died on 28th June of yellow fever.

"The extreme sick rate and mortality from fever amongst the 'native population,' occurring during the months of May and June in Freetown, and falling upon a great number of the inhabitants and attacking many persons at the same time with fever and pain in the bowels, bleeding from the gums and vomiting, in some cases black in colour, has been a disease taking the form of the so-called 'Sierra Leone fever,' and more severe in type than has been experienced for some years in this city.

"The death-rate since January has been estimated from the 'burial records' at about 35 *per* 1000, *or* 4 *per cent. of the entire population.* The registrar-general's return shows for May and June alone no less than 19 deaths amongst the resident natives; and this is the more remarkable since very few of these have been authenticated by 'medical certificate,' and the people have been left to make a report themselves as to the 'cause of death,' according to their own judgment, thereby proving the endemic of a fever by exposing a very obvious knowledge of its more fatal symptoms."

3. *Account of the* 1900 *Outbreak of Yellow Fever at Bathurst by Dr Chichester*

Dr Chichester, who attended the cases in the outbreak, furnished the following notes and most interesting comments. His comments are all the more interesting for he was probably unaware of the Cuban experiments which had only just been announced. He states:—

"Perhaps, and not improbably, in this case mosquitos were the agents responsible for the yellow fever spreading from its

original centre. It is worthy of note that all the cases (with one exception) occurred on the front street of the town, the street most infested with mosquitos, and *in people who slept without mosquito nets*, or with nets in a bad state of repair.

"The one exception referred to occurred at the Catholic Mission, and he slept without any net at all. The government officials who live in the front street escaped, but they are careful about their nets. All the European houses but three were attacked. One was at too great a distance for the flight of a mosquito, all the Europeans belonging to a second went away at the first outbreak, and the third which was close to the house primarily attacked, escaped; and I don't know why, as everything was favourable to an outbreak.

"The history of the epidemic is as follows :—On 18th May official news arrived stating that yellow fever existed in Dakar. The date of its first appearance there I do not know. The French ports north of the Gambia were at once quarantined, and later on those south of the Gambia were also placed in quarantine, when it became known that St Louis, Casamance, and Carabane had become infected.

"*Case No.* 1.—A Syrian came before me on 23rd May. The patient was in much pain, feverish, and had a headache. I admitted him into the hospital under the impression that he was suffering from influenza then prevalent in the town. However, next morning not liking the appearance of the case—he was very restless, semi-unconscious, had yellow tinging of conjunctivæ, his urine was albuminous—I had him transported to some huts erected for native small-pox patients. He died that evening, having vomited just before death 'coffee ground' looking matter. I took all precautions as if it had been a case of yellow fever, and reported it as death from fever 'of a doubtful nature.'

"*Case No.* 2 was an inmate of the same house in which the first case had occurred. He was attended by a native qualified practitioner, who certified that he died of remittent fever. The practitioner in question informed me that it was an undoubted case of malaria, but I am inclined to think that it must have been a case of yellow fever. His death occurred on 7th June, and he had been some five days ill.

"*Case No.* 3 also came from the same house. He was attended by the doctor above mentioned, and I only heard of his being ill on 9th June. I asked permission of the doctor to see him, but I could make out little that was definite at the time. Beyond headache and fever there was little to go on. No definite previous history could be obtained, and the urine had not been examined, but I took a slide of blood and found no malarial parasites. The next day I was called in consultation, and by this time the case had begun to look very suspicious. He was in a semi-comatose condition, the conjunctivæ were slightly tinged, the urine was nearly suppressed, and the small quantity drawn off was highly albuminous; liver and spleen were not enlarged; tongue was covered with a white fur and red round the edges. This looked to me very like yellow fever, and the question in my mind was, Was I sufficiently sure to have the place put in quarantine?

"The patient died that night, and a post-mortem next morning removed all doubt from my mind. I declared it a case of yellow fever and communicated the fact.

"On that and the following days I received instructions to have the house closed and watched, and to have those not attacked placed in quarantine on the other side of the river, and to have any other Syrians who might become attacked sent to the small-pox huts.

"*Case No.* 4, also from the house in Russell Street, died in the small-pox hut on 12th June, having been removed thereto that morning.

"*Case No.* 5.—This was a young French clerk living some 150 yards from the house in Russell Street. I was called to see him on 15th June, and was informed that he had been ill for some days. It was an undoubted case of yellow fever and I at once had the house and business closed. He died on the 16th of hæmorrhage, due to the rupture of a blood vessel in the stomach walls.

"*Case No.* 6.—This was an Englishman about forty-five years of age, who had been many years on the Coast. He had been in very bad health for some time previous but only called me in when he found that he was becoming much worse. He had certainly had yellow fever when I saw him for some four days.

Under treatment and careful diet he improved a little, but died of heart failure on the 19th.

"*Case No.* 7.—This case occurred on 16th June at the Telegraph Station. It was a mild case and ended in recovery.

"*Case No.* 8.—On 28th June I was called in to see a lay brother at the Catholic Mission who had been taken suddenly ill the previous day. It was another case of yellow fever, and the attack was exceedingly severe. He died within thirty-six hours from the time of onset.

"*Case* 9.—A long time elapsed between this case and the last case. He was a clerk in one of the companies, and his work lay behind the cash counter. I could not trace how he contracted the disease. He was taken to the old Military Hospital on 4th August and died on the 6th.

"This was the last case.

"The mortality was heavy—8 cases out of 9 died.

"That the disease did not spread further is without doubt due to the action of the local authority, and the European merchants in sending away from Bathurst all the Europeans that could be spared. More than half of the European population left in the early part of the epidemic.

"*Case* 10.—A clerk in the employ of one of the European houses, arrived in Bathurst on 11th October. He came from Mandina Bar, a town on the banks of the river some thirty miles distant, through which passes much traffic from Casamance, Carraban, and the surrounding country. Casamance and Carraban are towns in French territory, in which yellow fever has appeared, and which are no doubt still infected.

"On 13th October he was taken suddenly ill with fever, very severe pains in head, loins, and limbs, vomiting though not severe. The headache pains lasted all the next day and well on into the third day, when I was called in. The vomiting had by that time ceased for some twenty-four hours. When I arrived the pain had much decreased. His temperature was 103·4°; face a little flushed; eyes watery, and rapid pulse. His bowels not having been opened for some days I gave a purge which acted well, and quinine 6 g. hypodermically. His tongue was clean. No tinging of skin or conjunctivæ. Next morning I found about 12 per cent. albumin in his urine, and day by day

the percentage increased, until on the sixth day it amounted to over 60 per cent. I could not have the urine kept to measure, but the patient did not think the amount passed had much diminished. The blood was examined for plasmodia, but, beyond a few found at the first examination, I could find none. Quinine seems to have caused their disappearance, but it had no effect on the temperature which, from the day I first saw him up to the day of his death, ranged between 102·4° and 103·8°, except when on the sixth day it went up to 104°. On each day he was given 6 g. of quinine hypodermically, with the addition on the sixth and seventh day of strychnine and digitalis as his pulse was getting weaker.

"On the fifth day the tongue began to be coated on the surface, keeping clean and red looking round the edges; the eyes were more suffused and beginning to assume a yellow tint.

"On the sixth day the skin began to assume a yellowish tint and the patient himself noticed it, but it did not become marked. The *pulse rate was at no time in proportion to the temperature, and on the fourth day began to lessen to* 84, 76, *and* 72. On the fifth day it again increased in frequency while lessening in force. The temperature all this time remained pretty constant.

"On the seventh day he began to get weaker, and this increased despite the free use of stimulants, and he died on the evening of the eighth day of his illness.

"Delirium was not present till the last twenty-four hours. After the cessation of the initial pains *he constantly expressed himself as feeling quite well.* I have noticed this in nearly all the cases of yellow fever I have attended.

"*A post-mortem examination* was made, but I had to content myself with the examination of the stomach, intestines, liver, and spleen, owing to the difficulties in my way.

"*Stomach.*—The coats were softened and swollen, blood vessels prominent, and some mucus of a chocolate colour was present. No coffee-ground matter was found.

"*Small intestines.*—Pretty much the same can be said of them as I have said of the stomach.

"*Spleen.*—Was not enlarged.

"*Liver.*—Slightly enlarged, hyperæmic, and the spirit in which a small piece has been preserved soon assumed a greenish colour.

"Post-mortem staining of the body was very marked, not only in dependent parts but over the neck and chest, front of lower part of abdomen, and all over the limbs.

"*Case* 11, *Mons. Brunel.*—Another case is at present under treatment, a European belonging to the same firm. It is up to the present a mild case; but I have little doubt that it also is a case of yellow fever, and that it was contracted at Mandina Bar whither he had gone to take an inventory of the stock after the death of the man, of whose case I have just given the history."

Dr Chichester describes 11 cases and 9 deaths. In every probability there were more cases which passed unobserved, for a death-rate of nine in eleven is abnormal; moreover, at this time there was influenza, and the evidence shows that the senior medical officer regarded the cases as "malignant remittent fever"; and another officer made the diagnosis of "malaria." It is clear, therefore, that in this outbreak, as in previous ones, a considerable amount of divergence of opinion took place. The symptomatology given by Dr Chichester is unmistakably that of yellow fever. Faget's sign was present, also albuminuria, black vomit, hæmorrhages, jaundice; and quinine was without effect. In this outbreak, as in innumerable others, segregation of the non-immunes was the specific prophylactic measure. Dr Chichester considered Senegal as the source of infection. But as on very many previous occasions Senegal considered Bathurst as the source of infection, it is very probable that yellow fever was endemic to both.

CHAPTER X

THE SYMPTOMS OF SOME OF THE CASES OF YELLOW FEVER WHICH HAVE APPEARED IN WEST AFRICA IN RECENT YEARS

Saltpond 1895

1. *Gold Coast*—

22*nd March.*—Patient taken unwell; temperature 103°; pulse 100; headache; vomiting; symptoms increased.

23*rd March.*—Vomiting increased; suppression of urine (two days).

24*th March.*—Temperature 99·8°; pulse 80; vomiting and hiccough.

25*th March.*—Temperature 103°; jaundice appeared; conjunctivæ yellow; delirium; vomit consists of brown material.

26*th March.*—Temperature 105·8°; pulse 60; marked yellow skin.

28*th March.*—Vomited brown material; also a cupful of blood; then coma and death; deep yellow colour of body.

Notes signed by Dr Garland.

The diagnosis made in this case is "remittent fever." It is very obvious, however, that it is a most typical severe form of yellow fever. The notes show rapid onset, one paroxysm of fever lasting five days, and terminating fatally; marked Faget's sign; suppression of urine and jaundice developing towards the end; no remission. This can be no other disease than yellow fever.

The first case of diagnosis of yellow fever in 1897:—

Mr A., æt. twenty-two. Disease, yellow fever. Result, death. Date of admission, 15th May 1897.

Patient, a healthy young man, had arrived from England about two months ago; was taken suddenly ill on the morning of the 15th, about 7 A.M.

15*th May.*—Onset; had been feeling out of sorts during the afternoon, but had played a game of tennis about 5 P.M.

Symptoms.—Pyrexia (about 1 to 3 P.M.) headache; pain (not severe) in limbs and nausea; vomited.

16*th May.*—9.30 A.M., had little or no sleep during night. Frequent vomiting; temperature 102·4° (pulse not counted). 7.30 P.M., temperature 103·4°; pulse 104.

18*th May.*—Temperature 103·6°; pulse 84, soft and weak.

19*th May.*—9.30 A.M., temperature 104°; pulse 82, and fairly strong. At 2.30 P.M. again slight perspiration. During the day he passed a fair quantity of bilious urine and two or three blackish liquid motions.

20*th May.*—3.15 A.M., temperature 103°; had not passed urine for nine hours, 6.45 A.M.; 9 A.M., temperature 103.4°; pulse 105, weak and unsteady; slight yellow tint in skin; black vomit. Quinine has proved absolutely useless. 11 A.M., black motion; 11.20 A.M., after twenty minutes much discomfort, thirst and wandering ideas; patient became gradually weak and ceased to take anything; black vomit occurred more than once; no further passage of urine; hiccough set in.

Remarks.—At the time of writing these notes (25th May 1897) I am informed of two and three similar cases as having occurred recently at Cape Coast. The case is similar to the epidemic which prevailed at Elmina and Cape Coast in the early year of 1895. That was during the height of the hot dry season. The present meteorological conditions are the reverse. We are well into the rains. I stated in my report on a similar case in Elmina that in my opinion the disease is yellow fever. I adhere to that opinion. W. M. E.

Here also is a very typical case of yellow fever; sudden onset; high temperature; one paroxysm; Faget's sign; quinine no effect; suppression of urine; black vomit.

Another case—diagnosis yellow fever, 1897.

17*th June.*—The doctor reported temperature 105°; tongue

coated; breath very foul; pains in stomach; temperature in the evening 103°; pulse 104.

18*th June.*—Icteric tint appeared; temperature 102·6°; pulse 98; later icteric tint deepened; vomits distinctly black; motions also black; suppression of urine.

19*th June.*—Symptoms as before; death.

Elmina 1895

CASE OF J. S.—Diagnosis, "hepatic fever."

3*rd March.*—Great prostration; vomiting; vomit very black; coffee grounds; eyes yellow; skin primrose yellow; suppression of urine; death.

CASE OF E. C. C.

18*th April.*—Temperature 102·4°; pulse 100; intense headache; backache; nausea; epigastric pain.

19*th April.*—Temperature 103·4°; pulse 92.

22*nd April.*—Black vomit; death.

Post-mortem examination showed liver extremely congested; kidneys congested; stomach containing black vomit; skin yellow.

The house physician, Dr W. M. G., states that this is a suspicious case of yellow fever; the same as Mr S. He seems to have had little doubt that these were cases of yellow fever.

Cape Coast 1902

CASE OF A. N., æt. twenty-eight. Diagnosis, yellow fever.

1*st July.*—Temperature 100·4°; pulse 100; skin and conjunctivæ yellow; albuminuria; drowsy, anorexia; no malarial parasites found in the blood.

2*nd July.*—Urine bile stained; but patient feels better.

3*rd July.*—Feeling very much better; took solid food; this was followed by black vomit; skin became bright yellow; delirious, then coma.

4*th July.*—Vomited black; and later pure blood, and died.

Post-mortem.—Skin bright yellow; liver yellow; stomach and intestines deeply congested.

G. L. BARKER.

CASE OF Mr V.—Diagnosis, yellow fever.

1st July.—Temperature 102·2°; pulse 76; later in the day temperature 103·8°; pulse 78; later 82; conjunctivæ commencing to turn yellow; urine albuminous; at the end of day passed a tarry stool.

2nd July.—Vomiting set in; bilious matter: delirious.

3rd July.—Vomit black; followed by blood; very persistent; passed a tarry motion; coma, then death.

Post-mortem Notes.—Skin yellow, liver yellow, and mottled with red; kidneys congested, stomach congested, and containing black fluid similar to vomit; intestines ditto.

G. L. BARKER.

CASE OF A. S., æt. twenty-five. — Diagnosis, yellow fever, March 1903.

A. S., European clerk, æt. twenty-five, living in business house in native quarter; ten months resident in Cape Coast; seen 23rd March 1903; temperature 102·4°; pulse quick; face flushed; urine dark colour; tongue furred.

24th March.—Bad night, pain varies; temperature 103·4°.

25th March.—Temperature 101·5°; pronounced catarrh of stomach; intestine discomfort; Dr Rome Hall considered that the case was one of malarial remittent plus acute gastric enteritis, in fact typho-malarial remittent fever. On same day commenced to vomit yellowish-green mucus; much depressed.

26th March.—Vomiting ceased; temperature 101·1°; pulse 60; with temperature 100·5°; mental depression.

28th March.—Temperature 100·8°; tongue coated with dark brown colour; sordes very marked; eyes much more icteric; breath very foul; pulse depresses as before; could not see any jaundice; removed to hospital.

29th March.—Temperature at 6 A.M. 99·4°; pulse 70. The pulse for the next forty-eight hours remains slow, and did not increase with the rising temperature; eyes very yellow; slight general jaundice; vomited once.

30th March.—Temperature 102·2°; urine alb.; vomit bilious; temperature 100·5°; pulse 87.

31st March.—Jaundice increased; patient apathetic. "*I now question diagnosis.*"

1st April.—Jaundice increased; suppression of urine.

2nd April.—Jaundice much better; temperature 102·7°; pulse 96; vomited brownish material; in evening blood-stained material.

4th April.—Urine passed, considerable quantity of alb.; head brighter; patient seemed improving.

5th April.—General primrose-yellow colour of body.

6th April.—Purging; temperature 102°; pulse 128.

7th April.—Death, bringing up at the last some black vomit.

Dr Rome Hall at first considered that he had a case of ordinary malaria fever with typhoid symptoms. The disease was not blackwater fever. "I noted that . . .? was much greater than an ordinary remittent malaria. The pain and uneasiness in epigastrium was not marked. The offensive odour was most marked in this case. The presence of albumin in urine made me question my diagnosis. The vomiting set in late. In bilious remittent, black vomit is not known. The P.M. signs seem to me universally in favour of *yellow fever.*

"*Yellow fever* has several times in recent years been recognised on the French Ivory Coast and in Senegambia. The Ivory Coast is only 150 miles or forty-eight hours away from Cape Coast by the coast and steamers, and at the present moment, 14th April 1903, we are quarantined as regards the Ivory Coast against yellow fever. The first steamer with the quarantine flag came into Cape Coast from the Ivory Coast in the last week in January, and since that date there has been landing and other communications. The *Stegomyia* is universal in the merchants' tanks and in the water tanks of the natives. Old writers believed that slave ships carried yellow fever from the Guinea Gulf. Fantees describe malaria bilious remittent fever amongst themselves as Kondruku. In the disease Kondruku Fufu, the patient usually dies."

G. Rome Hall, S.M.O.

Note.—I have received the following letter from Dr Savage in confirmation of the cases of yellow fever in Cape Coast:—

Cape Coast,
20*th June* 1910.

"I have the honour to forward, as requested in your telegram of yesterday's date, the Colonial Hospital Case Book containing

records of European cases during the years 1902 and 1903. I have been in private practice in this town since January 1902 and I remember well the deaths of the European Bank Clerks recorded in the Case Book.

"These deaths caused considerable stir at the time at Cape Coast. Dr Barker, I observe, recorded in the case-book two of these cases (Bertian, 25. 6. 02. & Brown, 29. 7. 02) as Remittent Fever and Pernicious Fever (?) suspecting the case of Veners (4. 7. 02.) alone as Yellow Fever.

"Dr Barker I knew was strongly of opinion that the three cases were Yellow Fever cases for I had several conversations with him at the time on the cases.

"Dr Rome Hall's case of Alfred Smith I remember very well also. Dr Rome Hall had not the least doubt that the case was one of Yellow Fever and he was kind enough to send me at the time a copy of the clinical record of the case.

"I may remark here that at the time that Dr Barker was attending the cases at the Bank I was in attendance on Mr B., a European employee of Messrs The African Association for Bilious Remittent Fever. The local residence of this firm was then next door to the Bank on the Salt Pond Road. I kept no clinical record of the case, but I remember I had great difficulty at the time in differentiating between Yellow Fever and Bilious Remittent Fever as the diagnosis. Mr B. recovered and was invalided, and the last time I heard of him he was somewhere in the United States of America.

"I had not the least doubt that Dr Barker's cases at the Bank were cases of Yellow Fever. Some time after Mr B.'s recovery I urged upon Messrs The African Association, for whom I was then working under a yearly retainer, to remove their quarters, which in many respects were insanitary, to a better locality, which was done about a year later, but not until two other European employees of theirs, Mr T. B. and Mr John A. D., had died, one on the *21st March* 1903 and the other, a near arrival, on *2nd May* 1903. These two cases I diagnosed as Pernicious Malarial Remittent with cerebral symptoms and Non-Malarial Remittent Fever respectively, and I may state here that the duration of the illness was above 5 or 6 days.

"On 15th October 1903, the firm of Messrs The African

Association in the new premises to which they had removed lost another European employee, R. G. B. by name, also a new arrival. This case was undoubtedly one of Yellow Fever. There was the characteristic black vomit of Yellow Fever about the 3rd or 4th day of the disease and most abundant just before the patient died: the conjunctivæ and skin were yellow about the 3rd day of the disease, the urine was loaded with albumin: the pulse was full and bounding for the first two or three days of the disease and markedly soft and slow about the 4th and 5th day when the end came.

"I remember sending the body to the Colonial Hospital Mortuary for post-mortem examination and the appearances were characteristic of Yellow Fever.

"Since Mr B.'s death Messrs The African Association, I believe, have not lost locally a European employee, nor as far as I know has the Bank, who remain in the same quarters that they were in in 1902.

"I did not keep a full clinical record of any cases but I was so much concerned at the time by the loss of so many Europeans and of so many patients of mine that the cases were stamped in my memory and my recollection of them is vivid."

Dr Elliott, who saw many of the cases of yellow fever at Saltpond on the Gold Coast, lays stress upon the late development of icterus, the black vomit, and the tarry stools, and the uselessness of quinine in helping to distinguish yellow fever from the malarial group. The cases which he published in the *Journal of Tropical Medicine* are typical of yellow fever.

Dr S. O. Browne also published a case of yellow fever which he observed at Saltpond. The history shows sudden onset, a fever of one paroxysm, suppression of urine, intense black vomit, lividity and yellow colour of the skin after death.

2. *Symptomatology of the Freetown Cases of Yellow Fever*, 1910. (*Notes furnished by Dr Kennan*)

CASE I.—*2nd May.*—A Syrian, æt. twenty-three, stated to have been taken ill with fever and bleeding from the nose.

4th May.—Admitted into Colonial Hospital at 9.45 A.M.,

with temperature 102·6°. Said to have had fever and slight hæmorrhage from nose on 2nd inst. On the 4th it came on with increased violence, and he was brought to hospital. He was in a dazed condition, his skin a dirty yellow in colour, and he was very restless. The bleeding stopped towards evening.

5th May.—Had copious black vomiting, and a stool of the same colour. The vomiting continued, and just before death at 2.30 P.M. on the same day (5th), he bled profusely from the mouth, nose, and bowel. His blood was examined, and contained no parasites and no mono-nuclear leucocytes. No post-mortem examination held, as relatives refused permission.

CASE II.—*6th May.*—A Syrian male, aged fifteen years admitted to Colonial Hospital at 6 P.M. His temperature on admission was 104°, skin pronouncedly yellow, and scleræ the same. Eyes injected and bright, patient restless and delirious. Bowels were apparently empty as enemata had no effect. No previous history obtainable.

7th May.—Patient passed a restless night, and his temperature in the morning was 103·8°. No malarial parasites were found. He had "black vomit," and died at 11 A.M. on the 7th, Post-mortem refused by friends. (Both cases were under the care of Dr Burrows, M.O. Comment.—It is unfortunate that in both these cases there is no precise information as to the date of onset of the illness; in the first case it might have been on 2nd May. In the second case I am of opinion that the onset of the attack was at least three days prior to 6th May, that is he was probably taken ill on 2nd May, so that when admitted into hospital he was in his sixth day of illness. I have no hesitation in concluding that both were cases of severe yellow fever.

CASE III.—Mr H. H. T., Englishman, forty-one years, living in Garrison Street.

9th May.—Had a "touch" of fever with headache.

10th May.—Had a rigor temperature 104°, and violent headache. These symptoms continued with more or less severity.

11th May.—Examined by doctor; temperature 104·8° at 5 P.M.; headache very severe; loin pain was much complained of; face flushed and skin of a yellowish red almost brawny colour.

12*th May.*—Patient removed to the Nursing Home. His blood was examined independently first by Dr Kennan at about 6 P.M. and by Dr Burrows twelve hours afterwards, and no parasites (malarial) were found. He had had little or no Quinine before the blood examination. Urine highly albuminous, with trace of bile, acid and scanty. Temperature continued high sinking slowly from 104° on admission.

13*th May.*—Temperature 102·4°; in the evening, passed a fair night, but symptoms of collapse were noticed.

14*th May.*—Temperature 99° at 7 A.M.; condition was grave, respirations 54 per minute, but pulse was slow and fairly full. The lungs became congested, and there was rattling respiration which continued all day. Temperature reached 103° in the rectum, and the patient's condition became worse. He had "black vomit" (small) at 6 P.M., and at 8.15 P.M. there was a profuse discharge of "tarry" liquid from the mouth and nose, and the patient died.

Note.—The tongue was coated with a greyish fur and breath was very foul; the tongue was clean at the edges. The aspect of the patient on admission to the Home was the subject of spontaneous remark by the nursing sister, who very aptly described the eyes as peculiarly "bright and sharp looking."

Post-mortem examination made by Dr Burrows, in presence of Drs Kennan and Renner, at 7 A.M. on 15th May.—Brain (weight, 3 lb. 4 oz.), not unduly congested, but marked œdema of the pia-arachnoid over the frontal region. Heart (weight 13 oz.) fatty, and muscle substance friable and "dry." Valves, yellowish colour. Lungs (right, 20 oz.); (left, 17 oz.), congested posteriorly. Spleen (18 oz.), soft, friable, almost diffluent. Liver (72 oz.), enlarged, sharp edges and "fatty" (dull yellow). Right kidney, $6\frac{3}{4}$ oz.; left, 5 oz., both markedly congested. Stomach contained dark brown mucous fluid, extremely well marked injection of arborescent vessels in wall. Intestines contained fluid similar to that in stomach.

(Mr Taylor, who was an "old coaster," had arrived in the Colony from his last leave in October previous.)

CASE IV.—Case of G. R., reported by Dr Burrows. Syrian male, aged about twenty-five years, living at 26 Kissey Street.

18*th May*.—Complained of lassitude, yawning, and stretching.

19*th May*.—Tired feeling exaggerated.

20*th May*.—Bowels moved freely; no malarial parasites found, but patient had taken quinine regularly; temperature 101°; no headache; no vomiting; malaise and great thirst, with a griping pain in stomach.

21*st May*.—Temperature 102°; at 8 A.M., bowels acted; temperature 103·6° in evening; feels better.

22*nd May*.—Temperature 103 in the morning; tongue very foul and furred; temperature in the evening 103°; patient very distressed and uneasy; epigastric tenderness; had sweated a little in afternoon; complains of sleeplessness.

23*rd May*.—Temperature 103° in the morning; felt much better; conjunctivæ slightly jaundiced; looked rather tired and worn out; temperature 104° in the evening; pain and uneasiness increased; two natural loose motions passed; removed to hospital; patient was given nourishment, which he took at short intervals fairly well; he was restless up to 10 P.M., and then slept well for the rest of the night.

24*th May*.—Temperature 102·6° in the morning; strained a good deal to pass urine—about 6 oz. passed; and bladder being distended, it was emptied by catheter and 1 pint of dark urine (acid and one-tenth albumin) was drawn off; patient was drowsy and distinctly jaundiced; his face pinched and "worried looking"; he had alternate phases of quiet and restlessness during the forenoon, and began to become comatose shortly after 2 o'clock; pulse was full 104, but became slower as day wore on; urine 1 pint drawn off by catheter; temperature at 7 P.M., 101° in axilla, but skin felt cool and clammy; patient was restless all night, became delirious, and finally sank into a condition of "coma vigil."

25*th May*.—Temperature rose rapidly to 105·4° between 5 and 6 A.M., and patient became quiet, and died at 6.30 A.M.

Post-mortem examination, 25th May 1910, 9.30 A.M.

Yellowish skin and scleræ, mottly, bluish, spotty, rash, chiefly limbs and scrotum; liver yellow, "box-wood," exsanguine; spleen soft, not enlarged; kidneys slightly congested, large; stomach contained large quantity black fluid, congested; small intestines contained a quantity of fluid resembling that in

stomach; large intestines contained grey, small fæces; bladder contained about 1 pint of urine; lungs healthy; pericardium some excess straw-coloured fluid; sub-pericardial hæmorrhage in left interventricular groove; heart normal, valves yellowish, and yellowish serum clot in chambers.

Weight of organs.—Heart, 7 oz.; right lung, 16 oz.; left lung, 17 oz.; right kidney, 5 oz.; left kidney, 6½ oz.; liver, 40 oz.; spleen, 5 oz.

Note.—Mr Rebiez had arrived in Freetown from Dakkar between two and three weeks before the onset of illness.

CASE V.—Reported by Dr Burrows.

26th May.—An old coaster who had returned from England early in the year 1910 had two short and distinct rigors, soon followed by severe headache. He at once took 10 g. quinine, and followed them with 25 g. more within twenty-four hours; temperature that night 103° F.

27th May.—Temperature ranged from 99° to 100° all day; headache severe and continuous.

28th May.—Passed four copious dark green stools in morning, as result of purge; temperature 101°. Was seen by doctor for the first time same afternoon; temperature still 101°; tongue coated, fissured, and very foul; blood examined showed no parasites and no mono-nuclear leucocytosis; no albumin in the urine; temperature 104° at midnight, and cardiac distress slight.

29th May.—About 2 A.M. was very restless and agitated; bowels moved twice—"pea-soupy" and foul; temperature 101°; blood re-examined, no malarial parasites; urine contained albumin; tongue dry; covered with brownish fur; temperature 103° at noon, and at 5 P.M. 103°; removed to nursing home.

30th May.—Temperature 102·6°; restless; urine one-sixth albumin; stools copious; grey liquid and excessively foul; temperature 102° at 6 P.M; breathing became very laboured; pulse 94 to 100 per minute; was freely stimulated during night.

31st May.—Temperature fell to 99·4° at 2 A.M.; pulse running about 100 per minute; temperature dropped to normal at 3 A.M., and contained so for rest of day; urine highly albuminous; took all nourishment and stimulants without

trouble. At noon cardiac distress increased; urine suppressed; patient more or less comatose; twitching of muscles of face and neck. At 3.50 A.M. patient had "black vomit," and died. Seen immediately after death, the face was a bright yellow, but this speedily disappeared; bluish discolorations about neck, arms, penis, and scrotum.

Post-mortem was made at about 5 P.M., limited to examination of the stomach and liver. The former contained a fairly large quantity of almost black fluid, the stomach wall was congested, the mucous membrane was swollen and patchily injected. The liver was greatly congested, with no "box-wood" appearance about it. The small intestines contained similar material to that in the stomach. The conjunctivæ were slightly yellow, but yellowness in the skin could scarcely be said to be present.

CASE V.—Report on post-mortem on body of European named C. H., Captain, Mercantile Marine, 9th June 1910, by Captain Webb, R.A.M.C.

The body is that of a well-nourished man. Had been in the Colony one and a half years. There is marked general yellow discoloration of the skin and also of the conjunctivæ; patches of ecchymosis on the skin of the legs, and darker coloration of the scrotum and dependent parts; an incision through the abdominal parieties shows the abdominal fat stained deep yellow.

The liver was retracted slightly under the costal edge, and showed a yellow mottling through the peritoneal covering. This latter stripped off extremely easily, and the liver substance itself was very soft, breaking down on very slight pressure. The whole of the liver showed fatty degeneration and a box-wood appearance.

The stomach showed ecchymosis under the peritoneal surface, and contained a dark and markedly bloody fluid about 3 oz. The mucous lining was deeply congested, and numerous petechial points were present over the whole of the surface. The small intestines were distended and had ecchymosis on their surface, and contained thick black tarry material.

The spleen was somewhat enlarged, very friable and con-

gested. The pericardium contained about 2 oz. of deeply stained yellow fluid.

The heart was covered with a thick layer of fat, especially on its right and posterior surfaces. The mesentery was very fat and stained yellow. The bladder was full.

Comment.—From the post-mortem appearances the case was in all probability yellow fever.

CASE VI.—"Private, R.G.A., died in the military barracks at Tower Hill, on 16th July 1910, and by the courtesy of Colonel Sutton, S.M.O., R.A.M.C., I was afforded an opportunity of seeing specimens removed at the post-mortem examination, and the clinical notes on the case. Death was undoubtedly due to yellow fever" (Dr Kennan).

CASE VII.—Maisie Hayer, female Syrian, aged about thirty-five years, lived in Fourah Bay Road. Reported by Dr Mayhew.

17th July.—Stated to have been taken ill.

22nd July.—First seen by the doctor; temperature 104·2°; anxious expression; slightly jaundiced; pulse about 100; complaining greatly of cramping pains in arms and legs; given 10 gr. bihydrochlor. of quinine intramuscularly; had had attacks of fever on and off for some time; present illness began three days previously; had been vomiting, with looseness of bowels; removed to P.C.M. Hospital; given further injection of quinine, bihydrochlor. 10 gr.; tongue clean; tenderness in epigastric region; vomited once in evening grey liquid, not black; temperature remained 104·2°; put under mosquito net; passed per rectum what looked like undigested rice; cramping pains severe.

23rd July.—Temperature began to come down in early morning at 4 A.M., 103·6°; at 8 A.M. to 100°; no sweating; patient became collapsed; 12 midday, temperature 99°; patient vomited black material resembling coffee grounds three times at 8 A.M.; patient not having passed urine since admission, a catheter was passed, and about 8 oz. bile-stained urine was drawn off containing much albumin; at 2 P.M. patient was obviously dying; cold extremities; pulseless; gasping respiration. She died about 5.45 P.M. In a blood film no malarial parasites were seen.

Post-mortem.—The stomach contained a quantity of black fluid; the mucous membrane was studded with punctiform hæmorrhages. The liver was greyish in colour, but the light was too bad to make out much about it. Although the patient had been dead only an hour, post-mortem "staining" was well marked on the back.

CASE VIII.—Acting on information received, that a Syrian had died suddenly on the night of 26th July 1910, and that application for a grave had been made, and that he had not been attended by a doctor, I communicated with the coroner who, after he had made inquiry, ordered the body to be removed to the mortuary, where a post-mortem examination was made by Dr Renner, M.O. The result showed that death was due to yellow fever, and the verdict of the jury was given accordingly. The Syrian's name was Nazmadeen (male, adult). He had lived in a house on the same side of Fourah Bay Road, and a few houses removed from that in which the previous case of Maisie Hayer (Syrian woman) had occurred, and which ended fatally in Princess Christian Mission Hospital on the 22nd inst., that case having its onset on or about the 17th, *i.e.*, the day after that on which Private died in Tower Hill Military Barracks, his case being the first of the "recrudescence" series.

R. H. KENNAN, Ag. P.M.O.

CASE IX.—A native clerk, aged twenty-three, residing at Henry Street, W. Freetown.

27th July.—Stated he had been taken ill, suffering from an attack of fever, with much pain in chest and back regions; also that he attended the hospital on the same day as an out-patient in the Government official department.

28th July.—He went home and the symptoms were aggravated, so that he was unable to report himself to out-patients' department that morning. This was reported to the doctor, who went directly to see him. On this day he began to have vomiting very frequently, so that he could scarcely keep down anything in the way of medicine or nourishment.

29th July.—Visited by the doctor, who ordered him to go into the hospital. On admission temperature 104° F.; com-

plains of the pain over abdominal region, especially over liver and stomach. States that the vomiting was troublesome, and that it was all of liquid consistence, yellowish green in colour; but since coming in he has not vomited. Bowels fairly free, pulse 120, and respirations 28; tongue furred; eyes very much jaundiced; general condition well.

31*st July*.—Patient feels a good deal better this morning; the temperature 99·4°; states that the pain in chest and abdomen is a little better too, but in the back is still going on, though not so severely as yesterday. Slept fairly well; urine high coloured and full of deposit; urine examined; sp. gr. 1025; reaction strongly acid, highly albuminous; bile also found.

1*st August*.—Temperature normal; complains of much pain over the liver, and weakness; eyes very much jaundiced; urine very high coloured and greenish, with much deposit; was slightly delirious and restless during the greater part of the night; vomited once, but only the clear mixture which was given to him; had one stool of a yellowish colour.

2*nd August*.—Temperature 104° F.; slight delirium; restless and weak; urine stained bed-clothes dark yellowish-green colour; pulse 140; respiration 30; death, preceded by a slight convulsion.

Post-mortem examination.—Friends allowed a limited examination only. An incision 3 ins. long was made in middle line of epigastrium; the stomach was brought out, opened, and large quantity of coffee-ground fluid was emptied from it. A portion of stomach was removed towards the cardiac end, the mucous membrane was found to be marked out with arborescent congested capillaries. Liver: a small portion of liver was removed; this was typically boxwood colour.

Case X.—Mr C., European employee, æt. twenty-five, living in Rowden Street. Has lived in the Colony one year and three months. Takes quinine regularly and has had malarial fever. He had charge of a shop in Kissy Street. Patient was taken ill with chill, headache, pains in back and legs, vomiting. On the third day of illness, headache and pains more severe, temperature 103°, pulse 120, no jaundice, no albuminuria, no epigastric

tenderness, no malarial parasites in the blood, conjunctivæ congested. Subsequently patient improved and recovered.

Suspicious cases.—In addition to the well-marked cases of yellow fever described above, and about which there could be no question, there also occurred a certain number of suspicious cases which, knowing that *Stegomyia* existed in Freetown, might have been the mild form of the disease. I have stated elsewhere that this is of common occurrence in all outbreaks of yellow fever, but naturally most frequently met with in those countries where the authorities were not on the lookout for the disease. The following four cases are examples; there may have been others.

6th June.—A child was taken ill and developed a temperature of 104°, but recovered.

1st July.—Mr A. S., a European, was taken ill with lassitude; body pains; headache; malarial parasites were absent, and there was no albuminuria. On the following day the temperature was 101·2°. Patient recovered.

25th June.—Case of Dr J. W., arose feeling unwell; headache; white, became later more severe; temperature 100° at 7 P.M.; loss of appetite; some nausea.

26th June.—Temperature at 2 A.M., 102°; and at 7 A.M., 104°; later 104·6°, with a pulse rate of 94; marked photophobia; some restlessness and vomiting.

27th to 29th June.—Patient became convalescent.

Case of R. W.

20th June.—Taken ill, with symptoms of loss of appetite and temperature 101°.

23rd June.—Temperature rose to 101° and 103°. No malarial parasites were found in the blood; nor was there albuminuria; later, however, slight jaundice and gastric symptoms developed, but this subsequently passed off.

I lay no stress upon these cases, because now that the outbreak is over it would be quite impossible to prove anything definite one way or the other. But there did occur some months previously in Freetown cases which, when looked back upon in the light of the symptomatology of the well-marked

cases, might in the opinion of the medical men who attended them have been genuine well-marked types of the disease.

These cases are of the greatest importance, because if yellow fever, they show that infection was present in Freetown prior to the outbreak in 1910. If it is agreed that yellow fever was endemic in Freetown, there is nothing unusual in cases appearing prior to the 1910 outbreak. At a meeting of the medical men present in Freetown, held at Government House on 4th August 1910, it was stated that in the light of recent events a fatal case which had been diagnosed as one of phosphorous poisoning in November 1909 might in reality have been a case of yellow fever. The patient had no albuminuria, nor were malarial parasites found in the blood. Black vomit was, however, one of the more suspicious symptoms; the temperature was 103°. Another case was mentioned, in which in 1908 the diagnosis of gastric ulcer was made in the case of a Syrian. The patient in question vomited a large quantity of what appeared to be undigested blood, and died the same night. The doctor who attended this case now thinks that it might have been a case of yellow fever. Indeed, the opinion was expressed by many of those present at the meeting that cases of yellow fever had been mistaken for other diseases in the past, and that in all probability the disease was endemic.

Histories of Yellow Fever Cases at Secondee, Axim, and Sawmills, 1910. (Notes furnished by Drs Rice and Ralph.)

Case I.—Mrs C.

12*th April.*—Taken suddenly ill, with severe headache and prostration, and temperature 105°.

13*th April.*—Visited by Dr Ralph; temperature 105°.

16*th April.*—Jaundice developed, also vomiting; the vomit was greenish in colour, and scattered through it were black specks like "fly spots." There was no albumin in urine. Pulse 84; temperature 101°-102°.

17*th April.*—Temperature 104·4°; jaundice general.

18*th April.*—The motions were clay-like; no albuminuria;

vomiting diminished; jaundice deeper; pulse 78; bile in urine.

20*th April.*—Temperature 101·2°-102°; conjunctivæ deep yellow, also whole body.

21*st April.*—Temperature 101°-102°; vomiting almost ceased; feels better.

23*rd April.*—Temperature 97°-98°.

24*th April.*—Temperature 98°-99°.

25*th April.*—Temperature 97°-99°; taking solid food, but jaundice still very deep.

27*th April.*—Temperature 98°-99·4°.

28*th April.*—Sailed for England, but jaundice still marked.

Comments.—No albuminuria or suppression of urine developed.

Comment.—The kidneys in this case do not appear, therefore, to have been affected. The jaundice increased, and the slowing of the pulse is no doubt associated with this symptom. The fly spots in the vomit indicate the very early commencement of congestion of the stomach; they did not, however, deepen and pass into black vomit.

CASE II.—Mr R. C.

27*th April.*—Taken suddenly ill; temperature 104°; frontal headache; vomiting.

28*th April.*—Examined by Dr Ralph; temperature 101·8°-102·8°. Conjunctivæ congested; photophobia; tongue furred; pulse 80; headache became severe and persistent; vomited; urine scanty; no albumin; tinge of jaundice.

29*th April.*—Temperature 101°; headache persists; intramuscular injection of 9 gr. of quinine given without effect; vomiting troublesome and showed the black specks; urine contained a trace of albumin; pulse now 78; restless.

30*th April.*—Patient was able in the morning to attend to a little business and to sign his name to a letter. Very little urine was, however, passed during the day. At 10.30 A.M. had a convulsion, sank, and died. The "jaundice, which had been present from the beginning, became much more marked after death, the body being a dark brown colour."

Comment.—In this case the kidneys appear to have been early severely affected; the headache and vomiting were more

severe and persistent. It is therefore a more severe case than the previous one. It was the case which caused the medical officer, Dr Ralph, to consider that he was dealing with yellow fever. Quinine, it will be noted, had no effect.

Note.—In previous years Mr R. C. had lived at a considerable distance from the native town. Immediately prior, however, to his fatal illness he had lived in the native town, and no doubt became whilst there infected with yellow fever.

CASE III.—Mr W., white man.

8th May.—Taken suddenly ill in the early morning; temperature 105°; severe headache; some vomiting. At 8 A.M. seen by Dr Ralph. He showed the following symptoms: intense headache; face very flushed; eyes injected; tongue furred; great prostration; there was no albuminuria.

9th May.—Fair night; slight vomiting; two intramuscular injections of 9 gr. each of quinine given without any marked effect, temperature being 103°; urine very scanty and albuminous.

10th May.—Temperature varied from 98·4° to 100·8°; in the evening pulse 72; troublesome hiccough now set in with vomiting; the vomit contained black specks; muscular twitchings appeared and the jaundice deepened markedly; scleræ deep yellow; convulsions increased in frequency, and at 11 P.M. patient vomited a large quantity of black vomit.

Comment.—This case is more severe than the preceding one; the kidney is markedly affected, the jaundice was intense, and the pulse became slow, showing, no doubt, profound change in the liver; the gastric symptoms became more intense, ending in black vomit on the third day.

Note.—Mr W. had visited the bungalow occupied by Case II., but so had another gentleman, who did not get it. There was the other possibility that Mr W. contracted the infection at the warehouse, close to which there was a native house.

CASE IV.—Rev. H. B., white man, missionary, living in commercial town.

9th May.—Patient seen by Dr Ralph, and complained of headache; lassitude; temperature normal, but in the afternoon it rose to 103°; pulse 90; eyes congested.

10*th May.* — Constipation; intense headache; jaundice appeared; intramuscular injection of quinine.

11*th May.*—Injection of quinine was repeated, but without effect on temperature; pulse now 80; albumin appeared for the first time; jaundice deepened; towards end of day patient became restless and delirious.

12*th May.*—Temperature 103°, pulse 80; black specks present in the vomit; urine more albuminous.

13*th May.*—Symptoms progressed, and at midnight black vomit ejected by the pump-like action of the stomach, there being no effort. At 4 A.M. the following morning, 14th May, patient died.

Comment.—This case is like the preceding one. The kidneys, liver, and stomach appear, however, to have been more severely affected.

Post-mortem made twelve hours after death.—Body well nourished; surface stained pale yellow, with well-marked petechiæ about the neck and shoulder; *rigor mortis* still present; body slightly warm; conjunctivæ stained a deep saffron colour. *Heart and Lungs.*—Beyond staining of valves nothing abnormal. *Liver.*—Slightly enlarged; pale yellow on section; but was more congested than some of the other cases; considerable fatty degeneration was present. *Kidneys.*—Showed hæmorrhages under capsule, which stripped readily, but these were more marked at bases of pyramids; no hæmorrhage into pelvis. *Spleen.*—A little swollen; no marked microscopic change. *Stomach.*—Contained black fluid, with marked congestion of the mucous membrane. *Intestines.*—Lower part of small intestine contained black, tarry matter; small hæmorrhages could be seen under the peritoneum.

CASE V.—Mr W. R., white man, living in commercial town, close to the market.

9*th May.*—Dr Ralph called in and found patient complaining of headache; pains in the limbs; temperature 104°; pulse 84; later, face flushed; eyes injected; edges of tongue red.

10*th May.*—Passed fair night; temperature varied from 102° to 105° at 6 P.M.; pulse 72; no albuminuria; was given two intramuscular injections of quinine, which had no effect.

11*th May*.—Vomited during the night; black specks in the vomit; temperature 104°-105°; delirium marked; tried to get out of bed (shouting); albumin appeared for the first time.

12*th May*.—Temperature 105°; heart failing; albuminuria more marked; then suppression of urine for eighteen hours before death; the delirium was marked throughout the night; constant black vomit was present; marked convulsions took place five hours before death.

Comment.—A severe case like the two preceding ones; kidneys and stomach more profoundly affected.

CASE VI.—*Adam*, a native black man; post-mortem 14th May 1910; said to have died the previous night, 13th May; tall muscular man; *rigor mortis* passing off; conjunctivæ deep yellow; some "black vomit" on table escaped from mouth. *Heart*.—Beyond slight dilatation was healthy; valves deeply stained. *Lungs*.—Old adhesions over right base and about upper portion of lower lobe; congested at bases, but crepitant. *Liver*.—A little enlarged; almost lemon colour on section; tissue appeared to be softer than normal; I think fatty degeneration was present. *Spleen*.—Enlarged; capsule thickened; tough on section. *Kidneys*.—Showed small hæmorrhages in the cortex; not very marked under capsule, which stripped readily. *Stomach*.—Contained "black vomit." *Intestines*.—Lower part for a short distance contained black, tarry mucous matter. All tissues deeply stained, including cartilages and ribs.

CASE VII.—Kroo-boy, of the Gold Coast Machinery and Trading Co. Brought to the mortuary dead on 16th May 1910. Post-mortem made about twelve hours after death. Body well nourished, muscular; conjunctivæ stained deep yellow colour. All tissues were of a saffron tint.

Heart.—Valves showed the general staining, otherwise normal. *Lungs*.—Old adhesions on both sides, but crepitant throughout. *Liver*.—Slightly large on section, of a pale yellow colour, very suggestive of fatty degeneration. *Kidneys*.—Showed hæmorrhages in the cortex, chiefly at base of

pyramids. *Spleen.*—Enlarged, capsule very thick and tough, adherent to abdominal wall. No marked change beyond toughness on section. *Stomach.*—Contained small quantity of dark, almost black fluid, with marked congestion of mucous membrane. *Intestines.*—In lower part contained black tarry matter. There were small red patches under the peritoneum, which appeared to be hæmorrhages. Congestion of mucous membrane not so marked as in the stomach.

CASE VIII.—Mr B., living in commercial town, in an inferior class hotel, was under the care of Dr Hayford, but owing to the necessary evacuation of that part of Secondee at night, he slept in the Colonial Hospital (part of which has been turned into a dormitory) on the 17th inst. Dr Ralph first saw him on the morning of 18th May, when he had a high temperature, but would persist in going into the town. He returned at 2 P.M. obviously very ill, and was at once admitted into the hospital. Temperature 103·8°; flushed face; marked injection of the eyes, which were very bright and watery; tongue furred, red tip; pulse 90, full and hard; temperature 101° at 8 P.M.

19th May.—Urine passed during the night contained albumin. Conjunctivæ jaundiced. Was very restless and did not sleep much. Temperature 6 A.M., 101°; 6 P.M., 101°. Pulse much softer, not so full, 70. There was some nausea, and he vomited a lot of milk, but no sign of "coffee grounds." Was restless and talkative all day, gradually getting more jaundiced. Constantly trying to get out of bed.

20th May.—Urine contained more albumin. Temperature 6 A.M. 102°, 6 P.M. 103°; pulse better, 70. He was very restless and excited all day, but slept fairly well at intervals.

21st May.—Very restless and delirious all night. Vomited at intervals, the "black specks" in vomit much more pronounced. Urine almost solid with albumin. Very small quantity, about 5 oz., passed during the night. Temperature 6 A.M., 103°; 6 P.M., half an hour after death, 104°. He was very delirious and excited all day, tearing up mosquito net and blankets. At the end vomited large quantity of "black vomit." Jaundice gradually got deeper all day, and before death there were petechiæ about the neck and upper part of trunk. He had

slight convulsion at the end. No urine was passed for twenty-four hours.

Comment.—This case shows very well the increase of the gastric, hepatic, and renal symptoms.

Post-mortem made on 23rd May, about twelve hours after death. Body deep yellow with petechiæ about the neck and upper part of trunk; conjunctivæ deep saffron colour, body not well nourished, thin and spare. *Heart.*—Valves healthy, stained colour. *Lungs.*—Normal. *Liver.*—Slightly enlarged. On section pale yellow, seemed tougher than normal. I consider fatty degeneration was present. *Kidneys.*—Hæmorrhages into cortex very small, but present chiefly at the base of pyramids; capsule stripped readily. *Spleen.*—Enlarged, soft. *Stomach.*—Contained "black vomit," marked congestion of mucous membrane. *Intestines.*—Small quantity of black, tarry matter.

CASE IX.—Rev. A. T. R. B. came from Accra on 12th May and stayed at the Mission House.

20th May.—Patient was brought to the hospital at 5 P.M., semi-conscious, in a hammock, and was at once admitted; temperature 103°. There was no complaint of headache, only general feeling of weakness and discomfort. Given phenacetin 5 gr., to be repeated in half an hour if not sweating, with hot lime drinks. This started a profuse sweat, and temperature fell to 101.° When seen late in the evening he expressed himself as being better; no albuminuria; pulse full and hard, 80.

21st May.—Temperature 6 A.M., 101°; 6 P.M., 103·2°. Was given intramuscular injections of quinine, bihydrochloride 9 gr., at intervals of twelve hours, which produced no effect on the temperature; faint trace of albumin in urine; bowels confined, slight jaundice. Was quite cheerful and comfortable during the day, but complained that light hurt his eyes; pulse not so hard, 76. During the night began to complain of headache and was a little deaf. More albumin in urine, which was very acid, 102·2°.

22nd May.—Temperature 6 A.M., 102°; 6 P.M., 104·4°. Late in the afternoon mental symptoms developed. Incoherency and forgetfulness, but was able to write letters. Bowels open three times; stools paler; passed only 12 oz. of urine and very little with the stools.

23rd May.—Temperature 6 A.M., 102°; pulse 72, weaker. Slept fairly well. Bowels open during the night but very little urine passed. There was a little vomiting of blackish fluid. Jaundice was very much more pronounced, the conjunctivæ being of a deep yellow colour. In spite of stimulants his heart gradually failed, and he died at 10.30 A.M.

CASE X.—Native, post-mortem examination held 23rd May. Wife of a clerk living at Essikadu. Post-mortem held about sixteen hours after death. Well-nourished healthy woman; considerable enlargement lower part of abdomen; conjunctivæ deep yellow colour.

Lungs and Heart.—Normal, all tissues stained deep yellow. *Liver.*—Slightly large; yellow colour on section; very bloodless; looked like fatty degeneration. *Stomach.*—Contained small quantity of black fluid; mucous membrane congested. *Small intestines.* — Lower part contained blackish mucous material for a considerable length. *Spleen.*—A little enlarged, otherwise normal on section. Was perhaps a little tough. Uterus very considerably enlarged; definite hæmorrhages in both ovaries; Fallopian tubes very much swollen and enlarged; fimbriated extremities much congested. The patient had had a miscarriage (22nd May) previous to death the same day.

CASE XI.—Mr A. H. H., assistant commissioner of police, arrived in Secondee from Accra on 14th May. His duties consisted of superintending the evacuation of the declared infected area each evening at 5 P.M. This also necessitated a later visit to see that there were no Europeans sleeping in the area.

22nd May.—Early on the morning he was awakened by severe frontal headache, pain in the eyes, and photophobia; temperature 103°. He was seen by Dr Purkis about 6.30 A.M. On admission to the hospital his temperature was 103·4°; pulse full and hard; no albumin in urine.

23rd May.—Temperature in the morning and afternoon was 103°; pulse 90 full. Still had persistent headache; was very restless all night, but dosed at intervals. Intramuscular injection of quinine bihydrochloride, 9 gr., was given at 10

A.M. and repeated at 9 P.M., but had no effect on temperature. Stools were light clay-coloured. Had slight nausea but no actual vomiting; slight jaundice.

24th May.—Was slightly delirious during the night but slept at intervals; vomited after a cup of tea at 6 A.M. This contained small "black specks" in the mucus and tea; temperature 6 A.M., 103°. Passed no urine during the night, but a specimen obtained late on the 23rd contained albumin; temperature 6 P.M., 102·6°; jaundice much more pronounced.

25th May.—Vomited black grumous fluid; no urine passed during the night; bowels open, stools loose, light clay colour. Heart failed rapidly and he died at 7 A.M. There was no convulsions before death.

CASE XII.—Mr R. D., white man, agent, living in commercial town.

19th May.—Was taken ill suddenly. He vomited and had a high temperature 105°, first seen on the morning of 20th May. He then had a temperature of 102°. Tongue furred, face very flushed, eyes injected. He took 10 gr. of quinine in solution, and 10 gr. that night when his temperature was 100·6°, but without effect.

21st May.—Removed to hospital. On admission his temperature was 101°; pulse full and hard; skin moist; intense headache, otherwise little complaint of illness; temperature 6 P.M., 101°. Was given an intramuscular injection of quinine, 9 gr., which produced absolutely no effect on the temperature. No albumin in urine, which was acid.

22nd May.—Temperature 6 A.M., 101·2°; 6 P.M., 102°. Seemed more comfortable. Bowels open, motions normal in colour. Stated he had passed a good night. Took plenty of nourishment. The jaundice which had been but slight began to become deeper. Conjunctivæ very injected, and measles-like rash appeared on the face, upper part of trunk, and back of hands. Bowels open twice, was passing but little urine, only 12 oz. in twelve hours; no albuminuria; pulse much the same as yesterday. Liver was slightly enlarged and tender, but the patient is very stout; spleen not palpable.

23rd May.—Slept at intervals during the night. Tempera-

ture 6 A.M., 102·4°; 6 P.M., 102°; pulse not so good, given brandy and champagne as required; jaundice much more pronounced; conjunctivæ now deep saffron colour. The eyes look swollen and watery. Albumin in urine; more lethargic; not so inclined to talk but had tried to smoke. There was a little nausea and vomiting at intervals during the day; no "black specks" in vomit which was of a reddish colour like the dregs of beef tea.

24th May.—Temperature 6 A.M., 102°; 6 P.M., 103·4°. Had passed a fair night. Bowels open, is not passing much urine; jaundice very deep; measles-like rash fading; tongue fairly clean; skin moist; pulse soft—70. Is taking nourishment well with small quantities of stimulant. There was a little vomiting which had changed its character, being now marked with "black specks" more mucous than before; urine highly albuminous.

25th May.—Seemed fairly well; temperature 6 A.M., 101°; 6 P.M., 101°. Much more lethargic in the early part of the day; later became restless; said he could not stay in bed; kept on getting up and sitting on the side of his bed. No complaint of severe headache, only a dull ache; passing very little urine in the twenty-four hours; in consequence saline injections were ordered to be repeated every four hours.

26th May.—Slept a good deal last night; continues to take nourishment well; temperature 6 A.M., 99·4°; 6 P.M., 98·8°; urine very albuminous; vomiting not frequent, chocolate-coloured; pulse 80, fairly full and soft. The pulse began to fail a little about midnight. During the greater part of this day he was lying apparently asleep, bathed in a profuse sweat. Measles-like rash quite gone, but petechiæ appeared for the first time about the neck and trunk, also on the hands; jaundice very deep; hiccough very troublesome.

27th May.—Some twitching about the angle of the mouth and hands first noticed; temperature 98·8°. Was semi-conscious till the afternoon, when the muscular twitchings became much more marked; pulse failing; no urine since 5 A.M. Rectal saline injections were kept up. He died quite quietly at 10.45 P.M. There was considerable hæmorrhage from the mouth after death.

The post-mortem examination was held eleven hours after death. The face and neck deeply cyanosed; petechiæ about the

upper part of trunk and on the hands, *rigor mortis* well marked; body slightly warm, the whole of the abdomen and extremities of a saffron tint; conjunctivæ deep yellow.

Heart.—Valves stained deep yellow, otherwise normal. There was early evidence of fatty degeneration of the muscle; considerable amount of fat on exterior. *Lungs.*—Bases congested but the whole of both lungs crepitant. *Stomach* contained black grumous fluid. There was evidence of hæmorrhage from the mucous membrane which was deeply congested. *Intestines* contained similar hæmorrhagic matter, with marked congestion chiefly in the lower part of small intestines. *Bladder* appeared to be healthy and contained about 8 oz. of bilious urine. *Spleen* slightly enlarged and congested. *Kidneys.*—Cortex thin with hæmorrhages around base of pyramids and under capsule which stripped readily. *Pancreas.*—Beyond the deep staining, which was a marked feature of the whole of the abdominal viscera, no obvious change. *Liver.*—There was slight enlargement more marked in the left lobe than the right. On section it was of a deep yellow colour, and had rather the appearance of a "nutmeg." There were well-marked hæmorrhages in the tissues, covering the gall bladder, but not marked on mucous membrane. The gall bladder contained about 1½ oz. of deep green gall. The whole of the abdominal and thoracic viscera appeared to assume a deeper tint of yellow on exposure to the air. The blood was also of a darker colour than normal.

List of Doubtful Cases previous to the Outbreak of Fatal Cases, taken from the Secondee Case Book.

J. C. E. Hepatitis. Date 17/9/04.

Symptoms—Temperature 101°; pain in epigastrium; coated tongue; jaundice recovered. W. S. W.

Comment—Suspicious case (Boyce).

T. B. W. Remittent Fever. Date 20/9/04.

Admitted with temperature 105·2°; headache; tongue coated; recovered. W. S. W.

F. G. Remittent Fever.

Admitted into Colonial Hospital; temperature 103·6°, with marked bilious symptoms; recovered.

W. H. D. Hepatitis. Admitted 17/11/04.

Temperature 100°; usual symptoms hepatitis; recovered.

C. H. C. Insolation and Fever. Date 14/11/04.

Admitted with intense headache; nausea; vomiting; temperature 104°. Very suspicious case of yellow fever (Boyce).

J. B. Hepatic Congestion and Fever. Admitted 23/1/05.

Temperature 104°; symptoms—congestion of the liver and other bilious symptoms; temperature 100°-101°, lasted ten days; recovered.

W. H. Date 14/2/05.

Symptoms of intermittent fever and gastric catarrh, with marked gastric disturbance; eight days' duration; recovered.

A. T. N., District Commissioner, æt. twenty-eight. Admitted 23/2/05.

Diagnosis—Remittent fever germinated fatally.

G. F. S. Diagnosis—Remittent Fever. Admitted 26/2/05.

Temperature 103°; vomiting; fever characterised by gastric and bilious symptoms; recovered in ten days.

F. G. M. Diagnosis—Enteritis.

Temperature 103·4°; vomiting six days.

H. H. H. Intermittent Fever; recovered. Date 21/3/05.

Temperature 103°; nausea; vomiting and gastric disturbance; recovery; quinine did not affect the temperature.

G. F. S. Gastric Catarrh and Fever. Date 17/3/05.

Admitted suffering from vomiting; continuous fever and prostration; invalided.

T. H. F. Hæmoglobinuric Fever; recovered. Admitted 14/5/05.

Temperature 103°; weakness; urine and alb. looks like that of blackwater; skin very yellow; much bilious vomiting; temperature 104°; vomiting continued; recovery.

H. C. R., Mines Accountant. Blackwater Fever. Date 8/6/05.

Admitted to hospital with well-marked symptoms of blackwater; urine alb.; skin yellow; vomiting same afternoon; temperature 104·2°; recovery.

H. H. Intermittent Fever.

Admitted vomiting and great irritability of stomach; recovery.

E. D. M'F. Bilious Remittent Fever. Admitted 26/8/05.

Temperature 102·6°; jaundiced; headache; nausea; temperature rose to 104·6°.

A. W. H. Diagnosis not given. Death. Admitted 5/1/07.

6th January 1907.—Vomiting commenced; black vomit on the 7th; eyes intensely congested and jaundiced; skin yellow, with purple patches of discoloration; temperature 103°; delirium; coma; epistaxis.

Post-mortem.—Skin yellow; conjunctivæ yellow; lungs congested; liver fatty; kidneys congested; trace alb. in urine; intestines contained same material as the vomit. Undoubted case of yellow fever; temperature 102°, with a pulse of 96 (Boyce).

Mr K. Dysentery and Blackwater Fever. Admitted 13/1/04.

Symptoms.—Vomiting; epistaxis; blood in stool; blackwater; skin jaundiced; pulse 96; epistaxis; recovery.

John (Convict Prisoner). Cirrhosis of Liver. Date 26/4/07.

Symptoms.—Jaundice; bleeding from gums; eyes irritating; coma; death.

C. L. Blackwater Fever. Date 26/9/98.

Admitted with severe rigors; vomiting and incessant hiccough with blackwater; death.

A. M. D., Senior Nursing Sister. Blackwater Fever. Date 18/6/08.

Arrived 10th May 1908; attack of fever; severe vomiting; hæmoglobinuria in urine; recovery.

H. L. Hepatitis, Asthenia. Admitted 2/2/10.

Had suffered from malaria on admission; vomiting large quantities of bilious matter; abdomen tender; temperature rose to 103°; death.

R. C. Admitted 19/3/10.

Arrived in Secondee, Thursday, 10th March, from Accra; complains of vomiting; headache; temperature 101·6°; no albumin.

21*st March.*—Temperature 103°; nausea.

23*rd March.*—Temperature 102°; nausea; vomiting; skin yellow; later temperature 105°.

Note.—The patient lived in the same compound where Mr Aisly resided. Dr Ralph regarded the case as one of typhoid.

Outbreak of Yellow Fever at Saw Mills, 1910.

A case of yellow fever was reported at Saw Mills Camp, a station 12½ miles along the railway, north of Secondee. This camp consisted of a small village of about thirty-five houses occupied by natives, and of two European bungalows occupied by the white men.

The village was very dirty, and littered with innumerable odds and ends which contained water, and gave rise to enormous numbers of mosquitos. The outbreak at this small isolated camp is of great interest, as it shows in a most conclusive manner the independent occurrence of a sporadic case 12½ miles from the focus at Secondee. According to the history of the case, the patient had not been near Secondee for three weeks

previous to taking ill. It would therefore appear that this case originated in Saw Mills, precisely as the case in Secondee, from *Stegomyia*, which had become infected from the disease present in an unrecognised form in the natives. In other words, the case is further evidence of the endemic origin of yellow fever in West Africa.

Symptoms of the case.—Mr P. M., æt. forty-six.

15*th June.*—Reported to have taken suddenly ill, with headache and temperature of 105°.

16*th June.*—Conditions the same, with bilious vomiting and hiccough and sleeplessness.

17*th June.*—Conditions unaltered: temperature 105°.

18*th June.*—Was brought down to the hospital at Secondee. He developed black vomit; great weakness; temperature 98°; pulse 60; hiccough and vomiting became persistent; jaundice set in skin and conjunctivæ. During the night the temperature was 99°, and pulse very slow, 56; vomiting incessant. The quantity of urine passed was small, and contained albumin.

19*th June.*—Patient delirious; black vomiting continued up till death.

This is a very clear case of severe yellow fever, showing jaundice, Faget's sign, albuminuria, and intense black vomiting. There was only one paroxysm of fever, and no relapses.

Occurrence of a Sporadic Case of Yellow Fever at Axim, 1910.

As in the case of Saw Mills, so in this one at Axim, there occurs an independent sporadic case, the infection in which case cannot be traced to the outbreak at Secondee. We therefore are obliged to conclude that, as in Secondee and in Saw Mills, the disease originated on the spot—in other words, that yellow fever is endemic and exists amongst the natives. This opinion is that adopted by the Senior Sanitary Officer, Dr Rice, who observed on the spot the whole outbreak this year, 1910.

Symptoms.—Mr G. R., newly arrived from England.[1]

12*th July.*—Taken ill with headache, and temperature 104°; pulse 85; no albuminuria; no jaundice or vomiting; was given quinine.

13*th July.*—Temperature 104·2°; pulse 87.

14*th July.* — Temperature 101·8°; pulse 76; patient appeared much better and more cheerful. At 8 P.M., however, the doctor was suddenly summoned, and found the patient delirious; very weak; temperature 101·2°; vomited; first vomit greenish, the others were white.

15*th July.*—Patient delirious and raving; reported not to have passed water since the 13th; died.

Post-mortem examination showed the liver enlarged and yellowish; jaundice well marked (it had not appeared before death). The bladder was empty; the stomach was not injected; the spleen was not enlarged; the kidneys enlarged.

Although the notes of the case are scanty, the case appears to me to be a clear one of yellow fever. It is a fever of one paroxysm, lasting four days, and terminating fatally; the pulse was comparatively slow, gastric symptoms appeared on the second or third day. It is true that there was no black vomit, but there was the white vomit which has been often recorded. Nor did jaundice appear till after death: this is also very common. Then there is the suppression of urine. The liver is found to be yellowish. In my opinion, the symptoms and findings can only point to yellow fever.

[1] Mr G. R. was a fellow-passenger with me on my way out to the Coast (Boyce).

CHAPTER XI

DIAGNOSIS

Difficulties of diagnosis met with during epidemics.—Although a "well-marked" case of yellow fever cannot readily be mistaken for any other disease, it unfortunately happens that at the commencement of an outbreak the "well-marked" cases are rare, whilst the milder types to which I have already fully alluded, predominate. Therefore it becomes a matter of supreme importance to review the histories of past outbreaks, and to learn from them the practical difficulties which medical men have had to contend with, in arriving at the true nature of the disease which had broken out in their midst. After a searching examination of the outbreak of yellow fever in *British Honduras* in 1905, I wrote in my report to the Government as follows, upon the question of mistakes in diagnosis:—

"1. *Malaria.*[1]—In January 1905, two deaths occurred from remittent malaria in subjects aged twenty-two and forty-five respectively, and one death from intermittent malaria in a baby of one and a half months; in February one remittent case in a man of thirty-seven; in March one remittent case in a woman of thirty-two, and in April two remittent cases, one in a man of seventy, the other in a man of thirty. On 14th May, the death occurred of Miss B., from what was registered at the time as *bilious fever and hæmatemesis*, but

[1] The term "malaria" has become in the tropics a kind of medical dust-bin, into which all difficult or doubtful cases of fever are cast without question or microscopical analysis. It takes the place of syphilis in colder climates.

which there is now no doubt was yellow fever. Later, in May, and in June, July, and August, further deaths occurred from what was chiefly described as *malignant malaria* or *remittent malaria;* some of these, there is now reason to believe, as will be shown further on, were caused by yellow fever.

"In the practice of the local medical men there were, of course, numerous cases of malaria as in other years; there appears to me, however, to be no doubt that in January, May, and April the number of 'fever' cases were abnormally high. I am also informed by Dr White, of the Public Health and Marine Hospital Service of the United States, who had charge of the yellow fever prophylactic measures in New Orleans, that the same marked increase of what was returned as 'malaria' was noted previous to the official declaration of yellow fever. It is most probable that some of these cases were yellow fever.

"With regard to the symptoms which the cases of malaria presented in Belize, I have been furnished with the following details. Many were cases of persistent fever, lasting from ten to twenty days, refractory to treatment; *quinine without effect;* fever intermittent; temperature 100° - 103°; pulse slow, 60-80; urine high-coloured and containing bile; bowels constipated. Or again, 'the majority of cases dating from the end of 1904 and commencement of 1905 were complicated with jaundiced scleræ, occasional slow pulse and high temperature. The discoloration of the scleræ was slight, however, and passed off in a day or two.' In the severer forms 'bilious vomiting was recorded.' From what I have myself observed this year, and from the description of cases in former years where disputes have arisen over mistaken diagnosis, I am of opinion that it is often very difficult to distinguish between yellow fever and certain types of malaria, in which there may be present a very slow pulse; jaundice; vomiting; bleeding from the gums; delirium; retention of urine; traces of albumin; constipation; and where quinine does not have any marked effect. In autopsies upon these cases I have noted in one instance intense

congestion of the gastric mucous membrane, precisely as in yellow fever. Nor does the finding of the malarial parasites in the blood by any means settle the diagnosis in districts in which malaria is common; instances were recorded this summer in New Orleans, in which obvious cases of yellow fever presented the malarial parasites in the blood, and we know that malaria does not protect from yellow fever. Unquestionably, the post-mortem is the most reliable method of arriving at a correct diagnosis, and no stone should be left unturned to obtain an autopsy in any suspicious case. It is also very clear that in countries liable to yellow fever a close watch must be kept upon a rise in the mortality from 'malaria,' especially in months when such a rise is unusual.

"2. *The influenza cases.*—Commencing towards the end of April and lasting to the middle of May, there was a short three weeks' epidemic of a disease which was recorded as influenza, and which presented the following symptoms:—The onset of the disease was sharp, but did not last long, the patient going to work on the fourth or fifth day. All the members of a family might be attacked, one after the other, and both fresh arrivals and old residents were equally affected, as also the white and black population. There were no fatal cases. The symptoms consisted of a severe headache; temperature 104°-105°; rapid and extreme prostration; vomiting not as a rule present."

During my investigations last year, 1909, upon the outbreak of yellow fever in *Barbados*, I noted that yellow fever had been repeatedly mistaken for other diseases. The following extract is an example of many similar ones.

1. *Mild cases.*—Extracts from the *Quarterly Sanitary Report* of the parochial medical officer *re* yellow fever in Barbados:—

"Dr T. S. B., No. 1 District, St Michael, 31st March 1908. During this quarter I have attended 29 cases of fever, lasting as

a rule from three to five days: one case of yellow fever occurred in my district on which a post-mortem was made, and it was duly notified. Several of these cases presented some symptoms of yellow fever, and I recognised them as those which in the epidemic of yellow fever in 1881 were called 'febricula,' and were then considered by the medical men who saw that epidemic as very mild cases of yellow fever, as the initial symptoms were the same as those in which yellow fever afterwards developed. Some medical men have considered these cases to be 'gastric influenza,' but as no severe type of influenza was present while these cases occurred, I fail to see how 'gastric influenza' can be associated with these cases. Further, if they were of the nature of influenza, we would have had thousands of these cases instead of the few cases that did occur, as the swiftly spreading and sudden invasion of influenza is well known. It should be noted that when cases of gastric influenza recently occurred in England, there was at the same time an epidemic of influenza of the ordinary kind, which affected the nasal and respiratory parts with which influenza is generally associated."

The following very interesting account of gastric influenza has been kindly furnished me by a consulting physician residing in Barbados:—

2. "*Gastric influenza.*—The term 'gastric influenza' has been applied by some medical men in Barbados during the last year, to cases which other medical men regarded and notified as genuine yellow fever.

"I say this without hesitation, because one man saw two cases and another man one case in consultation with me, which were all regarded by two other men and myself as undoubted yellow fever. These were all fatal in from three to five days in white persons with early albuminuria, lemon-coloured urine; suppression of urine; black vomit; and yellow discoloration of the body; slight during life, and pronounced after death. One of these cases was verified by a post-mortem examination, which revealed a typical 'boxwood' liver; stomach showing arborescent congestion and containing black fluid. Other fatal cases characterised by 'black vomit' were heard of from time to

time, which were not notified, and which were commonly spoken of as 'gastric influenza.'"

3. *Epidemic jaundice* or *Weil's disease.*—This disease was likewise frequently diagnosed in cases of yellow fever. There are points of resemblance, notably sudden onset; lumbar pain; vomiting; high temperature and slow pulse; jaundice. There is not, however, the black vomit or albuminuria.

4. *Dengue fever* gave rise to much difficulty of diagnosis, and very many cases of yellow fever were returned under it.

As the result of my investigations in *West Africa* this year, 1910, it is abundantly evident that a very considerable amount of mistaken diagnosis has taken place in past years, more in West Africa, perhaps, than in any other country with which I am acquainted; for the reason, no doubt, that very little prominence has hitherto been given to this disease, as a disease likely to be encountered any day. Much has, we know, been written upon the existence of yellow fever in West Africa, but this has lain buried in old medical books or naval and military returns, which were never consulted. Further, with the increased attention given to malaria, I am afraid that it was too often taken for granted that coast fevers must all be due to the malarial parasites, without even making a careful blood examination. Both French and German writers upon yellow fever in West Africa have noted that this disease has been mistaken for other diseases, notably bilious remittent fever and pernicious forms of malaria, "bilious remittent fever with morbus cordis," *febris malariæ continua perniciosa nephritica*, etc. Examination of the hospital case books in towns on the Gold Coast in Lagos and at Freetown this year, 1910, showed numerous instances of mistaken diagnosis. The following are a few typical examples arranged under the heads of malaria, remittent fever, etc.:—

I. *Malaria*

There is no doubt that genuine cases of yellow fever have been diagnosed as simple malaria, pernicious or malignant

malaria, blackwater, etc. Moreover, this diagnosis has been made without a careful blood examination.

Examples of Cases diagnosed as "Malaria."

Symptoms of a case at Elmina in 1905:—18*th April.*—Temperature 102·4°; pulse 100; intense head and backache; nausea; epigastric pain.

19*th April.*—Temperature 103·4°; pulse 92.

22*nd April.*—Black vomit appeared, then death. Post-mortem examination showed skin yellow; liver and kidneys congested; black vomit in the stomach. Dr W. M. Elliott considered this to be a case of yellow fever, as also a case diagnosed in March previously as "hepatic fever."

In 1902 at Cape Coast the following fatal case of "malaria":—

Patient admitted with temperature 102°; pulse very slow in comparison with the temperature; later temperature, 108·2°; vomiting persistent; first brown then black; black motion; marked albuminuria; liver yellow; stomach, intestines, and kidneys deeply congested. In this year a severe case of yellow fever was also diagnosed, so that it is obvious this is also a case.

Malignant malarial fever.—Symptoms of a case at Elmina, 1906:—

History of persistent vomiting, ending with coffee-ground vomit; jaundiced skin; temperature 103·6° and pulse 100; fatal termination. No reason is ascribed for making the diagnosis malignant malaria: it is probably one of the numerous cases of assumption.

Case at Saltpond, 1898. Symptoms:—

Temperature 105·4° and rose to 107·2°; violent delirium; skin jaundiced; also cutaneous hæmorrhages over head and shoulders; coma and death. At the post-mortem stomach-wall intensely congested, and contains dark green treacly material; intestines congested; liver peculiar saffron colour with patches of congestion. It is stated that a blood examination showed this presence of pigmented corpuscles. Considering, however, that in the previous year 1897 there had been several cases of yellow fever, I am of opinion that this is also one of that disease.

In 1902 the following fatal case was diagnosed as malarial at Cape Coast:—

Temperature 103°; pulse 80, slow in comparison to the temperature; later pulse 74; vomiting and retching; delirium; later black vomit and black motion; hæmorrhages from the mouth.

Post-mortem.—Skin yellow; stomach congested; there was no blackwater.

Blackwater fever, as is well known, resembles in some respects yellow fever. The essential points to bear in mind are that in blackwater there is a history of undoubted previous attacks of malaria and exposure to malarial infection; secondly, that the disease is a hæmoglobinæmia, and that the "black" colour of the urine is due to the excreted hæmoglobin present in the blood serum. There is of course also albuminuria, but red corpuscles, cell and hyaline casts are usually present in yellow fever. The red colour of the urine, when present in yellow fever, is due to red corpuscles, and casts are very often present. Therefore there is an essential difference in the urine in the two cases. Moreover, the hæmoglobinuria is observed at the commencement of the illness in blackwater, and there is a history that it has come on suddenly or has been brought on by quinine or other factors. In blackwater the urine is diminished owing, it is thought, to the blocking of the collecting tubes in the kidney. In yellow fever there is also frequently suppression; the kidneys, in other words, are profoundly affected. In both diseases there is jaundice, but the jaundice of yellow fever develops during the progress of the disease, and often does not appear until the death agony. There is vomiting in both diseases, but the character of the vomit and the pumping action of the stomach is characteristic in yellow fever. As the mechanism of the jaundice in yellow fever and blackwater differs essentially, one does not find Faget's symptom in blackwater fever. Therefore taken altogether there ought to be no difficulty in differentiating the two diseases.

Closely following upon malaria comes the exceedingly

numerous cases of remittent, bilious remittent, and pernicious remittent fevers, which are often other names for yellow fever. It is admitted that these names convey very little precise information as to what they really are. In most cases they are supposed to be forms of malaria, but why, in the absence of all blood parasites they should be so considered, is very difficult to understand. The more I examine this group, the more I am convinced that in a large number of cases the fever is due to *Stegomyia* infection. The following are some good examples out of innumerable cases :—

II. *Remittent Fever.*

Symptoms of a case at Axim, 1906 :—

10*th May.*—Patient taken ill, feeling "sick and bilious"; temperature 105°; took 25 gr. of quinine.

11*th May.*—Temperature 104°; pulse 120.

12*th May.*—Temperature 104°; pulse 110-120; taking quinine in large doses.

13*th May.*—Temperature 103°; the quinine did not appear to have any effect whatever. Patient wandering.

14*th May.*—Temperature 101°; rigor; skin becomes cold and patient unconscious.

This is a one-paroxysm fever ending fatally on the fifth day, in which quinine had no effect. It is suspicious of yellow fever.

Symptoms of a case at Elmina, 1895 :—

On admission, temperature 103·4°; pulse 124; headache; vomiting; epigastric tenderness. On the following day temperature 101·4°; pulse 90. On the third day, temperature 102°; pulse 80; delirium. This is a suspicious case, especially in view of the fact that yellow fever appeared later in its severe type. There were many more cases of remittent fever during 1895 and subsequently, in some of which the symptoms were very suspicious, notably the pulse rate not following the run of temperature. There is, moreover, no doubt that Dr Elliott, who was present, had his suspicions aroused.

Case at Saltpond, 1895. Symptoms :—

22*nd March.*—Patient unwell; temperature 103°; pulse 100; headache and vomiting.

23rd March.—Vomiting increased; suppression of urine for two days.

24th March.—Temperature 99·8°; pulse 80; vomiting and hiccough.

25th March. — Temperature 103°; delirious; jaundice noticed; vomited coffee-ground material.

26th March.—Temperature 105·8°; pulse 60; yellow colour skin much deeper.

28th March.—Vomited brown material and a cupful of blood; then coma and death. After death the jaundice colour was much more pronounced. There can be no doubt this is a typical case of severe yellow fever.

Case at Accra, 1904:—

CASE I.—Patient had been short time in the Colony and was adverse to mosquito nets.

19th April.—Taken ill.

21st April.—Temperature 104°; pains over body.

23rd to 24th April.—Vomited black coffee-ground material and passed tarry stools.

25th April.—Vomiting persistent and violent; hiccough; delirium; coma; death.

CASE II., 1905.—Symptoms—intense headache; temperature 101·8°, later 104°; albuminuria; quinine without effect; vomiting persistent; became drowsy and less urine passed. Patient was taken ill on the 19th July and died on the 24th.

The first case is, in my opinion, yellow fever, and the second case is very suspicious.

Cases at Cape Coast.—In the year 1901 there were entered seven cases of remittent fever and two cases of bilious remittent fever; of these eight proved fatal. When it is remembered that the following year, 1902, yellow fever was diagnosed, it is most probable that the majority of the previous cases, fatal cases, were also that disease. In 1895, at Cape Coast, two fatal cases of yellow fever occurred and several cases of fatal remittent fever.

CASE I.—Symptoms—great weakness; jaundice; temperature 101° to 105°; anorexia; epigastric tenderness; delirium; patient passed a black motion; later felt better; followed by relapse and death.

CASE II.—Taken ill 28th May with nausea; vomiting and pain in back. *3rd June.*—Felt better and breakfasted. *4th June.*—Much worse; temperature 103°; persistent vomiting; intense lumbar pain. *7th June.*—Patient became jaundiced and died. This patient had arrived on the Coast on 15th May—that is, thirteen days previous to his illness.

In 1901 at Cape Coast there are a series of fatal cases of remittent fever and some cases of bilious remittent; as these were followed by severe yellow fever cases in the following year, there can be little doubt that some of these were genuine yellow fever cases.

In 1902 at Cape Coast there were also fatal cases of remittent fever, followed by typical cases of yellow fever.

Case of Pernicious Remittent Fever at Axim, 1910:—

There was sudden onset; temperature 103°; severe headache; pulse 88; temperature rose to 104°; the intramuscular injection of quinine was without effect; delirium developed; then coma and death. In my opinion, and in view of subsequent development, this was a case of yellow fever.

Cases of bilious remittent fever in Saltpond, 1898. Symptoms:—

CASE I.—Sudden onset; headache; vomiting; temperature 103·2°; skin tinged yellow; conjunctivæ congested and yellow; pulse 90; delirious; later temperature 104°; pulse weak, and death.

CASE II.—Vomiting; temperature 102°; convulsions, and death.

CASE III.—Temperature 102·5°; slight jaundice; pain in head and back; recovery.

CASE IV.—A similar case.

These cases are in all probability yellow fever. They were preceded and followed by genuine cases of yellow fever.

Cases at Cape Coast.—Severe cases of this disease were entered in 1901 and 1904, which were preceded or followed by yellow fever cases. See also case at Secondee, 1910.

After the remittent fevers there comes a list of rarer mistaken diagnoses — for example: Subacute rheumatism,

lumbago, gastric ulcer and gastritis, hepatitis, acute yellow atrophy, etc.

The following is a case diagnosed as hepatitis, which in all probability was yellow fever:—*Symptoms of a case at Elmina*, 1895.—Admitted with great prostration; skin yellow; eyes jaundiced; black coffee-ground vomit; suppression of urine; and death. Dr W. M. Elliott regarded this as very suspicious of yellow fever.

Looking over hospital case books shows that time and time again the medical officers have been greatly perplexed as to what should be their diagnosis in certain cases. They often shunned the notoriety of the more startling diagnosis of yellow fever, and tried to satisfy their conscience by a diagnosis such as the following, which is one of many similar ones:—

"*Resembling yellow fever.*"—Case at Elmina, 1902.—In this case there was intense headache; albuminuria; jaundice; black vomit and black motion; and intense yellow colour of skin before death. This was most probably a case of yellow fever.

III. *Dengue or Dandy Fever*

At present the differential diagnosis between yellow fever and dandy fever in West Africa is not of great practical importance, but probably in the future with the growth of sewage schemes and the increased facilities given to breeding culex, the disease will become as prominent as in the West Indies and other parts of the world.

IV. *Yellow Fever in Children*

Yellow fever occurs in children in forms which do not suggest yellow fever, and there is little doubt that the disease in them passes unrecognised; this was certainly the case, in my opinion, in the Belize outbreak in 1905. Sodré & Couto sum up the case thus:—"During an epidemic of yellow fever, if a child is affected by a fever lasting more than twenty-four hours and followed by symptoms of nervous irritation, the diagnosis in 95 per cent. of the cases will be yellow fever." Durham also draws attention to the part played by children in propagating yellow fever just as they do malaria, scarlet fever, etc.

CHAPTER XII

TREATMENT

THERE is no specific drug which is of any use. Nevertheless, by the careful consideration of the pathology of the disease, and of the probable mode of action of the virus, and bearing in mind the treatment of the experimental inoculation cases of Reed, Carroll, Agramonte, and Lazear, it is possible to adopt a system of treatment which, although exceedingly simple, nevertheless attains to an almost specific position. Summed up, the treatment resolves itself into absolute rest of body, alimentary tract, and abdominal viscera. Allow the patient to do nothing that will cause effort of any kind. In other words, remembering that the patient is suffering from a profound toxæmia, do all that is possible to avoid putting any extra strain on any tissue, but assist the tissues to neutralise or eliminate the virus. Therefore from the very beginning of the "feeling unwell" put the patient under mosquito netting, and forbid getting up to pass water or motions, or sitting up in bed. This action will rest the muscular and vascular symptoms. In the next place, knowing the extreme sensitiveness of the alimentary tract, avoid irritating it, for the least irritation will hasten the intense congestion of the mucous membrane and bring on black vomit. Nature will probably remove from the stomach by an early vomit its contents. Therefore it is not the stomach that requires clearing but the lower gut. This can best be done by a good rectal enema. There is no necessity for calomel provided an enema is handy.

FIG. 18.—A Mosquito-screened Ambulance for the removal of cases of Yellow Fever to Hospital. New Orleans, 1905.

[*To face p.* 208.

Those who have experienced the value of an ample warm saline enema inform me that there is nothing equal to it for the relief it brings. In this way the alimentary tract is cleaned out without putting any extra strain on that viscus. Thirst will be early noticed; again this is a natural reflex, and the patient should be allowed abundance of simple alkaline mineral waters. By this means the blood pressure will be kept up and the secretion from the kidneys maintained. Solid food should under no circumstances be given in the early stages of the disease. If it is given, gastric fermentation, followed by congestion, will be the result; therefore avoid solid food. Later, if the patient shows signs of great prostration or collapse, stimulate with champagne. It may be necessary to maintain the blood pressure by normal saline enemata. Following these lines of treatment, and with constant nursing, all authorities are agreed that at no stage in the disease should hope be given up. For the further guidance of the medical officer I reproduce the more detailed treatment advocated by Dr Guiteras of Havanna, who has had an extensive experience.

The following guiding rules in treatment, given in a recent lecture by Dr Wolferstan Thomas, who has himself had yellow fever, and is now in charge of the yellow fever station on the Amazon, are of practical value, and I reproduce them for the guidance of the medical attendant:—

"The most important points in the management of yellow fever cases are nursing, absolute rest in bed, restriction of all nourishment for the first few days, the use of alkaline waters, and the cautious administration of drugs. You must make yourselves responsible for the entire management of the case, and should:—

"1. Obtain as full a history of the onset as possible, and of the patient's habits, temperament, etc.

"2. Examine your patient thoroughly, and do not postpone the examination. Yellow fever is a disease which runs too rapid a course to permit of any delay.

"3. Examine a fresh specimen of urine. If you have been summoned within the first twenty-four hours it will probably contain no albumin. Give orders to preserve all the urine passed by the patient, and arrange that the necessary covered jars are provided, so that the full quantity for the twenty-four hours can be kept. See that a special jar is always ready for holding the last urine voided.

"Give orders that the patient shall urinate before the bowels move or before a clyster is given. This is important, as later on when the amount of urine is decreasing, much urine may be lost in the bed-pan. Examine the urine for albumin, and test the reaction at least twice daily, until convalescence is established. You have to ascertain the time when the albumin appears, and afterwards the information obtained from the daily examination of the urine as to the amount of albumin, urea, and the reaction will be one of the most important guides to the prognosis and management of the case. Albumin is generally present in this disease, appearing between the second and third day. In severe cases it may only appear for a few hours. In some cases it is never found throughout the course of the disease.

"4. Obtain a trained nurse. Too much care cannot be given to the patient, and severe cases will require two trained nurses. In default of trained nurses your patient will suffer, but it will not be your fault.

"5. Give instructions to have the temperature, pulse, and respirations taken and recorded every two to four hours, and check the results by doing it yourself at least twice in every twenty-four hours.

"6. Order the patient to be screened both day and night. Do not fail to insist on the utmost care being taken to prevent *Stegomyia* feeding on the patient. The lives of

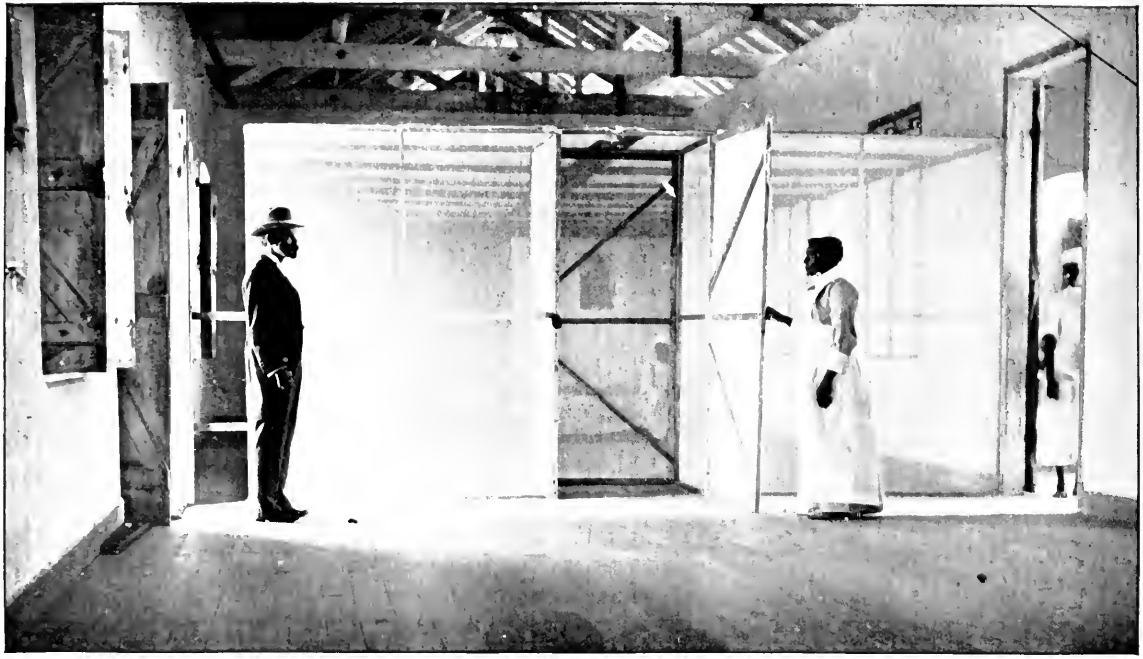

FIG. 19.—A simple form of Mosquito-screened Room, erected inside a ward for the reception of yellow fever cases. Barbados, 1909. (From *Mosquito or Man?*)

[*To face p.* 210.

all non-immunes depend in a great measure on this being observed. Even if you have not made a positive diagnosis, so long as there is the least suspicion that it may possibly be yellow fever, your duty is to insist on the screening. The screening saves your patient from being annoyed by mosquitos, and is an educational factor to the general public. If it is a case of yellow fever, and over the fourth day of the disease, the patient is non-infective, and screening is not absolutely necessary.

"Yellow fever is a disease which allows of little delay. It runs its course in a few days, and you must be prepared to offer battle. Fortunately many cases are of a mild type, and unless something exceptionally wrong is done the case will recover. Such cases are good for your reputation, and as yellow fever exacts a heavy toll, and as it will probably be your misfortune to lose many cases, you will need them. No one who treats many cases of the disease can avoid losing a certain percentage of his patients. There is no panacea for yellow fever as there is for malaria, but a good physician can, by his management of the case, markedly reduce the mortality.

"Eliminative and supporting measures, with the reduction of the congestion, should be our aim. Briefly, we can say that treatment depends on nursing, abstinence from all food, rest in bed, and hydrotherapy in the form of alkaline waters, to act as mild diuretics. Water is a very powerful remedy in this disease, and should you ever have to treat many cases of yellow fever, you will be surprised at the number of patients that recover without any other treatment than alkaline waters and good nursing."

Hints upon Treatment of Yellow Fever, based upon those practised by Dr Guiteras of Havanna

"The statistics of the mortality in our hospital show the importance of early treatment in yellow fever: not that we have any specific that must be applied at the beginning of the

attack, but that rest in bed constitutes an important factor in the treatment.

"Excepting the administration of a laxative and the employment of some palliative measure, nothing else has been done here in the first forty-eight hours, or even through the whole attack in mild cases where there was no special indication.

"Our treatment here is published in detail in an article by Major Gorgas, U.S.A., in the *Journal of the Association of Military Surgeons* for October 1903.

"We put the patient to bed at once and make him keep the horizontal position. Clean linen for the bed and person should be furnished plentifully. Windows and doors are kept open (mosquito-screened), only avoiding direct draughts. Quarters should be very clean. Patients seem to do best when treated in tents or newly constructed frame buildings.

"The skin is cleansed and rubbed with hot water and soap on admission. It is not unreasonable to suppose that these measures may lessen the chances of secondary infections, which may be the cause of the hæmorrhagic manifestations.

"Water is given freely through a drinking-tube. From 50 to 80 oz. of fluid are given in the twenty-four hours. Pleasant alkaline waters and infusions and ginger also are given. The patient is not allowed to sit up to empty bladder or rectum.

"Something is done at the same time to relieve suffering, and gain the confidence of the patient. External applications: sinapisms, ice, liniments. If the temperature be very high the surface is sponged with cool water and alcohol, or an enema of cold water is given.

"Phenacetin may be given to relieve pain at night, if necessary once or twice, but should not be insisted upon.

"The first symptom that is likely to call for special treatment is vomiting. If the bowels have not acted since the initial purge, seidlitz powder in broken doses to mix in the stomach, or minute doses of calomel or effervescent magnesia, is given. If the vomiting is persistent, the administration of liquids by the

mouth is discontinued, and only cracked ice is given, and rectal injections of warm physiologic salt solution. If the rectum has not been irritated by frequent purgation this can be kept up with advantage. Milk may be added later.

"Cocaine tablets and ice applied to the throat are used with advantage in vomiting.

"When black specks appear in the vomit, tincture of the chloride of iron, 5 gtt., every two or three hours, is usually given, with a little glycerine and lemon juice. For hæmorrhage from the mouth an antipyrine wash is used.

"The remainder of the treatment reduces itself to sustaining the strength of the patient, and stimulating the action of the heart and kidneys by the judicious use of strychnia, digitalis, and alcohol: the two former by the hypodermic method and the latter by the mouth or rectum. Alcohol is not well borne in large doses. The same may be said of strychnia. Champagne is the best form of alcohol.

"Cold-water rectal injections and calomel as a diuretic are tried in suppression of urine.

"The prolonged typhoid cases, often marked by pronounced jaundice, require alcohol and strychnia. Chlorate of potash is given with apparent advantage; in convalescence muriatic acid and nux vomica.

"Feeding with milk and lime-water is commenced on the third, fourth, and fifth day. The quantity of milk is gradually increased.

"About the ninth day a mild solid diet may be ordered.

"Very mild cases have recovered completely in ten days. Others will require the use of peptonised food, tonics, and massage."

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Mortality Rate

The very high death-rate, nearly 100 per cent., this year, 1910, in the outbreaks of yellow fever in West Africa, was a matter of considerable astonishment and perplexity to observers. But on the contrary such a high death-rate was to be expected, especially in a country where medical men were on the look-out for the typical severe forms of the disease. Naturally in any disease the mortality must be very high if the severe forms form the bulk of those diagnosed. In my opinion this apparent high rate is a positive proof that there were many other cases of mild yellow fever which escaped detection. Had all cases been diagnosed, I am convinced that the rate would have been very much less. Therefore the fact that during this year, 1910, a very large number of the cases died, does not mean that West African yellow fever is worse than in other parts of the world.

Another factor which very materially affects the death-rate in any epidemic is, whether the medical men are expecting the disease or not, whether it bursts on them as it were, or whether they are expecting it. Yellow fever cases carefully nursed from the onset are not so fatal as is usually supposed. Blair's table of mortality rate for the various ages based upon West Indian data is perhaps applicable to West Africa, and I insert it; but it must always be well understood that until the mild forms are recognised no death-rate approaches accuracy:—

Yellow Fever Percentage Death-rates for Different Ages according to Blair

Under 15 years	24·39 per cent.
From 15 to 20 years . . .	23·44 „
„ 20 „ 30 „ . . .	28·45 „
„ 30 „ 40 „ . . .	19·90 „
„ 40 „ 50 „ . . .	21·91 „
„ 50 on	22·22 „

Children usually get the disease in a very mild form, and one not easily recognised.

According to Lazard, in the New Orleans epidemic, out of 430 deaths, the following are the total deaths for various ages :—

1 to 2 years	8	31 to 35 years	36
3 „ 5 „	11	36 „ 40 „	47
6 „ 10 „	16	41 „ 45 „	36
11 „ 15 „	38	46 „ 50 „	23
16 „ 20 „	56	51 „ 60 „	27
21 „ 25 „	56	61 „ 70 „	10
26 „ 30 „	65	71 „ 75 „	1

By some the death-rate in the New Orleans epidemic was estimated at 13·11 per cent.

In the Barbados epidemic of 1909 the death-rate was 42 per cent.

Guiteras states, 1910, that the rate may vary from 4 per cent. to 36 per cent., and that it may go up as high as 92 per cent. The death-rate in one series of 227 cases which Guiteras observed was 22·7 per cent. But, as this observer points out, much depends upon treatment, and especially on the time when the patient was put under treatment. Thus in the experience of the Las Animas Hospital at Havanna, the death-rate amongst those admitted in the first forty-eight hours was 17·7 per cent., and in those later 32·3 per cent. Everything therefore depends upon careful treatment from the outset.

In the section devoted to race susceptibility will be found further death-rates, arranged according to nationality and race and length of residence in the Colony. It is a very old observation that recent arrivals were more prone to yellow fever than those who had been resident some time. It has also often been observed that whilst the negro race is not immune to yellow fever, as is well shown in the preceding pages on symptomatology in different parts of the world, nevertheless the mortality rate and the severity of the symptoms are less than in the case of the white race.

PART III

PATHOLOGY

CHAPTER XIII

EXPERIMENTAL PATHOLOGY

THE exact pathology of yellow fever must remain obscure until the virus has been isolated. In spite of this great disadvantage, however, certain facts have come to light, both as the result of very careful observation in cases of yellow fever, and by conducting experiments in men and in mosquitos, which have indicated the mode of action of the virus, and have in addition placed in our hands a specific means of combating the disease.

The Role of the "Stegomyia"

Beauperthuy drew attention from 1850-1860 in no uncertain manner to the fact that his long and patient observations had led him to conclude that the agent which propagated yellow fever was the *Stegomyia* or "house-haunting mosquito," and not emanations from the soil. In the second place, Finlay of Havanna came to the same conclusion in 1881, and succeeded in giving a mild form of yellow fever to susceptible persons by the bite of the infected *Stegomyia*. Indeed he both proposed and used this method as a protective measure against the severe form of the disease. There is no doubt that Beauperthuy in British Guiana was one of the first to announce that yellow fever was propagated by the "house-haunting mosquito." It is equally true that Finlay was the first to make direct experiments to prove this, and I therefore insert the following statement, made by Dr H. E. Durham, who, with the late Dr

Walter Myers, were sent out by the Liverpool School of Tropical Medicine in 1900 to study yellow fever in Brazil.

Dr Durham wrote in his report to the school:—

"It is incontestible that Dr Charles Finlay, of Havanna, was the first to undertake direct experiments to substantiate his ideas of the part played by the mosquito in the transmission of yellow fever. His method was to feed mosquitos upon yellow fever patients (not later than the sixth day), and then after an interval of from forty-eight hours to four or five days to allow them to feed upon susceptible persons; the idea was to produce a slight attack of the fever in order to produce immunity.

"At a delightful chat we had with the courteous doctor, on 25th July 1900, he told us many details concerning his experiments, which were commenced so long ago as 1881. Altogether, 102 persons had been tried in this manner, and in 17 instances some pathogenic effect had followed the bite; this consisted in slight fever appearing about the fifth, sometimes as late as the fourteenth day.

"In no instance was there a definite attack of yellow fever as the result, but Dr Finlay thought that a certain immunity had been conferred, since only four of these persons died of yellow fever, though the cases were followed out to ascertain their after history, in some cases for four years.

"Naturally it was not possible to exclude intercurrent infections by thus working in an endemic centre, still the mode and kind of experiment which has since led to more definite results was laid down.

"The kind of mosquito used by Dr Finlay was the *Stegomyia fasciata* (it was referred to in his papers as *Culex mosquito*); he selected this kind on account of its town-dwelling habits."

Fomites, cadaver, and excreta, not infectious.—After many years of intense and bitter controversy, it became universally recognised that yellow fever was not contagious from man to man. That the clothes and bedding no matter how contaminated by the patient, were non-infectious. That the exhalations of the patient, the vomit, excreta, and the mucous discharges

from the conjunctiva, were non-infectious. Numerous direct experiments had been tried on man with negative results (Mott, Blair, and others).

Extrinsic incubation period (*Carter and Reed*).—The above observations, together with a close investigation of the dates upon which subsequent cases of infection followed the first ones, pointed to the virus, or infection residing in some agent other than man. Harrison and Moxley enunciated this opinion in Barbados, and Carter of the United States reasoned that the agent must be the mosquito (see epidemiology).

Thus by a process of exclusion, direct observation and epidemiological considerations, men's minds were directed to the mosquito as the probable agent, and it was of course enormously strengthened by Ross's observations upon the Anophelines. We now know that the virus of yellow fever requires an incubation period of about twelve days in the *Stegomyia*, before the latter is capable of communicating the virus to a susceptible person.

This period is known as the *extrinsic incubation period.* Once the *Stegomyia* is infected, it remains so for a very considerable period. Three months has been stated as the result of observation, but the maximum limit has not yet been ascertained. For practical purposes it is sufficient to know that the *Stegomyia* infected in the autumn of one year can carry over infection into the following year, and this explains the often repeated observation, that yellow fever is exceedingly liable to break out several months after an epidemic was supposed to have died out.

Direct inoculation experiments.—The ground having been prepared in the manner described above, it was left to four United States Marine Hospital Service medical men to see if it was possible to infect man with *Stegomyiæ* which had fed on a yellow fever patient. The results of these inoculation experiments are detailed under experimental yellow fever (p. 100). They proved conclusively that the *Stegomyia* could transmit the

virus of yellow fever. A few experiments with other species of mosquito were negative, but were not, however, carried out upon a sufficiently large scale to make absolutely positive one way or the other.

Hereditary transmission of the yellow fever virus in the mosquito.—Several experiments have been made to determine whether the adult infected *Stegomyia* can transmit infection to the ova and larva as happens in the case of the tick, as shown by Dutton and Todd.

The French Commission under Marchoux obtained one positive result. On the other hand, Guiteras, Reed, Carroll, Agramonte, and Rosenau obtained uniform negative results. The question is therefore still open and is worthy of further research.

Intrinsic incubation period.—From the above experiments, it was demonstrated that the incubation period in man was at least six days, a period which coincided with the results of direct clinical observation.

The further very remarkable fact was also proved, namely, that the duration of infectivity in man was very short, limited apparently to the first three days of illness, during which time only the virus appears to be present in the circulation, for after the third day the patient ceases to be infectious.[1] We know, in support of these direct experimental observations, that the making of post-mortem examinations upon yellow fever cases is unattended with risk of infection. I have myself made very many, and did not contract the disease.

Blood inoculation experiments.—A series of direct inoculations into non-immunes was made by Reed, Carroll, and Agramonte, from the blood of patients suffering from yellow fever during the first three days of illness. In each instance there resulted an

[1] It is probably true that the statement that the virus is limited to the first three days of the disease is too dogmatic. All that we can state positively is that the virus is present during the first three days; but a sufficient number of experiments have not been made to exclude the presence of the virus on subsequent days.

attack of fever as detailed in the preceding pages. These experiments proved, like the *Stegomyia* experiments, that the virus existed in the blood during the first three days of the attack.

Experiments were also made to make certain that the disease induced by inoculation was not merely a toxæmia, but the result of the multiplication of the virus in the bodies of the inoculated; for this purpose *Stegomyia* were fed on the blood of those inoculated, and it was shown that *Stegomyia* so infected could transfer the virus to the non-immune. In the same way, it was proved that the blood of those inoculated contained the virus, and that the blood when reinoculated into a non-immune could produce the disease.

Lastly, experiments were made to ascertain whether the virus was capable of passing through a Berkefield or Chamberland filter, and it was found that it could pass, because the filtrate could give rise to an attack of yellow fever when inoculated into a non-immune. This same fact has been observed in the case of certain other diseases. It has also been shown that the yellow fever virus is very sensitive to heating and that it is readily killed.

These various experiments have started numerous investigations to try and isolate or demonstrate the virus, but hitherto without a very definite result.

The virus.—Before the Reed-Carroll experiments already alluded to, Sanarelli and others had maintained that the virus was a bacillus which could be isolated in all cases of yellow fever. This was, however, disproved, and in any case it could not explain the period of incubation in the *Stegomyia*, for it was obvious that the transference of the yellow fever virus by the *Stegomyia* was not mechanical as Finlay himself once thought.

It is reasonable to suppose that whilst in the body of the mosquito, the virus is undergoing some change. Yet what that change may be, whether like that described by Ross, in the case of the malaria protozoon in the anopheles, is not yet known.

Numerous attempts to demonstrate an organised virus in

the salivary glands of the *Stegomyia* have absolutely failed. Microscopical and cultural analyses of the tissues and blood of infected man have also so far yielded mostly negative results.

Blood examination.—Otto and Neumann made a very long series of observations of the blood of yellow fever patients using the ultramicroscopic method, and found very minute particles both in the blood and cerebro-spinal fluid, but they attached no importance to their presence.

More recently Seidelin has recorded the presence, in some cases of yellow fever, of minute intracorpuscular bodies having a ring or an amœboid form, and pigmented. He also describes some free forms in the blood, and has later demonstrated the presence of intracorpuscular and intracellular elements, in sections of organs, especially in the kidneys.

Schaudinn surmised that the cause of yellow fever might be found to be a spirochæte, and recently Stimson has described a spirochæte in the tubules of a kidney from a case of yellow fever.

At one time many observers came to the conclusion that the virus was bacterial. The *B. icteroides* of Sanarelli, the bacillus of Sternberg, attracted considerable attention at the hands of bacteriologists. Durham and Myers found an influenza-like bacillus in the cases of yellow fever which they examined. Careful detailed analysis has, however, shown that none of these bacteria can be regarded as the causative agents. They are mere epiphenomena, similar to the numerous varieties of higher fungi, which still earlier observers had demonstrated in cases of yellow fever.

Blood counts.—According to Durham and other observers, there is a marked leucopenia in the final stages of yellow fever. At the same time Durham points out that there is an increase in the leucocytes, in the capillaries, in the internal organs. According to Sodré and Couto, hypoleucocytosis is most marked in the severer forms of yellow fever, and less so in the milder. The polynuclear leucocytes are relatively increased.

Experiments upon animals.—This promising line of research

has been taken up by Dr W. Thomas in Brazil, who is employing anthropoids and other animals.

It is an old observation, that in yellow fever epidemics, domestic animals have appeared to suffer. Blair instances the case of a dog and of supposed cases in fowls.

W. Thomas succeeded in obtaining a reaction in guinea pigs four and a half to thirteen days after being bitten by infected *Stegomyia*. The symptoms consisted in a rise of temperature lasting two to three days, and dulness. He states that after the reaction the animals were immune to subsequent inoculation, and that during the illness their blood was capable of infecting *Stegomyia*. In the case of the chimpanzee a reaction was obtained five days after infection by the bite of an infected *Stegomyia*. These experiments require to be continued and repeated on a large scale.

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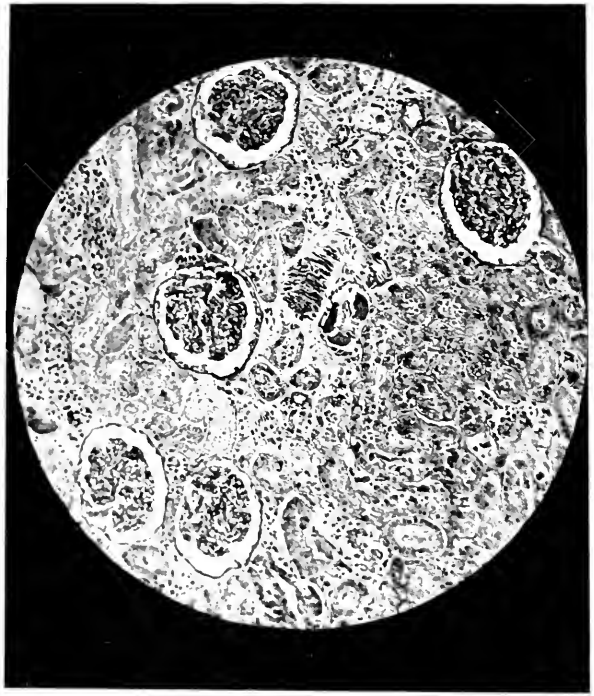

FIG. 20.—Section of Kidney from a case of yellow fever in West African native. Patient brought in dead. Kidneys congested. Note dilatation of Bowman's capsule. Stained Hæmatoxylin. (Mag. 200 diam.)

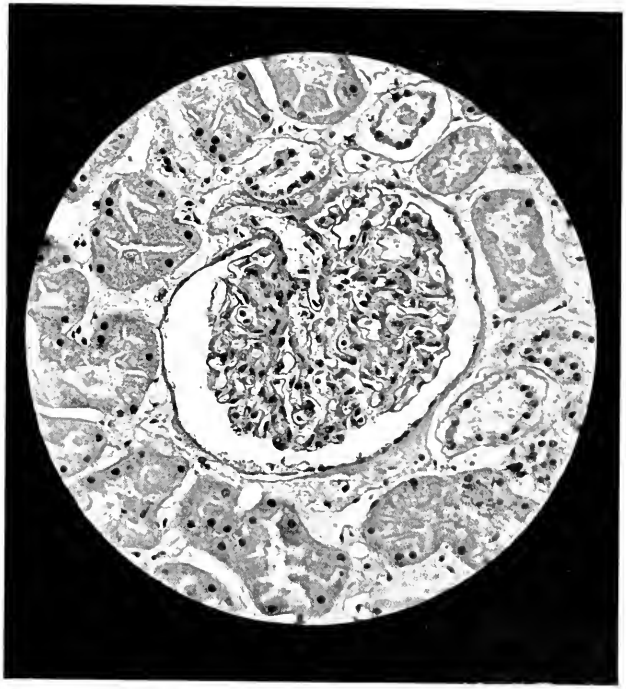

FIG. 21.—Section of Kidney from a case of yellow fever in West African native. Note marked dilatation of Bowman's capsule. Stained Hæmatoxylin. (Mag. 500 diam.)

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CHAPTER XIV

MORBID ANATOMY AND HISTOLOGY

Pathological Anatomy and Histology

THE appearances of the grosser and finer lesions in yellow fever are characteristic, as characteristic as the symptoms in a well-marked severe case of yellow fever. Should there be any doubt about the diagnosis of yellow fever during life, there ought to be no doubt after making a post-mortem examination.

The naked eye appearances of the body and viscera are such as cannot readily be mistaken for any other disease.

The skin is yellow, often appearing much more yellow than during life; there are large livid patches especially in dependant parts, but not limited to them; subcutaneous ecchymoses are frequently present; there may be also evidence of oozing of blood from the nose, mouth, conjunctivæ, or anus.

On making an incision into the body there is no sign of wasting; the body is often well nourished, abundant subcutaneous fat, stained yellow; the muscles appear normal. This simply means that the fever is as a rule so acute that there is no time for degeneration reactions to set in. The first visceral system to be carefully examined should be:—

The alimentary tract.—This system invariably exhibits some degree of congestion; very frequently the congestion is intense, especially in the stomach.

It has been frequently observed that although no black vomit may have taken place during life, yet after death the stomach may contain a large quantity of it. This fact was

noted by Dr Chichester in the 1900 yellow fever outbreak at Bathurst in West Africa, and has been many times recorded by other observers.

The gastric mucous membrane has a purple or hæmorrhagic appearance, most marked, according to some observers, towards the cardiac end. There is invariably present in the stomach some black vomit, or mucus, or watery serous fluid ("white vomit") tinged with blood.

The *intestines* usually contain dark tarry material. According to Blair's table of ninety-seven post-mortem findings, the œsophagus, towards the cardiac end, is deeply congested. In one of the autopsies which I made in New Orleans in 1905, I observed the same cardiac congestion. In Blair's table the stomach contained black vomit in seventy-nine cases out of ninety-seven, and in all cases it was congested. The œsophagus was congested in a very large proportion of the cases. The duodenum, the small and large intestines were congested in about one-third or more of the total cases; in many instances the contents were tarry.

Enlargement and congestion of the *mesenteric lymphatic glands* has been observed, but more usually they appear normal. Durham and Myers have described an enlargement of the lymphatic glands in a considerable proportion of their cases.

After the alimentary tract in importance comes the liver.

The *liver* is invariably altered in colour; most frequently it presents some shade of yellow, usually *boxwood* colour, but shades like bath-brick, tan, ochreous brown, deep yellow, pale yellow, reddish yellow are frequently recorded. The fact being that there is *some shade of yellow* with, in addition, some degree of *congestion*, the latter may be so pronounced that the liver looks like a yellow "nutmeg liver." Most observers agree that the term boxwood covers most accurately the shade of yellow which is most frequently seen. It is not a bright yellow colour such as is sometimes seen in jaundice. The neck of the gall bladder may be congested; this was the case in the

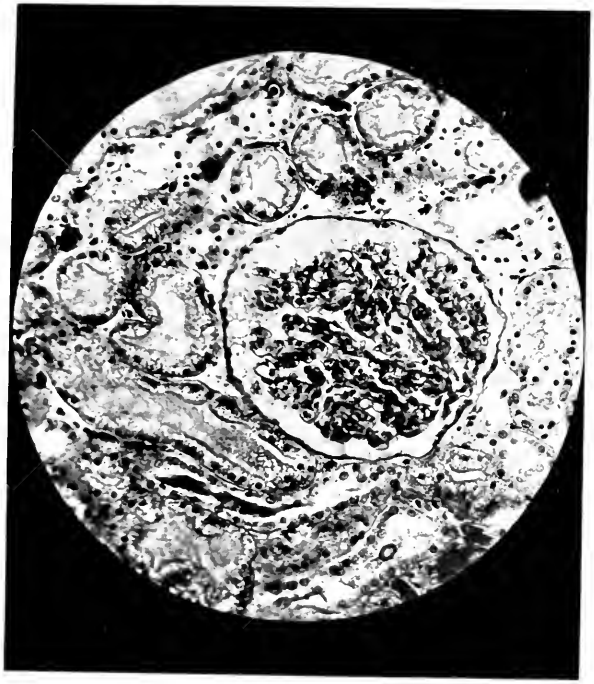

FIG. 22.—Section of Kidney from a case of yellow fever in West African native. Bowman's capsule and many of the convoluted tubules contain coagulum. Stained Hæmatoxylin. (Mag. 500.)

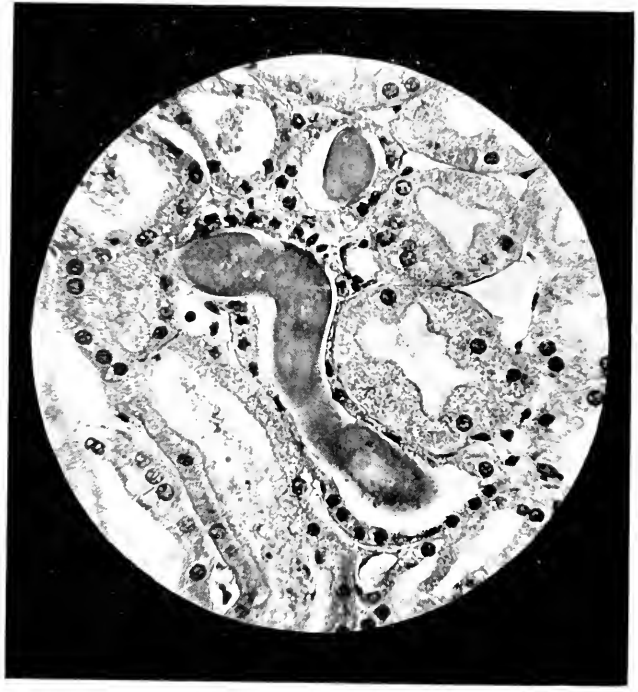

FIG. 23.—Section of Kidney from a severe case of yellow fever in a West Indian soldier. Note casts in tubules. Stained Hæmatoxylin (Mag. 500 diam.)

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autopsies made in Secondee this year, 1910. Next in importance come the kidneys.

The *kidneys* show some degree of *congestion*. They may be pale, fatty, or fibroid, but in addition the vessels are as a rule congested, especially in the pyramidal region. The bladder should be examined for the presence of urine. For if it were not possible to test the urine during life for the presence of albumin, the opportunity should be taken to do so, if any urine happens to be found in the bladder.

Vascular system.—The noticeable feature in connection with the vascular system is the general congestion of the vessels. The heart is very often flabby, but on the other hand it may present remarkably little change.

In six post-mortems which I made in New Orleans, small ecchymoses were present on the external and internal surfaces. Petechiæ or serous surfaces generally are very frequently met with in yellow fever.

Brain and spinal cord.—I found the brain intensely congested in one of my autopsies, and the condition has been often found.

Lungs.—As a rule there is no fluid in the pleural cavities; they are usually dry. There may, however, be considerable congestion of the lower lobes of the lung.

Spleen.—The spleen is not as a rule noticeably altered; it is usually normal in size.

The naked eye changes therefore point to the concentration of the pathological changes in the vessels, stomach, intestines, liver, and kidneys.

Notes of Autopsies made on Cases of Yellow Fever

Case 167, *Post-mortem No.* 8, New Orleans, 1905 :—

26*th August* 1905.—Skin yellow; spleen enlarged and dark, 6½ oz.; liver enlarged, yellow red colour, not a bright yellow; kidneys congested; heart, ecchymoses on surface; muscle flabby and soft; some bile-stained fluid in pericardium; stomach

contained typical black contents; ecchymoses in mucous membrane; patient died on fifth day of illness.

Case 162, *Post-mortem No.* 7, New Orleans, 1905:—

Skin jaundiced; spleen 11¾ oz., slightly yellow in colour; liver very typical boxwood colour; pleural cavity dry; heart flabby, pale, ecchymoses present; kidneys fatty; patient died seventh day of illness.

Case 180, *Post-mortem No.* 9, New Orleans, 1905:—

Skin markedly yellow; muscles dark red; small amount of fluid present in pleural cavity; spleen large, slaty in colour; liver small, yellow, granular, congested; kidneys large, fatty, ecchymosed; pericardium contains small amount of fluid; heart muscle pale, ecchymosed; lungs hypostatic congestion; mesenteric glands enlarged; stomach contains black vomit; cardiac end of stomach deeply congested; patient died fifth day of illness.

Case 157, *Post-mortem No.* 11, New Orleans, 1905:—

Lungs, pleural cavity dry; no hypostatic congestion; liver boxwood colour, slightly congested; kidneys normal in size, slightly fibroid; spleen 7½ oz., dark and firm; heart, ecchymoses on surface; liver boxwood colour, cirrhotic; brain œdematous, deeply congested; patient died eighth day.

Case 130, *Post-mortem No.* 12, New Orleans, 1905:—

Boy, æt. twelve, mesenteric glands enlarged; some fluid in pleural cavity; left lung deeply congested; slight excess of fluid in pericardium, bile-stained; ecchymoses on surface of heart; spleen firm, 4 oz., deep slate colour; kidneys normal size, congested; liver 43½ oz., of a very typical tan colour; pancreas congested at base; died twelfth day.

Post-mortem No. 13, New Orleans, 1905:—

Male, æt. twenty; brain deeply congested; lungs deeply congested at base; heart, ecchymoses on surface; liver yellow and congested; kidneys fatty and congested; patient died sixth day.

In 1909 I made two post-mortem examinations upon cases in Barbados. In one case the liver was congested, but the

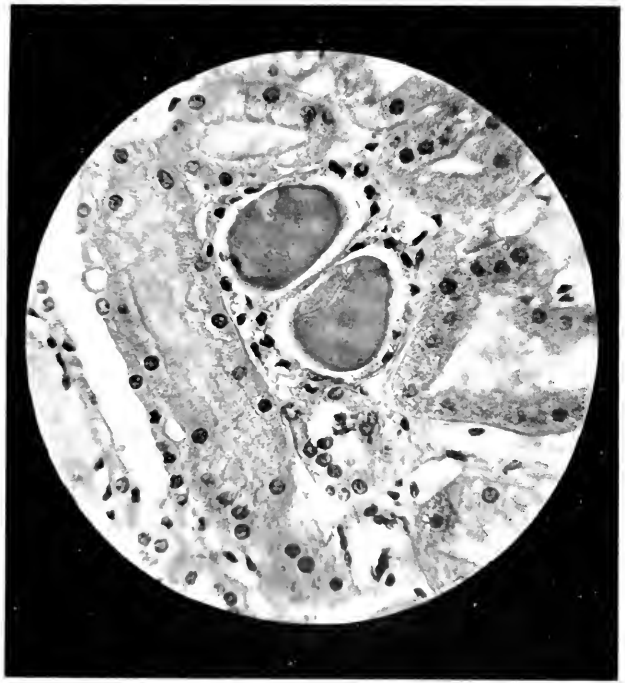

FIG. 24.—Section of Kidney from a severe case of yellow fever in West Indian soldier. Observe casts cut transversely in the tubules. Stained Hæmatoxylin. (Mag. 500 diam.)

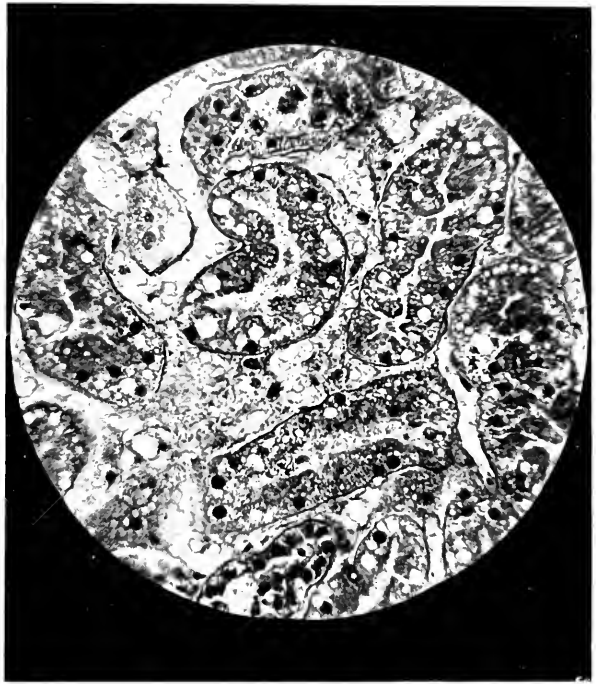

FIG. 25.—Section of Kidney from a severe case of yellow fever in a white man. The urine was scanty and highly albuminous. Observe the marked fatty degeneration of the convoluted tubules and congested capillaries. Stained Hæmatoxylin. (Mag. 500 times.)

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typical boxwood colour was not seen until the liver was cut into. The kidneys were congested, and the stomach contained dark bloody fluid ; the mucous membrane was deeply congested ; the pericardial fluid was slightly bile-stained, and the wall of the left ventricle was soft. In another case the liver had the characteristic yellow colour.

In the 1910 West African cases the liver invariably presented some shade of yellow colour ; in some cases it was obscured by the presence of congestion, so that the liver resembled the nutmeg type. Congestion of the gall bladder was a feature of some of the cases at Secondee. The kidneys and stomach were also congested.

Microscopic Anatomy

Liver.—In my experience the microscopic appearances are very constant, but they are not peculiar to yellow fever. Closely identical changes may be seen in cases of phosphorus poisoning, and in other diseases accompanied by rapid degeneration of the liver substance. The degeneration changes are most marked in the hepatic zone of the liver lobule. The capillaries in this zone are congested, exactly as in the nutmeg liver of venous congestion the liver cells between the capillaries have a dislocated, disorganised appearance, with complete loss of their outlines.

The cell substance has often a very characteristic vacuolated or sponge-like appearance, and stained with osmic acid it is found to be loaded with fat droplets, some very minute. The cell is, in fact, reduced to a fatty spongework around the nucleus. In these cases the appearance of the cells more closely resembles that seen in phosphorus poisoning than in any other disease with which I have had practical acquaintance.

There is slight but distinct change in the portal zone of the lobule ; there also appears evidence of cell proliferation, and there is slight leucocytic infiltration. But these changes are by no means so pronounced as in acute yellow atrophy, at least in my experience. The mucous membrane of the gall bladder on section may show considerable injection of the capillaries.

Kidneys.—The capillaries in the region of the straight and collecting tubules may be considerably dilated and filled with blood clot, and there may be evidence of capillary congestion throughout the kidney and in the glomeruli. The congestion is, however, most marked in the pyramidal portion of the kidney. In the parenchyma I have frequently observed *dilatation*[1] *of Bowman's capsule*, and often the presence of a coagulum inside the capsule. The epithelium of the convoluted tubules is vacuolated and contains fat droplets, and the cell substance is granular and the contours of the cells are ill defined. The appearance of the cells is very similar to that seen in phosphorus poisoning.

In the tubules hyaline casts are very common in my experience, and this fact has also been observed by Otto and Neumann, Durham, and other observers. Some of the casts may contain granular material and red corpuscles. There is an absence of leucocytic infiltration. The microscopic appearances are not those found in cases of blackwater fever where the collecting tubules contain pigment.

Stomach.—The microscopic appearance of the wall of the stomach corroborates the naked eye appearance, and simply shows dilatation of the capillary loops.

Lungs.—Microscopic sections show only evidence of congestion; there is no leucocytic infiltration.

Brain.—Apart from the congestion of the vessels, I have not observed any changes in the grey substance of the brain.

The pathology of yellow fever.—As stated in the commencement, it is very difficult without knowledge of the virus to explain the symptoms or tissue changes in yellow fever.

There is no doubt that fatty degeneration of the liver and kidney cells is a very constant change. To what is this change due? It is due to a specific poison, which, like phosphorus, acts

[1] Durham lays stress upon the dilatation of the capsules, and considers that the suppression of urine is probably due to blockage of the tubules by casts.

FIG. 26.—Section of Kidney in the region of the vasa recta, to show great congestion of the vessels. Stained Hæmatoxylin and Eosin. (Mag. 200 diam.)

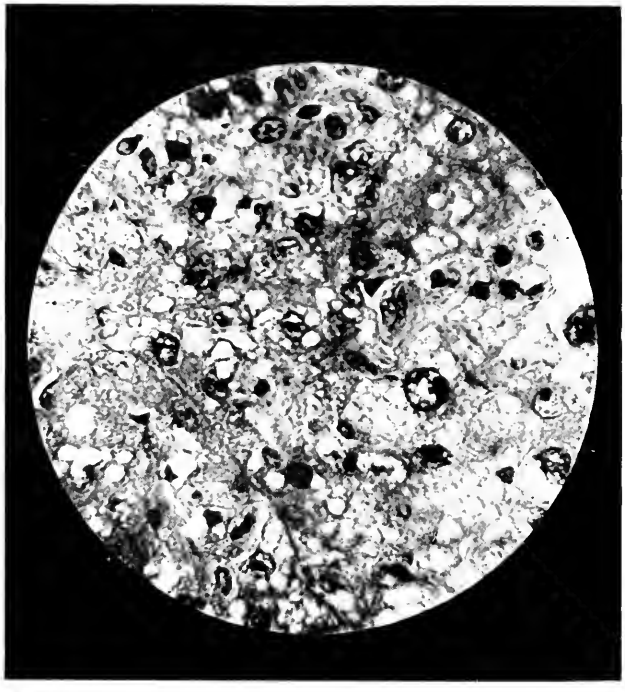

FIG. 27.—Section of the Liver from a case of yellow fever in West Indian soldier. Early fatty degeneration of the liver cells. Stained Hæmatoxylin. (Mag. 500 diam.)

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on the parenchyma; or is it merely a secondary phenomenon supervening in the liver and kidney as the result of bacterial invasion during the process of death? In my opinion the evidence favours the changes being induced by a toxæmic condition of the blood.

There must be a profound change in the vessel walls to produce the petechiæ, hæmorrhages, and black vomit which are so characteristic.

With regard to the mechanism of the jaundice we can say very little. It is often too readily assumed that the bile canaliculi are blocked by the swollen cells. The jaundice which is present is probably like that seen in other toxæmias and is "hæmohepatogenous," that is, there is hæmolysis of the blood corpuscles as well as blocking of the bile ducts by the thickened and altered bile, with the result that the bile-colouring matter and salts are absorbed and not excreted. To what is due the suppression of urine? Some, like Durham, think that as in blackwater the tubules become blocked; as mentioned above, the histological changes in the kidney would certainly favour this view.

The slowing of the pulse might be accounted for by the absorption of bile salts induced by the disorganisation of the liver.

The Urine Reactions

Ehrlich's diazo reaction.—Durham and Myers made a series of observations, and concluded that this test was of no value for yellow fever. Seidelin examined for it in 22 cases, and found it positive in about half the cases. He does not lay any stress upon the reaction from the point of view of diagnosis. Albertini made an examination of 142 cases, and found the reaction negative in 130 cases, and concluded that the reaction was not given in simple cases of yellow fever.

Durham, in common with all observers, lays great stress upon the presence of casts in the urine, and states that it may

be possible to find casts before albumin has appeared in the urine.

Durham also found both in his own case and in other cases of yellow fever that picric acid and heat were the most reliable agents to use to demonstrate the presence of albumin. The amount of albumin in the urine may be so great that a solid coagulum is obtained in the test-tube on boiling. All observers are agreed that the urine should be watched and tested very frequently from the commencement of illness.

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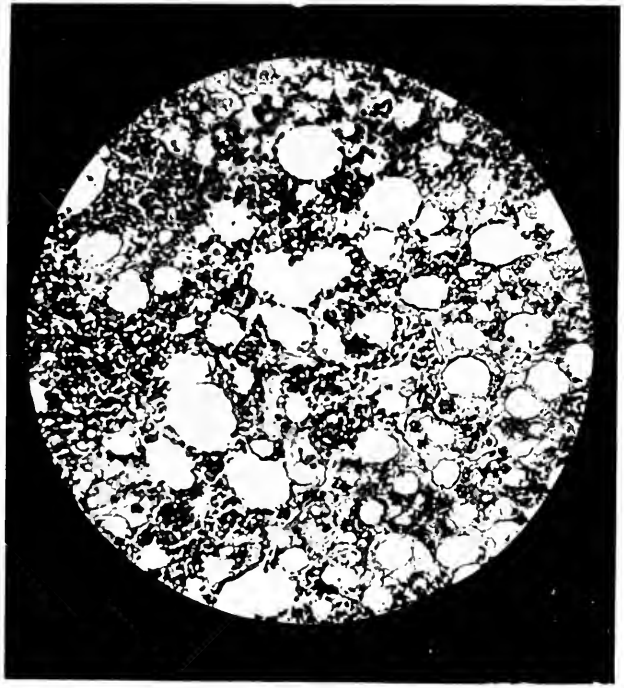

FIG. 28.—Section of Liver in advanced stage of fatty degeneration from a yellow fever case, Amazon. Stained Hæmatoxylin. (Mag. 500 diam.)

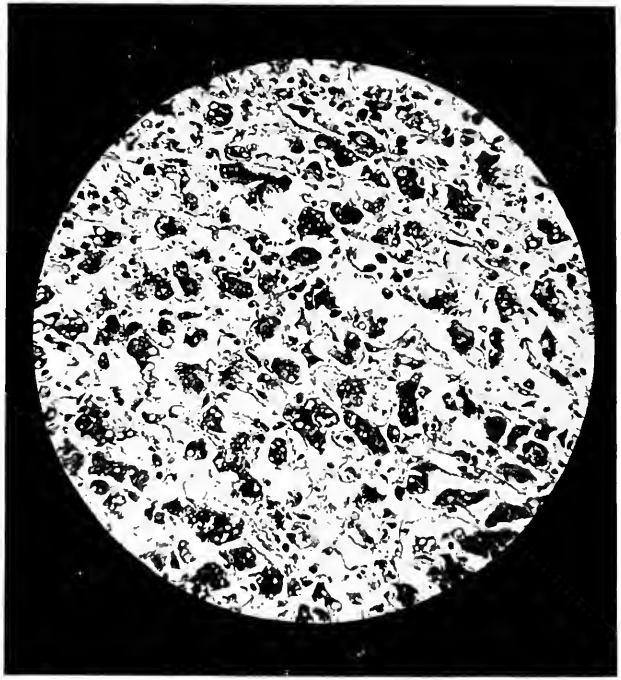

FIG 29—Section of Liver showing dislocation and fine fatty degeneration of liver cells, from case of yellow fever in native West African. Stained Hæmatoxylin. (Mag. 500 diam.)

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PART IV

EPIDEMIOLOGY

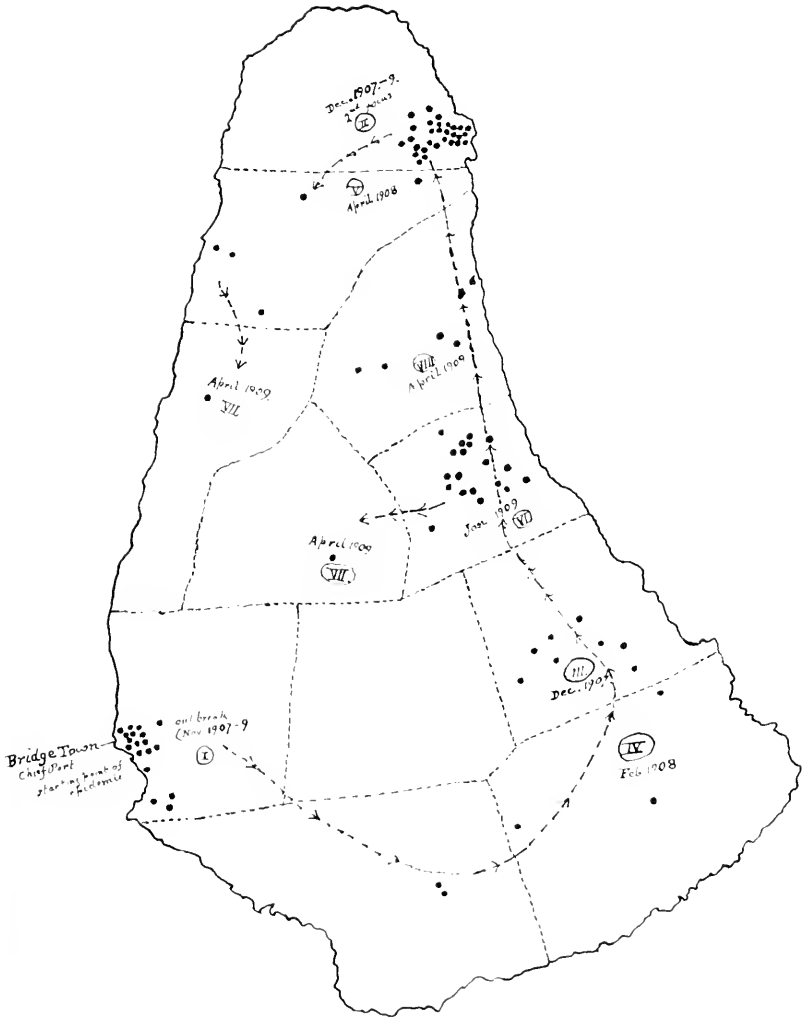

FIG. 30.—Plan of the Island of Barbados. The dotted lines indicate the parishes. The black dots indicate the cases of yellow fever, and the arrows the probable paths of infection. Note the primary focus in Bridgetown, and the scattered secondary foci.

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CHAPTER XV

EPIDEMIOLOGY

I.—*Mode of Diffusion*

SURGEON-GENERAL BLAIR, in describing the spread of infection in yellow fever, wrote significantly as follows:—

Its shifting lines of infection and gyratory movements suggest to the imagination the attributes of insect life.

This great observer was far nearer the truth than he imagined. The same characteristic phenomenon can still be observed. It was notably manifest in Barbados during the years 1907, 1908, and 1909, where, as the spot map shows, there were a series of small outbursts throughout the island.

The first outbreak was in the chief town, Bridgetown, at the end of the year 1907. In the beginning of 1908 scattered cases appeared in two other foci in the island, and later other centres made their appearance, and small outbursts of 18 and 29 cases respectively were registered. These scattered outbursts might have been due to two causes—first, the spread of infection from the original focus in Bridgetown in 1907, either by the transport of infected *Stegomyia* or, what was more probable, the migration of mild cases of the disease from the centre in Bridgetown to the outlying country districts.

A second alternative, however, might have been argued, namely, that the secondary foci originated *de novo*, that is, that the disease was endemic in the island, and that the outbreaks were recrudescences of an existing disease.

In my opinion there were weighty reasons against the

endemic view, chief amongst them the fact that the natives were those who principally suffered, which one would not expect if the disease were endemic amongst them. I therefore favoured importation, and the view that the original focus of infection was Bridgetown, from which the disease then jumped, as it were, to other centres throughout the island, following the lines of most frequent intercourse.

To return to Bridgetown. Early in 1908 there was a case of yellow fever notified, then a lull occurred until later in the year, when a fresh outburst apparently took place. I say apparently, because it is very probable that in reality there was not this gap, and that cases occurred which were not recognised.

A recrudescence also followed in the outlying villages.

The total number of cases in the epidemic was approximately 100. The disease commenced in the chief town, Bridgetown, in 1907, spread slowly to the villages scattered throughout the island, dying out in one centre to appear afresh in another; but for the most part appearing to remain quiescent and limited to a few cases until 1909, when the disease appeared to burst out afresh.

In my opinion this is only apparent, and due to the fact that the connecting cases passed unrecognised. The great interest of the Barbados epidemic centred in the large number of native black residents who were affected; more than half the cases were blacks, and there was a high death-rate. Another interesting circumstance was the progress of the disease in the various centres in Barbados. In the chief town, Bridgetown, the disease started in a particular infected quarter of the town, where the *Stegomyia* was very abundant, and where there was overcrowding. It was, moreover, the locality frequented by sailors. The outbreak was, however, attacked by prompt methods, such as isolation, fumigation, and destruction of larvæ, with the result that the outbreak ceased in April 1909.

In the outlying villages, however, the difficulty of complete fumigation of infected houses was found to be very great, and

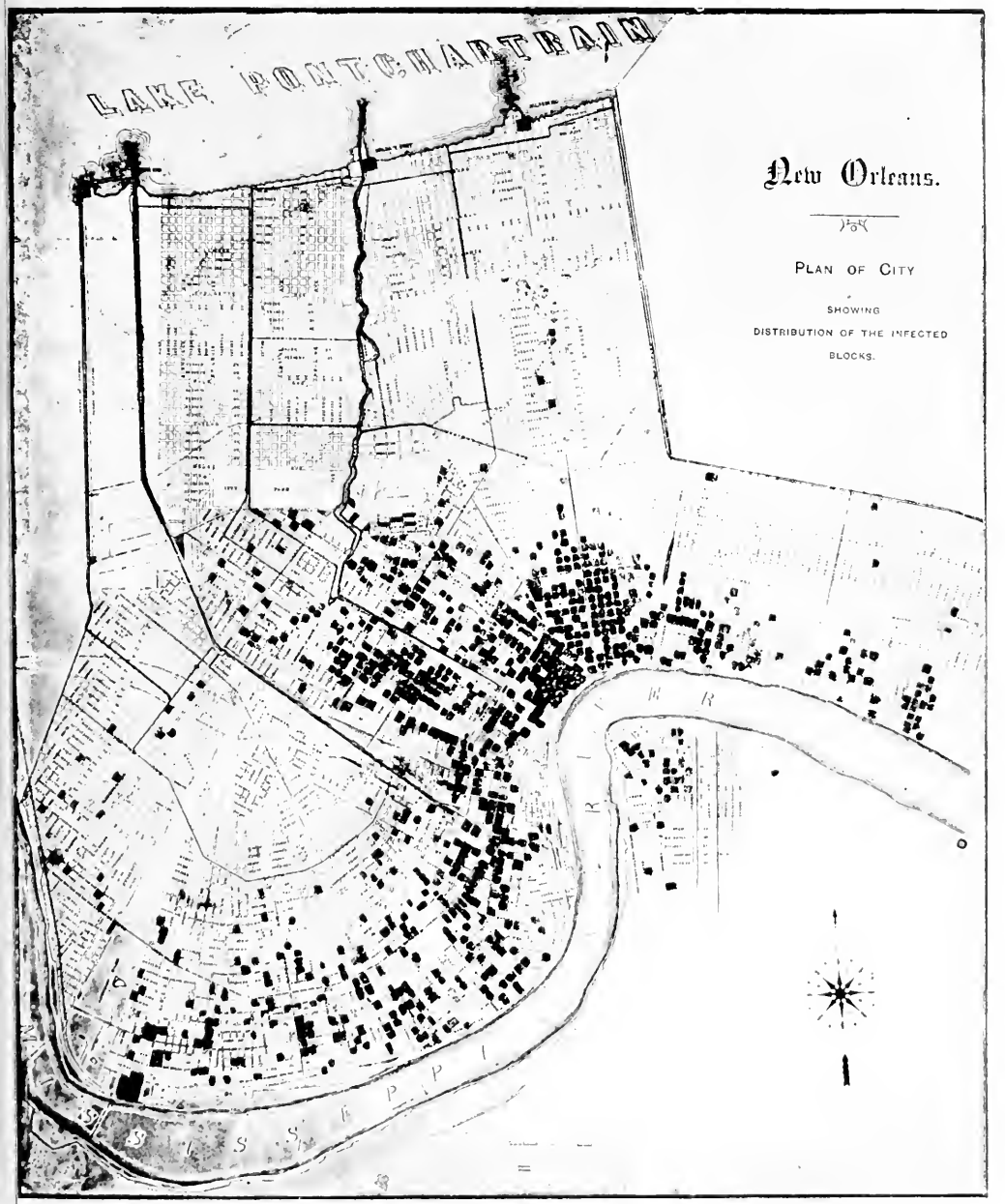

FIG. 31.—Spot Map of Yellow Fever Cases in New Orleans 1905 Epidemic. Observe that the infection is concentrated in one particular area, viz., the "old town," or labouring quarter, the quarter which, in the time of Faget, was the endemic focus of yellow fever.

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there is every reason to suppose that a considerable number of infected *Stegomyia* escaped destruction. The result was that sporadic cases of yellow fever lasted well into the autumn of 1909. This was the usual course of the disease in the days before complete fumigation of houses in which cases of yellow fever had occurred. The contrast of the progress of the New Orleans 1905 epidemic to the above is very complete.

New Orleans furnishes an example of an outbreak in a very large city of 239,000 inhabitants. As the plan shows, the disease centres in one particular part of the town, as in Barbados—the poor, overcrowded quarter, where the imported labouring classes resided. In this area the *Stegomyia* was breeding in immense numbers. Therefore there existed in New Orleans the two essential conditions for an outbreak of yellow fever: one, a very large non-immune population, and secondly, a very large number of *Stegomyia*. When, therefore, yellow fever was introduced, as was supposed, by a ship arriving from Central America, the conditions were favourable for an outbreak. It was, moreover, the hot season of the year.

Yellow fever was declared 22nd July 1905, but here, as elsewhere, there can be no doubt that the early cases escaped detection. On 12th August, 105 cases had been declared—this was the maximum point. Analysis of the chart shows a rapid rise to this point. In the first week in August, however, most energetic prophylactic measures had been set on foot, and no less than 70,000 cisterns had been screened, in addition to a vast number of houses fumigated, and the immediate isolation of any case of disease presenting the least suspicious symptoms.

The effect of this is seen in the curve. The vast bulk of infected *Stegomyia* were destroyed by the thorough sulphur fumigation; the supply of fresh ones had been completely cut off by the wholesale screening of cisterns, the removal of useless receptacles, and the abundant use of petroleum; and finally, all possible human carriers having been carefully screened and watched, there was no possibility of the disease gaining head-

way, and it had to fall back. There is, therefore, a steady fall during the autumn months, interrupted here and there by a sporadic case or two, but no outburst.

The most striking evidence of the extraordinary difference of the results of the prophylactic measures adopted in this campaign as compared with previous epidemics in New Orleans is furnished by comparison with the number of cases on corresponding dates in the previous epidemics. Thus, in the epidemic of 1898, 2 cases are reported on 24th July; on 12th August, there are 31 new cases; on the 14th, the new cases were 134; steady increase takes place, and on 31st August the new cases are 234. Increase still takes place, and this, too, in spite of the adoption of all the methods then known to science, and which consisted in most rigid house quarantine; towards the end of September there is slight decrease, but in the first week in October as many as 305 new cases are reported on the 4th; the figures then slowly decrease to an average of about 60 new cases in the first week in November, and after that the frost brings the fever to a close. The total number of cases amounted to 13,817, with 3984 deaths, as compared with 3384 and 443 deaths in 1905.

It would be impossible to obtain more striking figures of the thorough control which the organised medical forces had over the fever by the end of the first week in August, and solely by adopting one line of attack—the destruction of the *Stegomyia fasciata* in the houses, prevention of their breeding in the water cisterns, and early recognition and screening of the cases.

In Belize, British Honduras, the small outbreak which I was called upon to investigate, showed the same history of want of recognition of the early cases, and in consequence the "bolt from the blue" origin of the disease as it is often termed. In reality it is nothing of the kind, had one the means of diagnosing the early and mild cases. The so-called "bolt from the blue" is only the sudden appearance of what seems to be an isolated

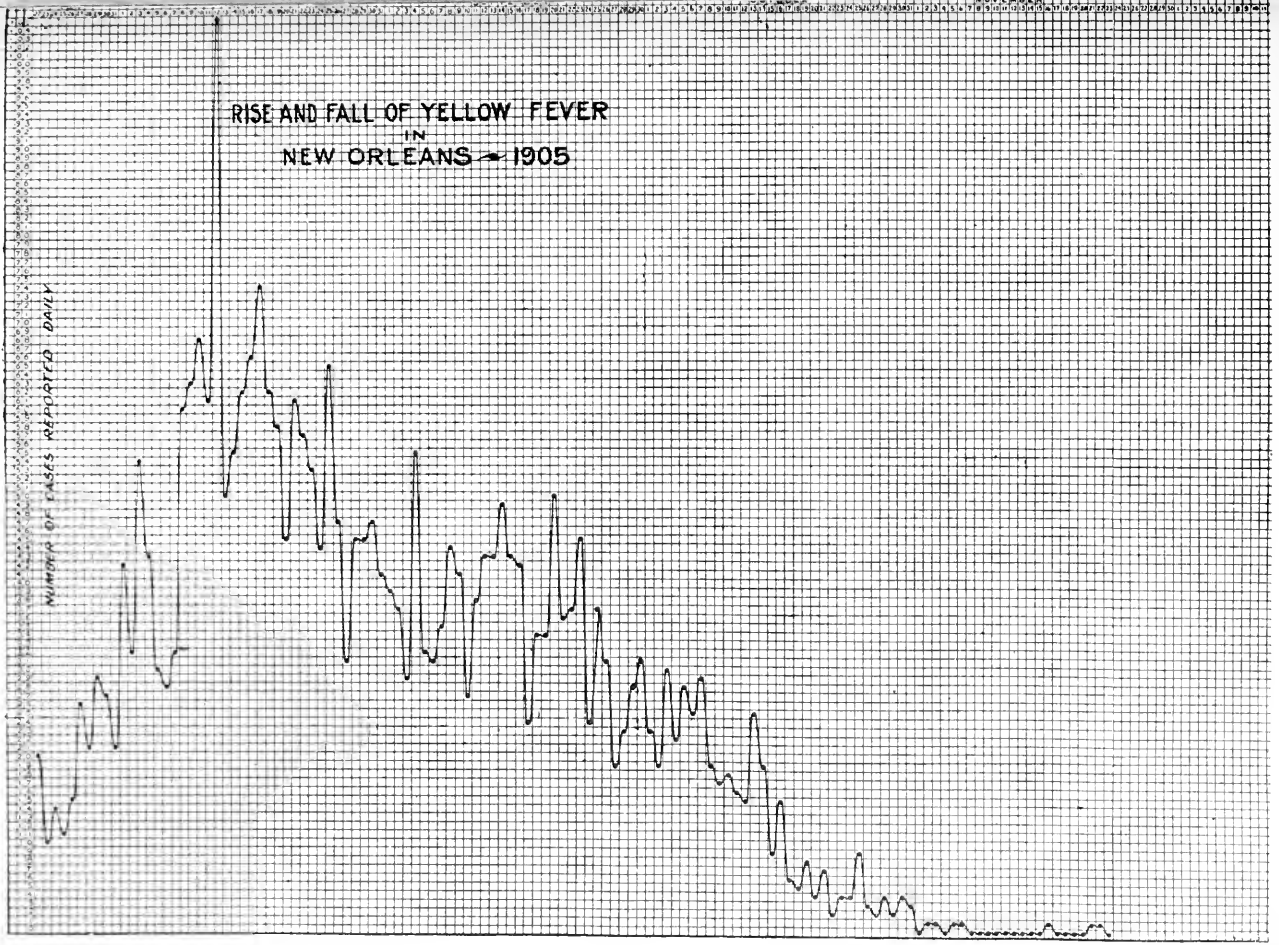

FIG. 32.—Observe the steady decline in the epidemic from the first week in August. In previous epidemics the fever continued or even rose during August.

[*To face p.* 240.

severe case of yellow fever. The outbreaks in West Africa this year, 1910, partake of the same character, and there is a sudden outbreak of fatal cases of yellow fever both in Freetown and Secondee. But as in New Orleans, in Barbados, and in Belize, there must have been other cases unrecognised. This statement raises the question of whether yellow fever did not exist all the time amongst the natives; in other words, whether it was endemic or not. In the case of New Orleans and Barbados the evidence is in favour of importation; in the case of Belize the evidence is about equal for importation and the endemic origin. In the case of West Africa, the evidence, in my opinion, conclusively points to its endemic origin.

In the first place, importation could not be proved; in the second place, there were four outbreaks in towns separated from one another. In the third place, the disease selected out the whites; there were very few recognised cases amongst the blacks. This was not the case in New Orleans, nor in Barbados, where the blacks suffered equally with all other classes of the community. Had the disease been imported in West Africa, the blacks should have suffered far more than they did. The inference therefore is, that the native black inhabitants had been partially immunised by previous attacks of the disease; in other words, that the disease has all along been endemic to the West Coast of Africa.

Yellow fever houses.—In Barbados I was shown several houses which had the evil reputation that if anyone went to reside in them they were very liable to get yellow fever. This is an old observation, which, in the period before the *Stegomyia* doctrine, appeared exceedingly strange. To-day, however, we have a very complete explanation in the *Stegomyia* doctrine. The following report, written by Dr Durham in 1900, to the Liverpool School of Tropical Medicine, just at the time that the *Stegomyia* doctrine was being proved in Havanna, is of great interest:—

"It seems to be fairly definitely established," he wrote, "that a yellow fever patient may become a danger by 'infecting

the house' in which he is placed. Given that a house is 'infected,' a visit by a non-immune person entails considerable risk of contracting the malady. It is alleged by some that visits made at night are more dangerous than those made during the daytime; but here the evidence is not very clear, and is more of the nature of an opinion. The nature of the essential factor present in an 'infected house' is as yet mysterious. One house after another in a street may become 'infected' without any apparent intercommunication of the inmates; the infection may skip over one or more houses and reappear at some distance. There are those who are bold enough to predict in a village that such a house will yield one or more cases of the fever on or about a certain day; and, naturally, they claim to be true prophets.

"Of the interesting and important facts which have been ascertained, those elucidated by Dr Carter in his study of outbreaks at Orwood and Taylor (Miss.) in 1898, are second to none. The conditions were such that the intervals between the introduction of 'infecting' cases and the onset of secondary cases could be followed with accuracy. Dr Carter finds that an interval of about fourteen to twenty-one days obtains before the first secondary cases occur. The house is then in an 'infected' condition, and exposure for a few hours (for example, in one case four hours and a half) can lead to infection, with the incubation interval up to the normal four or five days. This was exemplified to us by the history of a case at Quemados, for which we are indebted to Dr Reed. In a house which had been occupied by non-immune officers last year, two cases of yellow fever occurred this summer; one of these was unfortunately fatal. However, a male and a female nurse who had been occupied in tending the patients did not acquire the disease until about a fortnight after the death occurred. Other sources of infection could be excluded in these cases. No further cases occurred, as the house was cleared and liberally treated with perchloride. The slight epidemic, however, spread to other houses down the street, although they were detached and surrounded by a small amount of garden space.

"This curious and somewhat prolonged interval is suggestive

of a development of the infecting factor in or about some agent or matter in the domicile. What may be the nature of this supposed agent is not yet demonstrated; but the suggestion propounded by Dr C. Finlay, of Havanna, some twenty years ago, that the disease was spread by means of mosquitos, hardly appears so fanciful in the light of recent discoveries in ague conveyance, as appeared in the days when the idea was first broached.

"Dr Finlay's hypothesis is able to account for several curious points which obtain with yellow fever. Thus the limitation of the disease to the 'yellow fever zone,' where frost is unknown, the coincidence of yellow fever and rainy seasons, the cessation of the disease when the temperature falls below a certain point, and its non-recrudescence in an infected locality after a frost, are all compatible with an agency, such as a gnat, which becomes too sluggish to bite, or indeed which dies out in unfavourable climatic conditions.

"Such a theory also explains the curious spread of the disease from house to house, which has already been referred to. Another point is that the sanitary condition of a house may be good, and yet it may be severely 'infected.' An example of this was shown by the case of one of the leading hotels in Havanna, of good sanitary repute, but the source of many fever cases this summer.

"The above sketch will suffice to show that some means of transmission by the aid of an intermediate host—a town-loving host for this town-loving disease—is to some extent more plausible than might be anticipated. Whether that hypothetical host is of the nature of a gnat remains unknown."[1]

II.—*Periodicity and Seasonal Prevalence*

There is a tradition in most tropical countries that yellow fever makes its appearance at definite intervals. Thus Dr Pavy states that "a belief commonly prevails in the West Indies that outbreaks are periodic. Its occurrence at intervals of time may be considered as well established; but it cannot be

[1] This was written just prior to the great discovery of Reed, Carroll, and Agramonte.

admitted," he adds, "to be equally well established that the intervals are regular and alike so as to admit of being calculated."

The opinion at one time prevailed that it might be possible for a traveller to calculate when a particular country, liable to yellow fever, was or was not safe to travel in.

Whilst in Barbados in 1909, investigating outbreaks of yellow fever, I was informed that this belief still existed, and that the period was regarded to be thirteen years. A similar belief in periodicity has existed on the West Coast of Africa, where the period is believed to be seven years.

In the light of modern knowledge we can readily understand how in the days immediately preceding the great development of sea-trade, populations were much more fixed, and therefore less liable to fluctuation than to-day. The influx of strangers was far less common than now; people resided many years in a tropical town, and did not lightly face a long and perilous sea voyage in a sailing ship. In consequence families grew up who had not been to Europe.

When, therefore, an epidemic did break out, it attacked all non-immunes present in the particular area. Those who recovered, both children and adults, were partially immunised for a term of years, and, in consequence, there would be an apparent cessation of yellow fever in its severe or epidemic form; no doubt, however, mild cases did occur, and were diagnosed as remittent endemic fever.

After an interval of some years, it is probable that those who had yellow fever in their infancy had lost to a considerable extent their immunity, and had again become susceptible. Given a new population consisting of a large number of inhabitants who had become non-immunes and some new arrivals, and the conditions were present for an epidemic; and this invariably did occur. To this extent only is there periodicity. In the present day, with the daily arrivals of passengers by land and sea, there is constant liability to

yellow fever, provided there are infected *Stegomyia* in the locality.[1]

Seasonal prevalence.—In the older works on yellow fever, much attention was given to the seasons of the year in which yellow fever was most prevalent, and no doubt in the older days, seasonable prevalence was much more marked than it is to-day.

To-day pipe-borne water supplies are common in the larger towns. In the old days the inhabitants had to depend altogether on roof water and wells, and this would depend on the rains.

During the rains, therefore, more receptacles would be filled with storage water, and for many weeks there would be a very considerable increase in the breeding of the *Stegomyia*. To-day the facilities for breeding are, as a rule, fairly uniform throughout the year. In all probability more discarded receptacles, such as tins of all kinds, become breeding-places for the *Stegomyia* during and after the rains; but in the dry season the tendency to store domestic water in more receptacles would also favour breeding. Another factor which would greatly influence the breeding of the *Stegomyia* is the external temperature.

It was well known that yellow fever epidemics only occurred in towns in North America and in Europe in the hot months, the advent of cold put an end to them at once. In the tropics, however, this factor does not count to any great degree, owing to the far more uniform high temperature throughout the year. Therefore, outbreaks are liable to occur in any season, provided that the *Stegomyia* is in sufficient numbers and infection is present.

In West Africa opinion seems to favour the view that

[1] Faget pointed out the regularity with which each successive batch of poor immigrants into New Orleans were infected with yellow fever as soon as they took up their quarters near the port, a quarter which appeared to be a perpetual endemic nidus.

yellow fever is most common after the rains. Perhaps the months in which the disease has most frequently occurred have been from April to June; but this might simply depend upon the circumstances that at this period there were new arrivals. My analysis of the outbreaks in West Africa show that no month is free from the disease.

In the West Indies, April to September are perhaps the months most frequently implicated. It is a very old observation that frost puts an end to an epidemic. We now of course know that this is due to the stoppage of breeding of fresh supplies of *Stegomyia* and the torpidity of the *imagos*. But there is no doubt that infected *Stegomyia* may hibernate in houses during the cold weather, and become active again in the following year. Numerous cases of this kind have occurred in Spain and in the United States. The same phenomenon has time and time again appeared on board ships. A ship with cases of yellow fever on board sailed into a cold region, and the fever ceased. On the ship again returning into a hot climate, yellow fever reappeared. The explanation is that the heat caused increased activity of the *Stegomyia* on board, and no doubt also caused them to breed.

Trade Routes and Yellow Fever

Disease follows trade routes whether by land, sea, river, or rail. In West Africa there has existed abundant communication between the colonies both by land, sea, and river. So that admitting, for argument, that yellow fever was introduced some time in a past century into some colony or district in West Africa, it could have readily spread to all other parts of the West African Coast, for the conditions, no doubt, have all along been ideal: abundance of *Stegomyia* and abundance of natives. Therefore, yellow fever having once been introduced into West Africa, we must admit that it could readily have spread throughout the colonies.

This very fact would have necessarily led to the disease becoming in a few years endemic, so that for all practical

purposes it may be freely assumed that yellow fever is to-day endemic in West Africa, for at no period in the history of West Africa were effective measures taken to prevent the spread of the disease from one colony to another, by means of the innumerable channels of intercourse both by land, river, and sea.

Therefore in seeking an explanation of the outburst of severe yellow fever amongst the whites at Freetown, Secondee, Axim, and Saw Mills, this year, 1910, it is not necessary to look for some ship which might have conveyed the infection from Central America.

The infection was at hand in the native population. Therefore the question of the transference of yellow fever by trade routes in West Africa is under present conditions of little moment, compared with the infinitely more vital questions of eliminating the disease from the natives by *Stegomyia* destruction, and by protecting the non-immunes by means of segregation.

It is a very old observation that *from the moment Europeans settled amongst native races in the tropics and began to trade, yellow fever appeared.* When large populous trading centres grew up, such, for example, as those of Havanna, Rio, and New Orleans, then these became the foci from which yellow fever spread to other parts.

Ships are especially liable to transmit disease from one part of the world to another. In the seventeenth, eighteenth, and nineteenth centuries, North America and Europe became repeatedly infected by the arrival of infected ships.

Yellow fever was so common in commercial ports that it came to be looked upon as a disease peculiar to seaports; but it was soon observed that the moment the interior of the country was opened up through the energy of miners or planters, that yellow fever appeared just as formidably in the interior as on the coast; and moreover, that it did not remain confined to the flat coast-line, but ascended into the mountainous districts.

In other words, the situation of a town had nothing to do with the appearance or absence of the disease, given that the conditions of temperature, water, and infected natives were adequate and suitable to the propagation of the *Stegomyia*, yellow fever broke out.

Miners' and prospectors' camps, villages which sprung into existence during railway construction, were all the seats of election of the *Stegomyia.* They found ready at hand, and in abundance, the innumerable odds and ends in the shape of discarded meat, fruit, and condensed milk tins, etc., which invariably contain water after a shower of rain, and which are always to be found in and around workmen's dwellings. Moreover, the usual large collection of barrels necessary for the storage of water, in the newly erected settlements before the pipe-borne water supply is laid, furnish ideal mosquito breeding-grounds.

In connection with the transmission of yellow fever, as also of plague, two factors have always to be taken into account: man on the one hand, and the insect carrier on the other. Therefore in devising means for combating these diseases, these two aspects must be always before the medical officers. The various ways, for instance, in which man and the mosquito may elude his vigilance must always be borne in mind.

It must be remembered also that as in so many other instances, it is the unexpected which happens. In the case of man the danger usually arises because the symptoms may be so slight that they escape detection, and persons, not knowing that they are suffering from the disease, move from place to place.

It is for this reason that the rigorous systematic use of the thermometer should never be omitted in the case of passengers arriving by ship or train from infected towns. Under these circumstances it is also advisable to keep new arrivals under daily observation. It may even be necessary, if there is reasonable ground for suspicion, to detain passengers in a quarantine station or observation camp.

In the case more especially of native permanent communities, black or Indian-Spanish, amongst whom there is reason to suspect a mild endemic form of yellow fever, every precaution must be taken to deflect the traffic so far as possible from them.

If the difficulties of supervision of travellers requires great vigilance, those attending the prevention of the migration of the *Stegomyia* are still greater.

Time and time again experience has taught us that the infected *Stegomyia* is very liable to elude all our vigilance. It may hide in those parts of the ship which were considered most unlikely, and which were in consequence not fumigated; for instance, they have been discovered in the engine-room of a ship.

Since sailing ships have been replaced by steam-ships, the risk of ships carrying yellow fever has been, of course, greatly reduced; but it does occur, and I have known instances where a stowaway has carried infection, and where infected mosquitos have been carried.

Sailing ships are still largely engaged in the coasting trade in tropical countries, and in them the conditions are much as they were in the eighteenth century; therefore it is imperative to rigorously inspect and fumigate them. In many places canoe traffic is also largely made use of. Their danger lies in the fact that native passengers arriving by them are in all probability ambulatory cases. This form of traffic should therefore be rigorously guarded, and excluded from centres of population. It is, of course, very difficult to do this in many countries, and may entail the employment of much additional assistance.

Daylight communication.—It is a useful practical rule to remember that intercommunication and transference of passengers should be carried out by day rather than by night. Practice shows that there is less risk of infection during daylight than at night-time. In the years when yellow fever appeared regularly in epidemic form in Rio and in Santos, it

was the practice of merchants to leave these towns in the evening by train and sleep with their families in the country. In the morning they returned to town and transacted their business during the whole of the day. A case of yellow fever amongst them was most unusual (Sodré and Couto).

Conveyance by railroad.—As in the case of ships, so in the case of railway carriages, the *Stegomyia* can be transported ; but this does not occur as often as on ships. The conditions are very different on railway cars—there are usually strong currents of air which tend to sweep out mosquitos.

Conveyance by baggage and merchandise.—The danger of this method of transference is not great. It may occur, however, and where bales of goods are packed in places surrounded by native huts, and where the *Stegomyia* is swarming, there is a risk of a *Stegomyia* being included in the baggage or the bale. The danger of *Stegomyia* being found in *packed* baggage is admitted by all authorities to be small. When they are carried it is usually found that they are secreting themselves in the folds of coats or dresses ; therefore they should be looked for in loose clothes. Therefore every care should be taken to pack goods in the open, and away from mosquito breeding-grounds and native houses. This year, 1910, objection was taken by the Government of the Gambia to the bales of Kola nuts packed in Sierra Leone. Theoretically there was a risk, and it would have to be contended with in the case of all yellow fever free districts.

Conveyance by lighters.—Dr Durham investigated a number of lighters at Para, employed for conveying merchandise to and from the wharves to the steamers lying in the river. He found the larvæ of *Stegomyia calopus* in the bilge of the covered lighters. On the other hand, the open lighters were free, which he believes was due to the presence of tarry matter mixed with the water. He concluded that infected *Stegomyia* could be carried both by the lighters and by tenders from the shore to the ships.

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CHAPTER XVI

RACE SUSCEPTIBILITY AND IMMUNITY

A LARGE amount of misconception exists upon the question of race susceptibility. It is sometimes supposed that the Latin races are the most susceptible to yellow fever, others think that the black races are immune. As a matter of fact, the most recent observations prove the accuracy of the old writers of authority upon yellow fever.

Writers of the eighteenth and nineteenth centuries concluded that no race was exempt, and that the apparent exemption of the black races, of the Creoles, and of the Spanish-Indians, would readily disappear if these people went to Europe for a long stay. Modern observations prove that all races are susceptible, provided they come from climates where yellow fever does not exist. *That in fact it is entirely a question of immunity.*[1]

Those living and born in a country where disease is endemic, at a very early period in their life get an attack of the disease, which naturally confers a certain degree of immunity; later they may get subsequent attacks, but each successive attack is less serious; when manhood is reached the subject is in all probability completely immune. This was the case in Cuba, Vera Cruz, Rio, Santos, Para, New Orleans, etc.

It is this fact which explains why yellow fever has always been regarded as a *disease of newcomers;* it does not matter in the very least whether the newcomers are Scandinavians,

[1] Findings of the American-Cuban, French, German, and English yellow fever commissions.

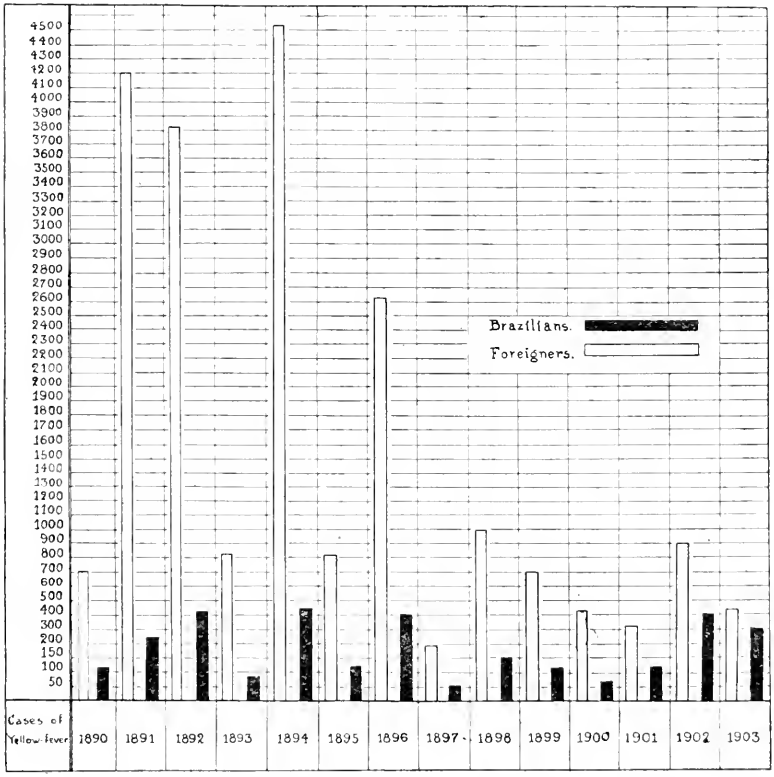

Fig. 33.—Table prepared by Otto & Neumann to show the mortality from yellow fever from 1890-1903, that is the period of active anti-*Stegomyia* measures. The striking feature of the table is the remarkable disproportion of the death-rates amongst the newcomers (Foreigners) and the inhabitants (Brazilians). The table demonstrates the comparative immunity of the inhabitant.

[*To face p.* 252.

British, Latins, Syrians, Mohammedans, Russians, Indians, "yellow skins," blacks or whites, the question alone is: Are they newcomers or not? Have they resided for a long term of years in a country where there is no yellow fever?

It is an old observation that those blacks and "yellow skins" who resided in yellow fever free districts suffered from yellow fever when they reached a town where it was prevalent. During the great yellow fever epidemic in Philadelphia, blacks and whites were equally affected, and there are numerous other examples to the same effect.

The Creoles who leave the West Indies to be educated in Europe, or the Central Americans who left Vera Cruz or other endemic foci for a few years, return as non-immunes and are liable to yellow fever.

It is an old and often repeated observation[1] that the blacks of West Africa rarely, in comparison with the whites, get yellow fever; this has been amply borne out by the outbreaks of yellow fever in West Africa this year, 1910, and in all previous years. On the other hand, last year, 1909, during the yellow fever epidemic in Barbados, *out of a total of* 86 *cases*, 54 *occurred amongst the black inhabitants.* Yet these same blacks were the descendants of the African immunes. They had, therefore, in the meantime become non-immune and susceptible, owing to the fact that yellow fever had ceased to be endemic in Barbados.

In the Martinique epidemic of 1909, the blacks were likewise attacked. The fact that the poor native black inhabitants of Barbados were more susceptible even than the whites, proved, in my opinion, that yellow fever was not endemic.

[1] Hirsch quotes fifteen authorities to show that yellow fever, although it does occur, is less severe and is rarer in the pure negro than in the whites. Nearly every writer on yellow fever has drawn attention to this observation. Bérenger-Féraud describes two cases in negroes, and cites four other authorities. Drs Durham and Myers also described cases amongst the negroes in Para. In the preceding chapters I have also recorded cases amongst the pure negro. The evidence, therefore, is that the disease does occur amongst them, but is rare.

Similarly, the fact that the black races in West Africa do not exhibit, as a rule, the severe yellow fever symptoms, nor die from the disease like the whites, is proof, in my opinion, that they suffer from a mild form of the disease which confers subsequent partial immunity.

We know positively[1] that neither their colour nor their race confers absolute immunity to yellow fever, and that they are as liable as the white European to become infected, provided they have lived out of Africa for a long period.

As to the question of the susceptibility of native races to yellow fever, this is a matter of considerable importance. The following memoranda are of use :—

We know the natives of Central and South America (Indian-Spanish race "yellow skins") appear to be non-immunes. We know that they have perished in great numbers in the interior towns in Guatemala and Spanish Honduras in 1909, and in many other years. On the other hand, we possess positive evidence that in large towns like Rio, Santos, Havanna, Para, etc., the indigenous inhabitants suffered far less severely from yellow fever than foreigners. The figures are conclusive upon this point, and demonstrate that in the large towns the indigenous inhabitants were immunised in childhood by mild attacks of the disease.

There is also evidence based upon tradition that the ancient Mexicans suffered severely from yellow fever.

We know that the Hindoo race is non-immune. A statement is made on the authority of Hirsch that the Chinese in South America appeared immune to yellow fever. With regard to the negro race we know that mulattoes, quadroons, and other mixed varieties are quite as susceptible as the whites to yellow fever. All authorities agree that the pure blooded negro can get yellow fever, but that in the majority of cases it is less severe than in the whites.

In other words, observers agree that there is some degree

[1] Blair, Hirsch, Chassaignac, La Roche, and others.

of immunity amongst the blacks or Spanish-Indians. Scheube is of this opinion, and so were the older observers like Blair. The following figures given by Chassaignac during the 1905 [1] New Orleans epidemic are of great interest. He states:—

It was conclusively shown that during the 1905 epidemic, negroes and whites were equally liable to contract the disease, but that the former had it in a particularly mild form. The statistics were as follows:—

In one series of observations the mortality amongst 90 white cases was 20 per cent., and amongst 950 coloured cases, 1·2 per cent. In a second series of observations the mortality amongst 80 cases amongst the whites was again 20 per cent., and amongst 247 coloured cases, 3 per cent. In a third series out of 500 cases amongst the whites there were 51 deaths, and out of 200 coloured cases, 1 death.

Chassaignac adds that the evidence is of the greatest importance, because it shows that the blacks are equally susceptible with the whites, but that they get the disease in a remarkably mild form, which can be readily overlooked.

La Roche states that in Jamaica the mortality rate for the white troops was 102 per thousand, and for the black soldiers, 8 per thousand. In the Bahamas the rate amongst the white troops was 59 per thousand, and amongst the blacks 5 to 6 per thousand.

These observations also disprove the belief that blacks are immune.

Blair states that out of 1790 imported blacks, none contracted yellow fever during the 1852 epidemic.

There is further evidence to show that the imported negroes in the slave days did not get the disease in the severe form in which the whites did.

If we examine the events of this year, 1910, on the West Coast of Africa, it is clear that the blacks did not suffer in the same proportion as the whites. Taking the relative proportion of

[1] J. Lazard states that there were 452 fatal cases amongst the whites and 6 amongst the negroes in the 1905 New Orleans epidemic.

the whites and blacks present on the Gold Coast and in Sierra Leone respectively, there is a relatively very high case and death-rate amongst the white population, and an exceedingly small one amongst the black population.

The older observers on the West African Coast, like Staff-surgeons Barry, Fergusson, and many others, were emphatic that the negroes did not suffer from yellow fever to anything like the same degree as the whites in the various epidemics on the coast.

On the other hand, as previously stated, we know that the present-day descendant of the West African native, as he is met with in Barbados, is not immune ; we know that the same descendant serving in the West African regiment in the barracks at Freetown is not immune, and that from time to time in the various epidemics, cases and deaths have occurred.

These apparently conflicting observations can, in my opinion, be explained only upon the ground of acquired immunity. The West African negro has an acquired immunity, and the evidence is in favour of his having been in this position in slave days, just as I believe he is to-day.

Therefore slaves exported from West Africa and newly arrived in the States or in the West Indies were to a certain degree protected by previous attacks in childhood, and did not suffer like the other races. When, however, new generations of negroes had grown up under the new conditions in Barbados or elsewhere, where there was considerable or complete protection from the *Stegomyia* owing to its smaller numbers or complete absence, they naturally became non-immune. They no longer got the mild attacks that their ancestors were accustomed to suffer from, either in Africa or during transport on the old-time slave-ships.

The evidence, in my opinion, points strongly, not only to yellow fever being endemic on the West African Coast, but that the home of yellow fever may be, after all, West Africa, and not the New World.

Whether the immunity just described is inherited in any

degree, it is very hard to decide. If there is an inherited immunity, it is soon lost as demonstrated above; and the immunity as it exists in the African negro to-day can be explained by his having acquired this immunity by inoculation.

Vice versâ, it is a well-known and often proved observation that those whites who, being born in yellow fever countries, survive childhood, grow up to be resistant to yellow fever. The old physicians said that they became acclimatised, whilst Creoles who left for a long stay in Europe became de-acclimatised.

We now know, thanks to the experimental observations of the American, Cuban, French, and Brazilian observers, that acclimatisation means becoming infected by the *Stegomyia* with a mild attack of yellow fever which confers partial immunity. The word "acclimatisation" of the eighteenth and nineteenth centuries means "immunisation" to-day.

The most striking figures showing the effects of local immunisation is furnished by the mortality tables of Rio since the year 1890. A glance at this table shows that the mortality amongst new arrivals was ten to eighteen times greater than amongst the natives of Rio. A more conclusive demonstration of immunity through previous attacks could not be furnished, and it finds its exact parallel to-day in the history of yellow fever in Africa. The figures for Rio are :—

Year.	Brazilians.	Foreigners.
1890	76	636
1891	249	4083
1892	374	3716
1893	43	777
1894	384	4372
1895	64	754
1896	304	2599
1897	15	141
1898	110	955
1899	86	639
1900	39	303
1901	79	220
1902	201	777
1903	188	376

Since the general application of anti-*Stegomyia* measures in Rio, the enormous difference between the death-rate amongst Brazilians and foreigners has appreciably decreased, and no doubt will disappear, owing to the fact that all the inhabitants are rapidly becoming non-immunes. Applying this reasoning to West Africa, we know that outbreaks of yellow fever have been far more frequent than is usually supposed during the last hundred years.

We know that in this year, 1910, yellow fever broke out independently at four separate points in British Colonies, viz., Freetown, Secondee, Axim, and Saw Mills.

We know that inquiry on the spot failed to prove importation. We are forced therefore to conclude that the disease was, to use the phraseology of the older observers on the coast, a product of the place itself. In other words, that the disease was endemic.

If, then, the disease is endemic—a view which is held by some British, German, and French authorities, who have investigated the disease upon the spot—it remains to ask: By whom is the virus of yellow fever maintained?

From whom do the *Stegomyia* derive their infection? The answer to this can only be the black races; we know that the whites are not immunised in sufficient numbers. On the other hand, we know that the black races are very considerably immunised, or how otherwise would they escape yellow fever? Then it is to the black races that we must look for the source of supply of the yellow fever virus; it is they who, in childhood and adolescence, have the disease in a mild form: but mild though it be, quite sufficient to infect the *Stegomyia*, as the inoculation experiments of the American Commission proved. In other words, the black natives have the so-called mild or ambulatory form of yellow fever.

These mild forms pass unrecognised amongst the natives just as the sister disease—malaria—does, but, nevertheless, like malaria, it is there.

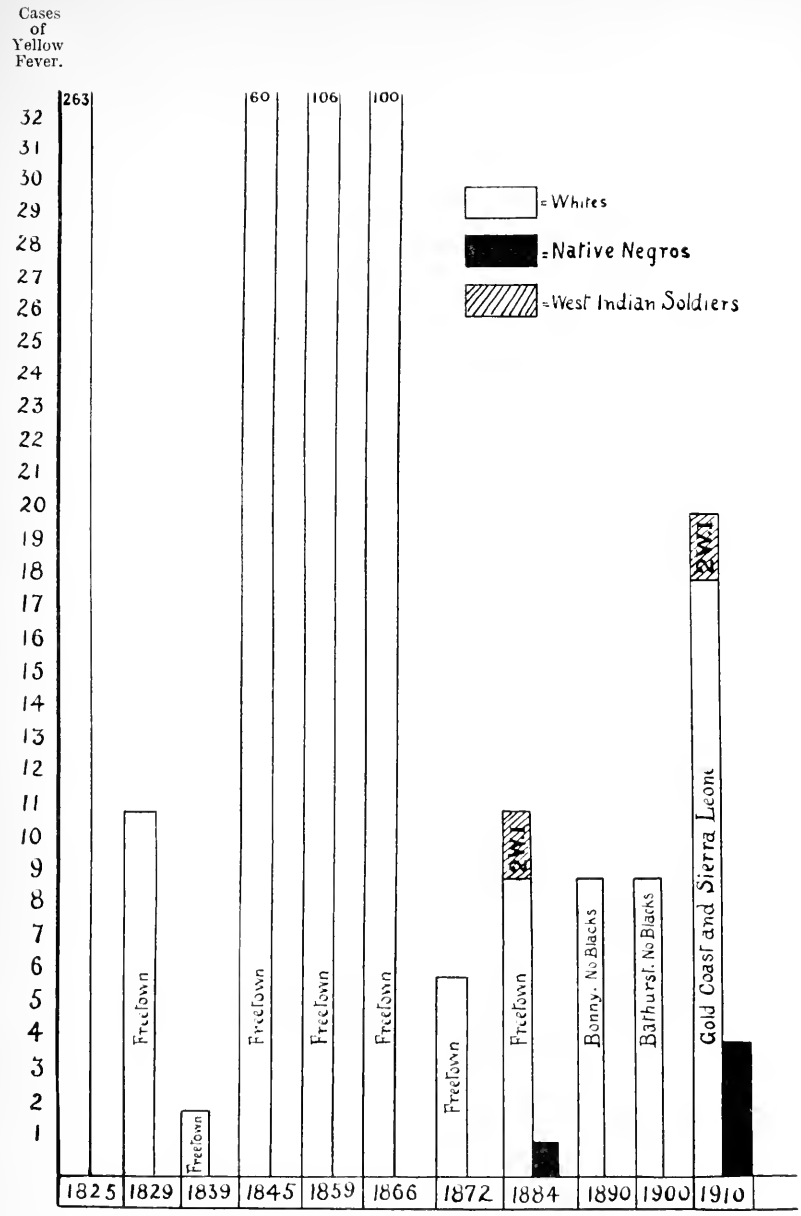

FIG. 34.—Diagram showing some of the outbreaks of yellow fever in West Africa from 1825-1910. Until 1884 there is no record of deaths from Yellow Fever amongst the black natives. From 1884 note the marked disproportion between the white and black death-rate.

[*To face p.* 258.

From analysis of all the facts I can come to no other conclusion than that a very large proportion of the "remittent" and "bilious remittent" fevers of West Africa to-day are the well-known mild forms of yellow fever. The African native is as saturated with yellow fever as he is with malaria, and his escape from severe yellow fever, and its very frequent occurrence amongst the whites, is proof of this contention.

But, it may be argued by some, if yellow fever has been endemic in Africa so long, how comes it that all the whites have not perished?

As a matter of fact the death-rate from *diagnosed* yellow fever has been exceedingly high; in the second place there is no doubt whatever that a vast amount of yellow fever has passed *unrecognised* amongst the whites; in the third place European whites have during the last decade taken measures which, without their being aware of it, protected them. They have lived to a certain extent segregated in larger and better houses, and the use of the mosquito net is now very general. Both these measures afforded a very great degree of protection. It is well known to students of yellow fever, the remarkable comparative immunity which even partial segregation gives. Were this not a well-recognised rule in connection with all infectious diseases, mankind would have long since disappeared.

The final proof that remittent and bilious remittent fevers are in a large number of instances the mild forms of yellow fever, will be given when by the destruction of the *Stegomyia* in West Africa, these fevers will, if my reasoning be correct, disappear in great part with yellow fever, precisely as they have done from Barbados, Rio, and from ships, from the Southern States of America, and from Spain.

Liability of new arrivals to yellow fever.—It is surprising how few people stop to consider what is the significance of the remarkable proneness of new arrivals to yellow fever. This is

nevertheless one of the oldest and best authenticated observations in connection with the disease.

In *Health Progress and Administration in the West Indies*, I have drawn frequent attention to it and given the statistics of the mortality rate. The older observers summed up the liability of new arrivals, by stating that the lower the temperature of the country in which the newcomer had resided, the greater was the liability to yellow fever. Blair gives the following two tables:—

Mortality Incidence according to Race.

No.	Nationality.	Percentage.
1	West Indian Islanders . . .	6·9
2	French and Italians	17·1
3	English, Irish, and Scotch . .	19·3
4	German and Dutch	20·0
5	Swedes, Norwegians, and Russians .	27·7

This race proportion, although it has not to do with the temperature of the native country of the arrivals, nevertheless may be brought about by the fact that the inhabitants of the West Indies were no doubt considerably immunised before they arrived in British Guiana. In the same way the French and Italians might have come from South America and the West Indies, in both of which places they would have been liable to attacks of yellow fever. The evidence is overwhelming that race does not confer complete immunity. In the 1905 New Orleans yellow fever epidemic, yellow fever was most prevalent and severe amongst the Sicilian workmen, who formed a large proportion of the labouring population of the city, and who were for the most part new arrivals; but in the previous epidemics when the labouring population was chiefly new arrivals from Ireland, these were most affected. It was again the question of non-immunes. The following useful table of Blair's shows the mortality rate amongst new arrivals:—

Table of 489 *Cases of Yellow Fever admitted into the Seaman's Hospital, Demerara.*

Time in Harbour.	Total Cases.	Deaths.	Percentage.
Under 1 week . .	19	1	5·2
1 to 2 weeks . .	84	17	20·2
2 „ 4 „ . .	137	38	27·7
4 „ 6 „ . .	98	31	31·6
6 „ 9 „ . .	96	34	35·4
9 „ 12 „ . .	32	5	15·6
12 „ 16 „ . .	17	1	5·8
16 upwards . . .	6	2	33·3

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PART V

ENTOMOLOGY

For much practical assistance in the preparation of this chapter, I am indebted to my friend and colleague, Prof. Newstead; and for permission to reprint the section dealing with the *Stegomyia* in West Africa, I am indebted to the courtesy of Dr Guy Marshall, Secretary of the Entomological Research Committee.

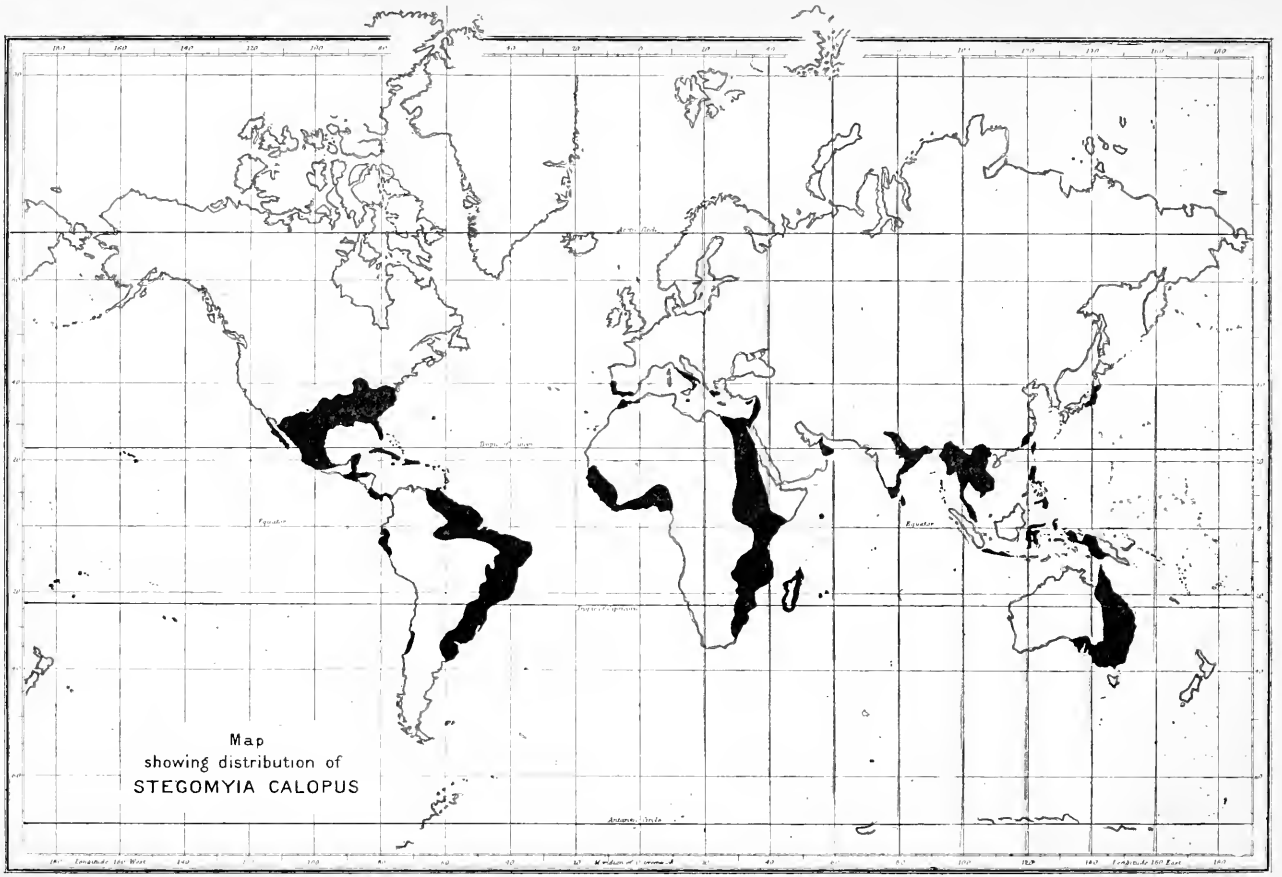

FIG. 35.—Note that the distribution of the *Stegomyia* is more widespread, especially eastward, than is yellow fever. The relative volume of the mosquito is not indicated; it is much more abundant in some areas than in others.

[*To face p.* 265.

CHAPTER XVII

GEOGRAPHICAL DISTRIBUTION OF THE *STEGOMYIA CALOPUS*

It will be seen from the following summary and from the chart, that the distribution of this mosquito is widespread over the tropical regions of the globe.

In Central America, in the West Indies, in the Gulf Ports of the United States, in South America, and in West Africa, a large number of investigations have been made which show that the mosquito was, or is, exceedingly abundant in these parts. On the other hand, less is known of the relative abundance of the *Stegomyia* in the eastern ports and in Australasia, although the presence of the mosquito has been noted.

Since 1899, the period of the organised attack upon yellow fever, the abundance of the *Stegomyia* has been substantially reduced in very many places, notably in the Gulf Ports, Central America, the West Indies, and in parts of Brazil.

Moreover, improved sanitary control of towns and the introduction of pipe-borne water have also slowly brought about a great reduction in the number of breeding-places in centres of population. So much so has the reduction taken place that to this alone can be ascribed the disappearance of endemic yellow fever from towns like New Orleans, Panama, Vera Cruz, Rio, and Santos, and from many of the West Indies.

There is no doubt that a very large volume of *Stegomyia* is necessary to keep up endemicity. This fact has been pointed out by Gorgas and by other observers, and from my own

investigations in the West Indies, in Central America, and in West Africa, I have come to the same conclusion.

I have pointed out in *Health Progress and Administration in the West Indies*, the remarkable contrast of fifty years ago with the state of affairs to-day, as regards yellow fever in the West Indies; formerly, almost every new arrival became infected with yellow fever; to-day this does not occur, and the West Indies are regarded rightly as pleasure and health resorts.

The only way in which it is possible to explain this extraordinary diminution in the disease is by the equally great diminution in the numbers of the carrier of the disease, the *Stegomyia*. There can be no doubt that this insect has been in places enormously reduced in numbers. The reduction has therefore led to the interruption of continuous yellow fever infection, such as is necessary to maintain an endemic state.

From time to time, however, small outbreaks of yellow fever occur, as last year, 1909, for example, in Barbados.

These are explained by the persistence of localised foci in certain towns of the *Stegomyia* in very large numbers. When, therefore, a case of yellow fever is introduced from some country where it is still endemic, it lights up a small localised outbreak confined strictly to the particular *Stegomyia* area. If the infected *Stegomyia* and the larvæ are killed in such an area, yellow fever stops in a few weeks and cannot progress.

Considerations like these make it of the greatest practical importance to ascertain in all localities, whether the *Stegomyia* is present in sufficient numbers to keep up endemic yellow fever, or only to maintain an outbreak or a few cases, were the disease imported.

For this reason, I make it a rule to make house to house examinations, in order to arrive at an accurate percentage distribution of the *Stegomyia*.

I assume that if every house and yard is breeding the *Stegomyia*, that is, if the *Stegomyia* is present in a town to the extent of 100 per cent., that in all probability yellow fever is

FIG. 36.—Typical appearance of a neglected Yard and unprotected Water Barrels. The barrels and kerosene tins swarm with *Stegomyia* larvæ. From Belize, British Honduras, 1906.

[*To face p.* 266.

endemic. If, on the other hand, the percentage is only 10 per cent., then the disease is not endemic. Of course, in many towns the percentage distribution is not by any means uniform. For example, in one particular, poor, neglected, and overcrowded quarter in a town or village, the *Stegomyia* may be present to the extent of 100 per cent., whilst in the suburbs the percentage may be less than 5.

In such a case as this, it is possible for yellow fever to become epidemic and to remain endemic in the former quarter, whilst in the 5 per cent. area only a few sporadic cases may be recorded. In the history of yellow fever in North America, in Europe, and recently in the West Indies, this striking relationship of yellow fever to the number of *Stegomyia* has been over and over again noted.

Further information upon these points will be found in my British Honduranian and Central American Report on yellow fever, and more recently in my investigations in the West Indies recorded in *Health Progress and Administration in the West Indies.*

I.—Summary of the Geographical Distribution of the *Stegomyia calopus.*

Africa.—*British East Africa*, Mombassa; *Natal*, Durban; *Egypt*, Cairo, Ismailia, Khartoum, Nile generally; *Senegal*, Senegambia, Sierra Leone, Gold Coast, Togoland, Dahomey, Nigeria; *Mashonaland; Mauritius*, Port Louis, common near shore, scarcer in high parts; Fernando Po, Cape Verd Islands; *Portuguese East Africa*, Delagoa Bay; *Seychelles*, Victoria; *Transvaal*, Komatipoort; *Zanzibar*, Nairobi.

America (North).—*Lower California; United States:* "In considerable numbers in the Southern United States and as far north on the Atlantic coast as Virginia" (Howard). Alabama, Arkansas, Florida, Georgia, Illinois, Indiana, Kentucky, Louisiana, Maryland, Mississippi, Missouri,

North Carolina, Savannah, South Carolina, Tennessee, Texas.

AMERICA (SOUTH).—*Brazil*, Manaos, Para, Iquitos, etc., Rio de Janeiro, etc.; *British, French, and Dutch Guiana*, Venezuela; *Chili*, Valparaiso; *Ecuador*, Colombia, Guayaquil; *Peru*, Callao.

AMERICA (CENTRAL) (along the Atlantic coast-line).—*British Honduras*, Belize, Cuba, Puerto Cortez; *Costa Rica*, Limon, Bocas del Toro; *Guatemala*, Livingston, Puerto Barrios; *Mexico*, Acapulco, Carmen, Champoton, Ciudad Victoria, Coatzocoalcos, Cordoba, Cozumel, Frontera, Juanajuata, La Paz, Las Penas, Linares, Mazatlán, Mérida, Monterey, Nantla, Orizaba, Pachuca, Perihucte, Pochutla, Progresso, Rincon, Antonio, Salina Cruz, Saltillo, San Blas, Tampico, Tepic, Tlacotalpam, Tonala, Tuxpan, Vera Cruz; *Nicaragua*, Bluefields; *Panama*, Ancon, Colon, Culebra.

WEST INDIES AND THE BAHAMA ISLANDS.—Barbados, Bermuda, Cuba, Dominica, Grenada, Jamaica, Nassau, Nevis, Porto Rico, San Salvador, St Kitts, St Lucia, St Vincent, Trinidad.

ASIA (including East Indies).—Andaman Islands; *Arabia*, Port Said, Muscat; *Assam*, Lushai Hills; *Burmah; Celebes; Cochin China*, Hatien, Tonkin; *India*, Rhim Tal, Calcutta, Ceylon, Ferozepore, Kumaon, Lucknow, Madras, Purneah Quilon, Travancore; *Japan*, Tokyo; *Java*, Batavia, Garvet, Samarany, Soekaboemi; *Malaya*, Perak, Singapore; *New Guinea; Palestine; Philippines*, Luzon, Mindano, Panay; *Siam*, Phrapatoon.

AUSTRALIA (including OCEANIA)—*Fiji Islands; Hawaian Islands*, Hilo, Honolulu; *New South Wales; Pitcairn Islands; Queensland*, Bupengang; *Samoa*, Apia; *Solomon Islands; South Australia*, Port Darwin; *Victoria.*

EUROPE.—*Cyprus*, Larnaka; *Greece*, Poros; *Italy; Malta* (reported 1910); *Portugal*, South Portugal; *Spain*, Dol; *Gibraltar*, Malaga.

FIG. 37.—A Row of Upturned Bottles, used to make an edge to a flower-bed in Freetown, Sierra Leone. They are favourite receptacles for *Stegomyia* larvæ. (From *Mosquito or Man?*)

[*To face p.* 268.

II.—Investigation of Breeding-Places

I have learnt, as the result of long experience, that the only sure way to arrive at a correct estimation of the number and kinds of mosquitos present in a town, is to make a systematic house to house inspection of all articles containing water.

For this purpose, it is necessary to have a block- or house-plan of the town, and to divide the town up into sections, and then to work over each section house by house.

In specially printed note-books, divided into columns for cisterns, tanks, barrels, tubs, wells, kerosene tins, "odds and ends," broken crockery, bottles, and "other receptacles" (as flower vases, lily tubs, etc., etc.), the number of water-containers found is systematically entered, and a note made as to whether *Stegomyia* larvæ are present or absent.

When I and my assistants (usually the sanitary inspectors) have made our survey, all the odds and ends and discarded tins are collected together and brought out into the street for the dust cart to remove; if larvæ are found in barrels or cisterns, the water is emptied, or if that is impracticable, kerosene oil is poured in; the occupier of the house is admonished and reasoned with, and the sanitory inspector enters the name and address of the offender in his book, and if the nuisance occurs again, action is taken. In order to discover all the discarded tins, etc., it is very often necessary to get the wild bush in the compound cleared; and this invariably discloses a large number of receptacles containing stagnant water and harbouring innumerable larvæ.

In addition to the examination of the yard, the interior of the house must not be overlooked, for it is quite a common occurrence to find the larvæ of *Stegomyia* in collections of water allowed to remain stagnant in the house; for example, in flower-vases, saucers of flower-pots, glasses employed for striking cuttings of the croton plant, water ewers, indoor water cisterns, fire buckets, etc. I have found the larvæ on more than one

occasion in the water used for cooling the irons in a blacksmith's shop.

The abundance or otherwise of the *Stegomyia* may be influenced by the nature of the occupation of the towns-people. In a fishing village, or where canoes are abundant, enormous numbers of larvæ are frequently harboured in the rain-water which collects in the canoes. If there is much cooperage, rain-water often collects on the upturned bottoms of the barrels, and harbours larvæ; in some villages, conch or snail shells are abundant, and these contain larvæ; in other places calabashes or cocoanut husks abound. In Belize, British Honduras, I found vast numbers of larvæ in the irregular depressions and forks in the logs of logwood piled up on the wharf ready for the steamer; the purple, almost black-coloured water in the holes did not in the least affect the larvæ. Then again, every country has its own special receptacles liable to contain larvæ. In Louisiana, Central and South America, the large wooden rain-water vats are the common offenders. In many of the West Indies large stoneware jars (olive jars) create a nuisance. In Trinidad the "antiformicas" placed around flower-beds to protect the flowers from the attacks of the umbrella ant usually harbour larvæ. In Freetown, the habit of making "ornamental" borders to flower-beds by sinking into the earth a row of inverted bottles is a fertile source of *Stegomyia;* the cup-shaped depression at the bottom of the bottles holds water, and in these the *Stegomyia* deposits her eggs. Imperfectly broken glass on walls is another source. Rot-holes of all kinds in trees, the axils of the Aroideæ, of the Traveller's Palm, and of many other plants, form receptacles which may prove a nuisance. I have found larvæ breeding in the puddles formed on the flat mud roofs of houses in Cape Coast Castle. The roof-gutters of houses are common receptacles. Less frequent breeding-places are marsh-holes, puddles, and drains. I have found *Stegomyia* larvæ in all these latter places, but in my opinion, less frequently. In my experience, this mosquito

FIG. 38.—A Sama Tree, with Epiphytes, which hold water and *Stegomyia* larvæ.

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most frequently selects, for breeding purposes, wooden receptacles of all kinds, especially barrels in which there is a thin coating of minute green algæ; next in frequency, all small collections of water in tins and cans of every description, when protected by the shade of foliage from the sun's rays and the heavy rains. In West Africa I have not met with epiphytes growing in any abundance upon the trees; indeed the Bromeliaceæ are conspicuous by their absence. In the West Indies the reverse is the case. When they are present they collectively hold a large quantity of water and support a very large number of larvæ.

As the investigator gains experience, two facts begin to strike him. Firstly, the very small quantity of water, from a teaspoonful upwards, which will suffice as a breeding-place for the *Stegomyia;* and on this account, the smallest odds and ends which may contain water should be examined. Secondly, the immense number of discarded empty sardine tins, milk tins, meat tins, and tin cases of all kinds, which are to be found in all towns opening up to commerce. A veritable tin can invasion extends up from the coast towns into the interior villages. The more traders, the more tin cans; the nearer the more primitive villages are approached, the less become the white traders, and the less, in consequence, the number of discarded tins. In other words, tinned foods of all kinds, oil tins and tin packing cases are most abundant where there are white settlers and traders. This has brought about a condition which immensely favours the development of the *Stegomyia.* The total water-holding capacity of these discarded tins is very great indeed, vastly greater than the inexperienced would at first sight suppose. For the tins are not always obvious when you enter a compound; the fact being that the larger number are concealed amongst the weeds and low bush which invariably is present in the majority of compounds, and on waste places in and immediately around towns and villages on the banks of rivers and streams, and along the seashore. These receptacles

becoming filled by the first shower of rain, and being to a great extent protected from the sun's rays by the overhanging grass and leaves, the water does not evaporate, and ideal breeding-places are thus afforded for the *Stegomyia*. To sum up, the breeding-places of the *Stegomyia* are almost exclusively *artificial*, including all receptacles in which by accident or design, water is stored, and not repeatedly renewed. It is for this reason that all anti-mosquito by-laws must be specifically directed against *stagnant water*, which, in the tropics, has been rightly termed "the great enemy of mankind."

Stegomyia fasciata is usually regarded as a clean-water breeder, and so it is for the most part; occasionally, however, it will be found in very dirty water, in company with the larvæ of various species of *Culex* and *Chironomus*. I have sometimes met with it in drains and marsh-pools in the vicinity of houses.

The striking feature about the *Stegomyia*, as Beauperthuy long ago recognised, and one which every investigator soon appreciates, is its essentially *domestic* nature. It is the true *house-haunting mosquito* of the tropics, and like the cat and dog is never far from the abode of man. I have never seen them in swamps, far away from human habitations.

These features in the life-history of the *Stegomyia* render it easily amenable to control, or even extirpation. It is for that reason that it is very essential in every town to make a precise survey to ascertain where the *Stegomyia* is breeding.

III.—*S* *ia* Surveys

To arrive at an accurate percentage in making a *Stegomyia* survey, I adopt either the house or the compound as the unit. In some countries it is very easy to make the house and its yard the unit. In less advanced countries the towns and villages are divided up into compounds or lots, and in each of these there may be two, four, or six houses. Therefore, if the "lot" or "compound" is taken as the unit, the number of

houses in each should be recorded where possible, but it is not always easy.

In examining a house and its attached yard, the probability will be that the larvæ of the *Stegomyia* will be found in several receptacles. I make a record of this in my note-book, as it bears upon the question of the total numbers of the mosquito; but for calculating the percentage I regard it simply as one house in which the *Stegomyia* is present. In the case of a compound or yard containing more than one house, if I find receptacles in that yard harbouring larvæ, I assume that all the houses are infected, as they are equally exposed to the *Stegomyia*.

The aim and object of the survey is to ascertain the number of houses in a town or village in which, or immediately around which, *Stegomyia* are breeding, and which, therefore, are infested with the mosquito. I have worked out these percentages for a great number of the larger towns in the West Indies and British Guiana, and also in British Honduras, and I am of opinion that they give a very fair idea of the distribution of the *Stegomyia*.

The size of the vessel in which the larvæ are found only affects the question of the total number of the insects. The presence of only two larvæ in a teaspoonful of water, contained perhaps in a snail shell or the broken end of a bottle fastened into a wall, is not less significant than the presence of hundreds of larvæ in a barrel; for it shows how ubiquitous that particular mosquito must be.

IV.—Some Characteristics of the *Stegomyia* and its Larvæ

Having now had several years' experience of this mosquito, I record here those features which appear to me to be most characteristic. The most salient point is the essentially domestic instinct of this mosquito, which is, above all others,

the most "house-haunting" species. I have never found it breeding far from the abode of man, not more than, say, 50 to 100 yards.[1] It is fond of dark situations, breeding preferably in shaded barrels and odd receptacles. Therefore a most favourite site is any water vessel, such as a jar or barrel stowed away in a corner in the kitchen, or in a bedroom.

The mosquito avoids windy places, and therefore selects not only quiet stagnant water, but places where the air is stagnant. As soon as the *imago* emerges from the pupa, it makes for the dark places in the house. It alights preferably on dark or black material. So far as I have been able to judge, I do not think that it flies any great distance at one time, although of course its travelling may be very greatly assisted by the cover of trees or a long line of huts, which would enable it to progress from point to point sheltered from wind and rain. Some observers give 100 yards as its maximum distance of flight. Whatever this may be, however, I consider that it may safely be said that this mosquito does not, as a rule, fly long distances. It seeks cover as soon as it emerges from the pupa, but it may travel from house to house, and is certainly capable of entering ships moored in rivers. In conformity with its house-haunting domestic nature is the fact that it is probably the most common mosquito found on ships; numerous observations in recent years, and the endless records of yellow fever on board ship in the nineteenth century, amply testify to this fact. Given the suitable conditions of freedom from draughts, darkness, and warmth, it can remain secreted for weeks in the holds, galleys, engine rooms, or bunks of a ship. It is for this reason that it is so essential to screen ships which trade in rivers in yellow fever countries, or to insist that they shall be moored several hundred yards from shore.

With regard to the appearance of the mosquito itself, it is very readily recognised. On the wing, it appears grey in

[1] Bouffard places the distance limit at 100 metres; Le Moal gives it as 250 metres.

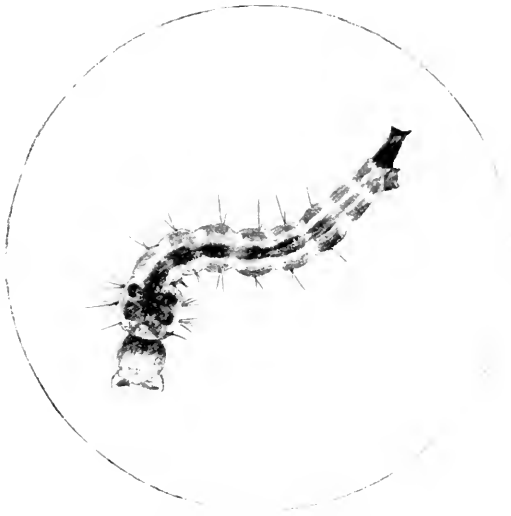


FIG. 39.—"Wiggle Waggle," or Larva of *Stegomyia fasciata* (magnified about 6 times). (After Newstead.)

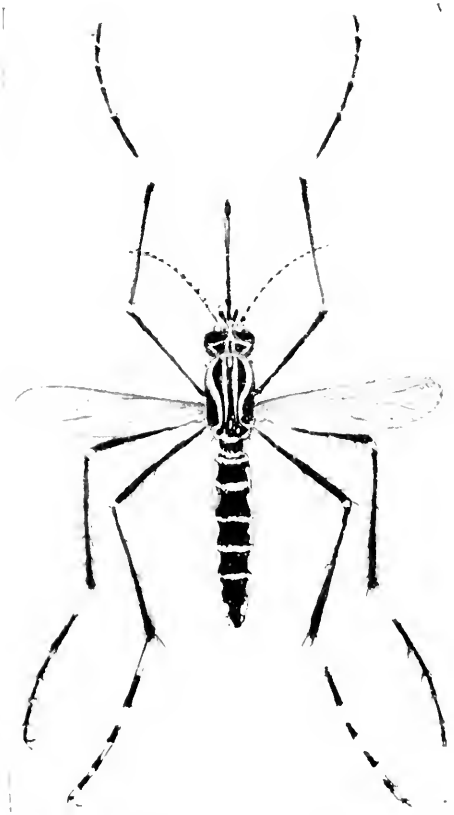


FIG. 40.—*Stegomyia fasciata*, F. (*calopus*, Mg.) ♀, the mosquito which carries yellow fever. (After Newstead.)

To face p. 274.

colour, and it glides from point to point just like a small bit of "fluff." On account of its colour and markings, it is known as the "Scots Grey," or the "'Tiger' mosquito." When it alights, the two long banded white hind legs continually waving up and down are very characteristic.

As I have said above, this species is usually, though by no means always, a clean-water breeder; this habit is doubtless due to the fact that, being essentially a house-frequenting mosquito, it naturally seeks out the water nearest at hand, and this is, of course, the domestic drinking, cooking, and washing supply. It has therefore come to be known as a clean-water breeder; so much so, that its presence in water is taken as evidence of the good quality of the latter. From this belief has sprung a further deduction, namely, that the presence of the larvæ in water is beneficial; for it is supposed that they feed on harmful bacteria, and therefore tend to purify the water. I have made experiments to determine whether there is any truth in this belief, and I have found, as indeed might be expected, that the contrary is the case, and that water containing larvæ becomes much more crowded with bacteria than water without larvæ.[1] The natural food of the larvæ appears to consist of minute algæ.

Stegomyia fasciata bites in the daytime as well as at night,[2] and in my experience it is noiseless. When it has bitten a person suffering from yellow fever in the infectious stage, the virus, whatever its nature, requires twelve to thirteen days to mature in the body of the mosquito before the latter is capable of transmitting the infection. This period is known as the "extrinsic incubation period." When once the mosquito is infected, all evidence points to the fact that it retains the infection for a very long period; three months has been noted,

[1] "The Effect of Mosquito Larvæ upon Drinking Water," Boyce and Lewis, *Annals of Tropical Medicine and Parasitology*, March 1910.

[2] Goeldi (*Os Mosquitos no Para*, p. 103) states that *S. fasciata*, in Para, bites persistently by day, and that while it does bite also at night, such cases are certainly exceptional.

but it is quite possible that the infection lasts as long as the life of the mosquito. It is precisely because of the length of duration of the infection in the *Stegomyia* that it is possible to explain the well-known sporadic outbreaks of cases of yellow fever which frequently occur long after an epidemic is supposed to have disappeared. There is no conclusive evidence that the infected female *Stegomyia* transmits the virus to its eggs and larvæ.

The following entomological notes have been kindly furnished by Mr Newstead:—

V.—Differential Diagnosis of *Stegomyia fasciata*, with Descriptions of two nearly Allied Species. (By R. Newstead, M.Sc.)

Stegomyia fasciata, *F.*

General characters as seen with a pocket lens, × 16:—Head dark, with a distinct double white median line and with white lines laterally and round the eyes; palpi black, white at the tip; proboscis black. Thorax brown, with *two brilliant silvery broad lateral curved lines*, which converge from in front towards the middle of the thorax, these becoming much narrower and continuing parallel to one another as far as the scutellum; in the middle there are two parallel yellowish or whitish lines running the whole length of the thorax. Scutellum very marked, owing to its being completely covered with silvery white scales. Pluræ with several patches of brilliant white scales. Abdomen dark, with white bands on the bases of the segments. Legs black, the femora for the most part pale beneath, in many cases with a distinct white line running from the base almost to the apex and situated on the inner surface, a white spot is also visible at the apex; tibiæ black; the first and second pair of legs with two white bands on the tarsi, the hind pair with five white bands, the last joint being wholly white.

Wings with the veins darkly scaled, the upper fork cell being distinctly longer than the second and its base slightly nearer the root of the wing.

Length 3·5 to 5 mm.; the average length is about 4·5 mm., but very small specimens are often met with.

The following descriptions of two closely allied species of mosquitos may assist the student in determining *Stegomyia calopus*:—

Stegomyia (*Scutomyia*) *sugens*, *Wiedemann*

Characters as seen under a lens, × 16:—Head black, with a thin median whitish line and a white patch on each side; palpi black, white at tip; proboscis black. Thorax dark brown with several scattered whitish scales giving, under the hand-lens, the appearance of a fairly distinct broad median pale line; there are also pale areas laterally. On the anterior portion may also be seen *four silvery white spots*, two on each side and somewhat widely separated. Scutellum white; pleuræ with patches of white. Abdomen deep black, with white bands on the bases of the segments. Legs black; the femora with a white spot at the apex and a distinct white ring a short distance from it, rather pale ventrally. The tibiæ of the fore and mid legs with a somewhat indistinct white band towards the basal half, *those of the hind pair with a very marked band near the centre*.[1] The tarsi of the first two pairs with three narrow white bands, those of the last pair with five broad bands, the last joint being all white.

Wings with the veins darkly scaled, the first fork cell being longer than the second, their bases being almost level.

Length 4 to 5·5 mm.

Stegomyia (*Kingia*) *africana*, *Theobald*

Characters as seen under a lens, × 16:—Head black, with a yellowish spot in the middle; proboscis black; palpi black,

[1] According to Theobald (*Mon. Culicid.* i., p. 301), this tibial band is not present in all specimens.

with the tips white. Thorax black, with *two short glittering lines* directed upwards on the anterior part, and a similar small spot at the base of each wing; scutellum white; pleuræ with several silvery spots. Abdomen dark brown, unbanded, with pale rather indistinct lateral spots on some of the last segments. The last segment with two bright metallic spots; venter with bands of the same metallic appearance as those on the thorax. Legs black; femora with metallic white patches on the inner surface; tibiæ of the first two pairs of legs black, of the hind pair with a white basal band, narrow on the upper surface but much deeper on the ventral surface. Tarsi of the fore and mid legs with two rather indistinct white bands; hind pair with four bands, the third being very broad, and the fourth narrow. The two latter are separated by a small black band.

Wings similar to *Stegomyia fasciata.*

Length 4 to 4·5 mm.

The following synopsis may also assist the student in discriminating these three species:—

	Stegomyia fasciata.	*Stegomyia sugens.*	*Stegomyia africana.*
Head . .	Distinct double median and marginal lines.	Indistinct white median line and lateral white patches.	Yellowish spot in centre.
Thorax . .	Dark brown, with two narrow yellowish-white parallel median lines and two silvery lateral curved lines (Lyre-pattern).	Dark brown, with four silvery white spots on the anterior part, and pale areas due to scattered silvery scales	Black, with two short white metallic lines directed upwards on the anterior portion, and a spot of similar appearance at the base of each wing.
Abdomen .	Dark, with white basal bands and lateral spots.	Dark brown, with white basal bands and lateral spots.	Dark *unbanded*, with pale rather indistinct lateral spots on some of the last segments. The last segment with two bright metallic spots.

	Stegomyia fasciata.	*Stegomyia sugens.*	*Stegomyia africana.*
Legs . .	Femora often with a distinct white line continuing from the base almost to the apex; white apical spot; pale ventrally. Tibiæ black.	Femora with a white ring near the apex and a white apical spot; scattered white scales ventrally. Tibiæ with a white band near middle, more distinct in the hind pair.	Femora with metallic patches on the inner surface. Tibiæ of fore and mid legs black, of hind pair with a white basal band much broader on the ventral surface.
Legs . .	Tarsi of fore and mid pairs of legs with two white bands; hind pair with five bands, the last joint being all white.	Tarsi of fore and mid pairs of legs with three narrow white bands; hind pair with five broad bands,* last joint all white.	Tarsi of fore and mid pairs with two indistinct white bands; hind pair with four bands, the third very broad, and the fourth very small.

* These are deeper than those in *Stegomyia fasciata.*

VI.—Characteristics of the Egg and Adult Larva of *Stegomyia fasciata*

The egg.—This is very elongate, blackish in colour and rather sparsely studded with minute white hemispherical bodies of whitish secretionary matter.

The larva.—One of the marked habits of the larva is that it occasionally swims and wriggles along the surface of the water like the larvæ of certain Anophelines. It has been shown (see p. 282) that it is capable of remaining submerged for relatively longer periods than is commonly the case among the larvæ of numerous other Culicines.

The siphon is about one-fourth of the entire length of the abdomen, and about two and a half times longer than the width at the base. This character is, however, not altogether reliable, as the larvæ of other Culicines possess siphon tubes of similar dimensions.

The distinguishing morphological characters, which can only be determined by the aid of the microscope, are as follows:—

Antennæ smooth, the tuft being represented by a single

short hair; at the apex there is a minute but distinct second joint and a few very delicate hairs (figs. 1 and 2). The labial plate possesses eleven to twelve teeth on each side and a larger terminal one; the base is also symmetrically crenulated as shown (fig. 3). The thorax is rather hairy, some of the hairs arising from four distinct chitinous hooks (fig. 4) situated two on each side of the thorax. On the eighth segment of the abdomen are the lateral combs; each of these is composed of from eight to ten serrated spines, varying in form and also in the number of serrations (figs. 5 and 6). The siphon or pecten spines (figs. 7 and 8) are variable in form and number, there being in the specimen under observation twelve; immediately following there is a triple hair. The last segment is very short, being almost rectangular and bears a number of bifurcated hairs[1] (fig. 9); the papillæ are stout, about one and a half times the length of the segment and with rounded ends.

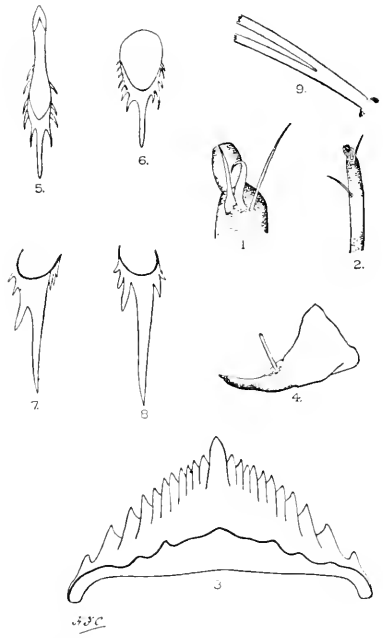
VII.—On the Life-Cycle and Larval Habits of the *Stegomyia*

Period of the life-cycle.—For information on this subject we have to rely chiefly upon the evidence which has been adduced by Goeldi (*Os Mosquitos no Para*). It must be noted, however, that climatic conditions have a marked influence on the developmental cycle of this insect in any given locality, and it may be taken as a general rule that cold will retard any one of the stages either of the ova, larvæ, or pupæ; while a rise in temperature will so shorten the cycle as to bring it within the shortest period possible.

The egg.—Under normal conditions the incubation period in the Amazon region has been found to vary from three to eight days; the average, however, may be taken as three to four days.

Larval stage.—The minimum period, as given by Francis

[1] These are not simple as stated and figured by Weschê (*Bull. Entom. Research*, April 1910, p. 25).



H. F. Carter ad nat. del.

FIG. 41.—Characteristics of the larva of *Stegomyia fasciata*.

1. Apex of antenna.
2. Antenna, showing the tuft, composed of a single hair.
3. Labial plate.
4. Thoracic hook.
5. Serrated spine from centre of lateral comb.
6. Serrated spine from side of lateral comb.
7. Pecten spine from base of siphon.
8. Pecten spine, situated near the apex of the siphon.
9. Bifurcated hair from the ninth abdominal segment.

[*To face p.* 280.

(*Publ. Health and Mar. Hosp. Serv. Rep.*, xxii., 1907, p. 382), in water kept at an even temperature of 80° F., was seven days. In Newstead's record (*Journ. Trop. Med. and Paras*, Liverpool, iv., p. 143) nine days are given, and the temperature that of 23° C. (=73·4° F.). Mitchell[1] states that the larval stage extends over a period of from eight to thirteen days "in fairly warm weather."

Pupal stage.—The duration of this stage varies from one to five days. Mitchell (*loc. cit.*) gives one to five days; Newstead two to three days in a temperature of 23° C.

Adults.—The female lays her eggs in from six to fifteen days after taking the first meal of blood, but Mitchell (*loc. cit.*, p. 148) states that the female may feed two or three times before laying the first batch of eggs. The average number of separate batches of eggs laid by a single female may be given as two to three; but as many as nine batches have been laid in some cases.

The eggs are extruded singly, and the number laid on each occasion varies from twenty-seven to ninety-seven. Goeldi[2] found that as a rule the females died immediately after the final act of parturition, though in two instances females survived for twelve and fourteen days respectively. He also states that fertilised ova may lie latent in the body of the parent for from twenty-three to one hundred and two days, and that the female may lay her eggs at the end of these periods, respectively, after taking a meal of blood. It is evident, therefore, that ovulation is retarded until suitable food is obtained.

It is generally held that the females feed almost exclusively upon warm-blooded vertebrates, and it is usually supposed that such food is necessary for the development of fertile eggs. Goeldi succeeded, however, in inducing females to feed upon honey, a diet upon which they survived for periods varying from thirty-one days to, in one instance, one hundred and two

[1] *Mosquito Life*, p. 148, 1907.
[2] *Os Mosquitos no Para.*

days, though it is evident that such food has a retarding or neutral effect upon ovulation.

Males of *Stegomyia fasciata* also survived on honey for periods varying from twenty-eight to seventy-two days.

These important data point to the fact that in a state of nature, both sexes may, as occasion serves, feed upon the nectar of flowers, though one has failed to find, in the innumerable publications which have been issued regarding the habits of this insect, any evidence that this actually takes place under natural conditions.

Food of the larvæ.[1]—So far as one can gather there is no evidence as to the exact nature of the food of the larvæ. In captivity they feed largely upon amorphous matter and upon the macerated remains of minute crustaceans (*Cyclops* sp., *Diaptomus* sp., etc.), minute fragments of aquatic plants, and occasional diatom and unicellular plants.

The larvæ of *Stegomyia fasciata* have been found in association with those of several other species of mosquitos, notably with those of *Culex fatigans* and to a less extent with *Limatus durhami*, etc. Duprée has made some interesting discoveries regarding the habits of the larvæ, which are communicated by Mitchell in her excellent memoir (*loc. cit.*, p. 147). It is stated that the young larvæ are remarkably tenacious of life under water; they tolerated as much as three hours' submergence, and in some cases were resuscitated after five hours; on the other hand, adult larvæ tolerated total immersion for one and a half to two hours. This habit enables them to feed at the bottom of cisterns of normal depth and to remain submerged for unusually long periods.

Resistance to frost.—There is apparently no direct evidence to prove that the larvæ of *Stegomyia fasciata* can survive at a temperature of freezing point, although they have been found

[1] Boyce (R.) and Lewis (F.)—"Effect of Mosquito Larvæ upon Drinking Water," *Annals of Trop. Med. and Parasit.*, vol. iii., 1910. Experiments were made to determine whether larvæ fed upon and removed bacteria in water. The conclusions arrived at were that the bacteria were increased.

by Francis (*loc. cit.*) at Mobile, Ala., U.S.A., living in tubs placed in sheltered positions during frosty weather, and when the water in vessels exposed to the open air was coated with ice half an inch thick. But it is noteworthy that some large healthy larvæ, which were found in the living-room of a hospital, died when placed in an ice-box where the temperature was about 50°. It is quite evident, however, that this insect can survive at a relatively low temperature, as may be gathered from the statements given by Mitchell,[1] who says that larvæ were found by her in November at Baton Rouge at a temperature as low as 34° F.; and further that pupation took place in water at 53° F. Cold "stiffens" the adults, but one was observed by her to revive afterwards.

VIII.—Viability of the Ova after long Exposure to Dry Atmospheric Conditions

Mr F. V. Theobald[2] was apparently the first to discover that the eggs of this mosquito will remain fertile for a long period, although exposed to normally dry atmospheric conditions. In this instance the eggs were forwarded to England from Cuba in a perfectly dry test-tube. After a period of two months they were placed in "tepid water," and the majority of them produced larvæ. Many of these larvæ survived until the tenth day, and six of them pupated at the end of three weeks, one of which gave rise to a perfectly well-formed female. Unfortunately no details are given as to the temperature in which this experiment was conducted; but it is important to note that the insects were reared in a greenhouse.

Newstead[3] has conducted similar experiments with eggs of this mosquito from material forwarded from Manaos by Dr H. Wolferstan Thomas in the year 1906. The eggs were laid on moist white filter-paper; these were dried in the air and sub-

[1] *Mosquito Life*, p. 148, 1907. [2] *Mon. Culicidæ*, vol. iii., p. 143.
[3] *Journ. Trop. Med. and Parasitology*, Liverpool, vol. iv., p. 143.

sequently placed in a desiccator with chloride of calcium for twenty-four hours, and finally transmitted to England in glass tubes, tightly corked. The following data indicate the results of this experiment:—

September 9 to 11.	Eggs laid at Manaos, Amazon.
October 26	Eggs reached England. Placed in water at a temperature of 23° C. (=73·4° F.).
„ 27	Twelve larvæ hatched during the previous night and one after twelve hours' immersion.
„ 28	Larvæ began moulting.
„ 30	All larvæ completed first moult.
November 4	Larvæ pupated.
„ 7	First *imago*, a male, hatched. This example lived for six days.
„ 8	A male and female hatched.

The breeding-jar was kept in an incubator at an even temperature of 23° C. For the greater part of the time the insects were in complete darkness; but a little light was admitted occasionally during the day.

Summary

Eggs remained dormant and practically dry 45 to 47 days.

Life-cycle

Eggs. Incubation period	6 to 12 hours.
Larval stage	9 days.
Pupal stage	3 days.
Complete cycle	12 to 13 days.

From these data it will be seen that the life-cycle was completed as rapidly as the minimum period given by Goeldi[1] in his classical memoir on the mosquitos of Para. This is all the more remarkable seeing that the larvæ and pupæ were *kept in almost total darkness*, and also in a highly vitiated atmosphere. Subsequent experiments have proved, however, that eggs kept for a longer period than two years lose their vitality and become completely desiccated. Surgeon Francis has also shown that

[1] *Os Mosquitos no Para*, p. 6.

eggs "may remain viable for six and a half months when kept dry." It should be noted, however, that in this instance the eggs were not artificially dried, as was the case with those which were forwarded to Liverpool from Manaos, but were allowed to remain attached to the sides of the jar in which they were laid, above the level of the water, and "set aside in a wardrobe in a room which had no fire in it all winter, and the doors and windows were open night and day." The temperature in which these eggs were kept is omitted also in this case. At the end of the period stated above, eggs placed in a temperature of 80° F. produced larvæ six days after the parent insects had emerged from the pupæ. The complete cycle in this case was shorter by about two days, than that obtained by Goeldi in a tropical climate and apparently under normal conditions.

Peryassu has also succeeded in rearing larvæ from eggs which had been exposed to dry atmospheric conditions for a period of five months; and adds that "this was the maximum time they resisted, and after this they did not hatch."

Boyce brought specimens of larvæ alive to Liverpool, which were collected in Puerto Barrios in Guatemala on 26th October. They were kept in a test-tube exposed to the great variations of temperature which occurred in travelling from Guatemala to New Orleans, New Orleans *via* Washington to New York, and then across the Atlantic to Liverpool. The journey occupied twenty-five days.

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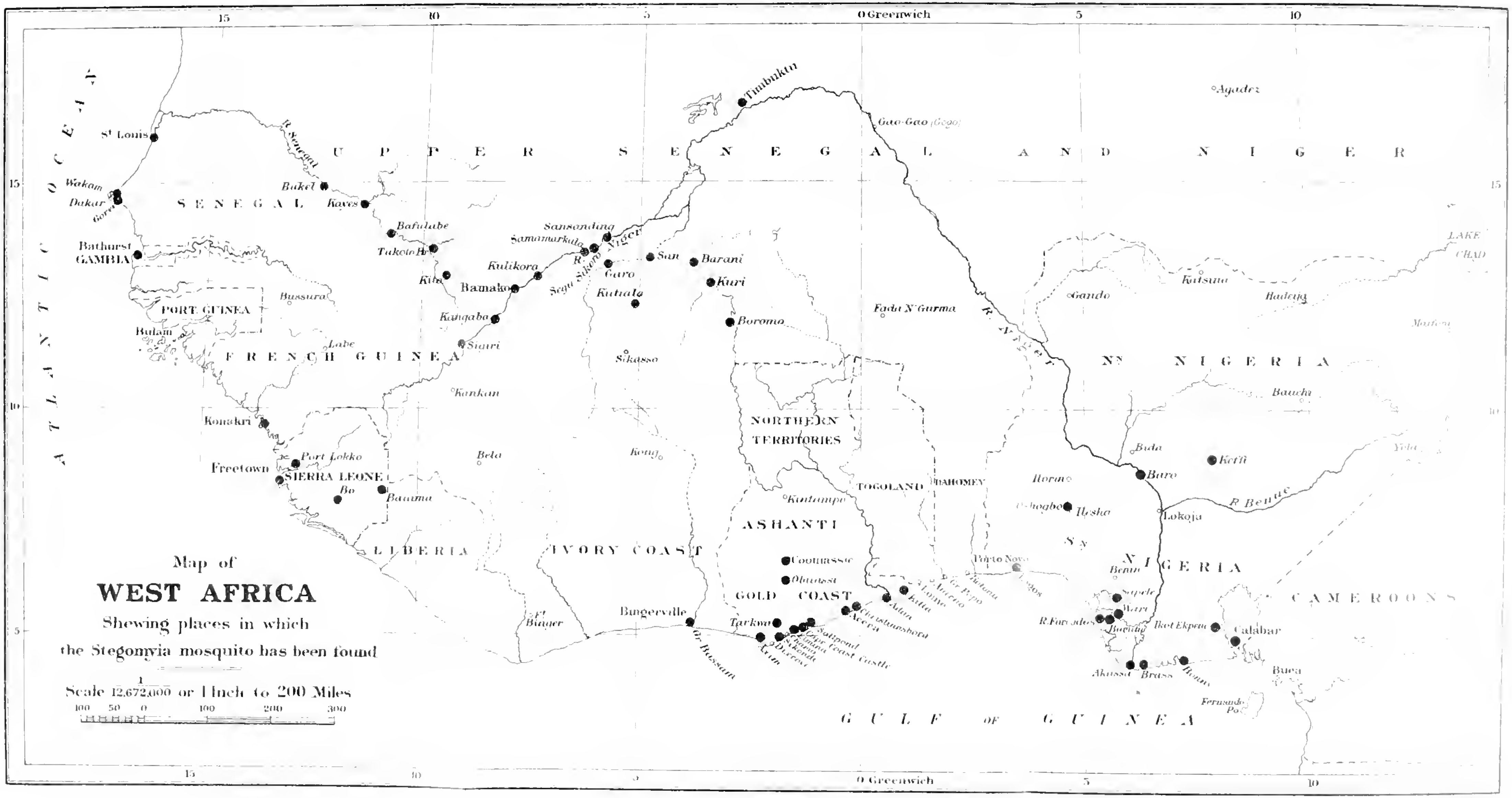
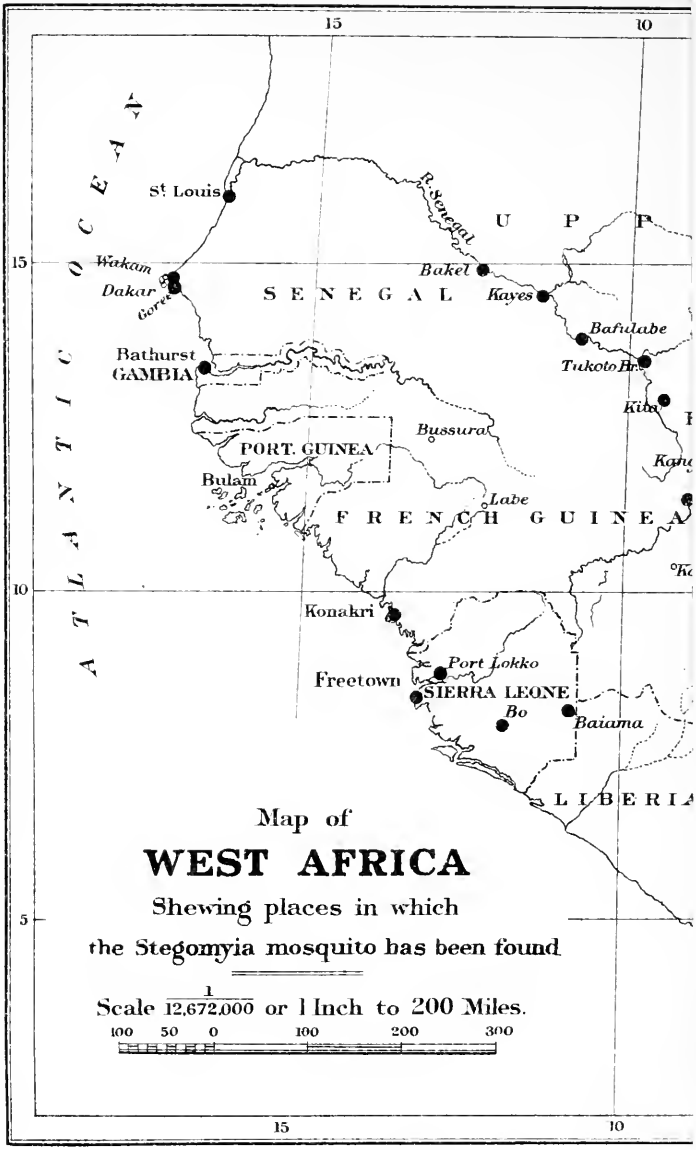


Map of
WEST AFRICA
Shewing places in which
the Stegomyia mosquito has been found
Scale 1/12,672,000 or 1 Inch to 200 Miles
0 Greenwich
ATLANTIC OCEAN
UPPER SENEGAL AND NIGER
SENEGAL
FRENCH GUINEA
PORT GUINEA
SIERRA LEONE
LIBERIA
IVORY COAST
NORTHERN TERRITORIES
ASHANTI
GOLD COAST
TOGOLAND
DAHOMEY
Nn NIGERIA
NIGERIA
CAMEROONS
GULF OF GUINEA
St Louis
Dakar
Goree
Bathurst
GAMBIA
Bakel
Kayes
Bafulabe
Kita
Bamako
Kulikora
Kangaba
Siguri
Kankan
Sansanding
Segu
R. Niger
San
Barani
Kuri
Boromo
Sikasso
Kong
Timbuktu
Gao-Gao
Agadez
Fada N'Gurma
Gando
Katsina
Bauchi
Konakri
Freetown
Port Lokko
Bo
Bauma
Bela
Bingerville
Gr Bassam
Coomassie
Obuassi
Tarkwa
Axim
Accra
Christiansborg
Cape Coast Castle
Saltpond
Kintampo
Porto Novo
Lagos
Ilorin
Oshogbo
Ilesha
Buda
Baro
Keffi
Lokoja
R Benue
Benin
Sapele
Warri
Burutu
Akassa
Brass
Bonny
Iket Ekpene
Calabar
Buea
Fernando Po
Lake Chad


15
10
ATLANTIC OCEAN
St Louis
R. Senegal
U P P
Wakam
Dakar
Gorée
Bakel
SENEGAL
Kayes
Bafulabe
Bathurst
GAMBIA
Tukoto
Kita
Bussura
PORT. GUINEA
Bulam
Labe
FRENCH GUINEA
Konakri
Port Lokko
Freetown
SIERRA LEONE
Bo
Baiama
LIBERIA
Map of
WEST AFRICA
Shewing places in which
the Stegomyia mosquito has been found
Scale 1/12,672,000 or 1 Inch to 200 Miles.
100 50 0 100 200 300
15
10

CHAPTER XVIII

DISTRIBUTION OF THE *STEGOMYIA* IN AFRICA

Stegomyia calopus, according to Otto and Neumann, has been long known in the Senegambia, Sierra Leone, and Slave Coast, and in other parts of the West Coast of Africa. There can be no doubt that the species has been present certainly through the eighteenth and nineteenth centuries, that is to say during the period of recorded outbreaks of yellow fever. Whether it was originally introduced into the West Coast, or whether, like other mosquitos, it is an original native of the coast, it is quite impossible to be certain, seeing that we know so little of the early history of insect life. We do know, however, that the *Stegomyia* could readily have been introduced by any ship, from the sixteenth to the twentieth century, trading between yellow fever countries and West Africa. But the reverse could equally well have taken place, and indeed some authorities, Goeldi for example, regard the West Coast as an original home of the *Stegomyia*. But whatever its origin, it has been long an inhabitant of the West Coast. Evidence is in favour of its having greatly multiplied during the eighteenth and nineteenth centuries, that is to say, during the period of the opening up of the coast of Africa to Western civilisation. As soon as towns increased in size and new ones sprung into existence, the *Stegomyia* was given enormously increased opportunities for breeding, as compared with the primitive periods. No doubt the mosquito, like yellow fever itself, increased with commercial development until comparatively recently, when, thanks to

scientific research, steps were taken to wage war on the breeding-places. We know that the mosquito is present in Togoland, Dahomey, Nigeria, Gold Coast, the Cameroons, Sierra Leone, Gambia, and Senegal. It has also been recorded, so it is stated, in German and British East Africa and in Durban.

In 1901 Major Ross, and in 1904 Dr Prout, drew attention to the prevalence of *Stegomyia* in Freetown. The late Dr Dutton pointed out in 1902 that this mosquito was the species most commonly met with in Bathurst. From recent observations carried out by Graham and others, there is no doubt that *Stegomyia fasciata* is to-day the common mosquito of the Coast towns.

As yellow fever has again this year (1910) declared itself both on the Gold Coast and in Sierra Leone, it is now a necessity, before it is too late, to take steps to ascertain accurately the prevalence and distribution of this pest all over Africa. Yellow fever has penetrated to the Sudan, and it may spread to the East Coast and North of Africa, if not already there in an endemic form.

1. *The "Stegomyia" in Sierra Leone*

Freetown.—In 1901, Ross pointed out the prevalence of *Stegomyia fasciata*, and organised mosquito brigades to do away with it and other mosquitos. Dr Prout also drew attention to its prevalence.

This year, 1910, careful surveys have been made both by myself and Dr Kennan and his assistants, with the following results:—

In the month of August 1910, I made, with Dr Kennan, the senior sanitary officer, a house to house investigation of Freetown, and covered altogether 200 houses with their yards and out-houses.

The streets selected were representative of the various classes in Freetown, including the merchants' quarters, those of the

small traders and Syrians, and the native residential quarters of the well-to-do and the poorest.

The 200 houses and yards contained 87 barrels, 144 buckets, 50 earthenware pots, 66 tin cans, 17 stone jars, 121 odd receptacles, and 27 wells.

Therefore, distributed amongst the 200 houses there were no less than 500 receptacles capable of holding water, and in most instances water was present. Larvæ were found in 88 houses, distributed amongst 174 receptacles out of the 500 potential water-containers; reckoning, however, that 88 houses out of 200 were found harbouring larvæ, the percentage works out as 44 per cent. The streets examined by me were Circular Road, Regent and Goodrich Streets, Wilberforce Street, Fourah Bay Road, Howe Street, Charlotte Street, Little East Street, Charles Street, and Kissy Street.

When I made my inspection of Freetown several previous cleanings up of the town had already been made, and innumerable odds and ends removed. Moreover, as there is a pipe-borne water supply the necessity for barrels and water receptacles is greatly diminished. Nevertheless, as the survey shows, there were yet numbers of unnecessary water-containers and wells which are worse than useless. The larvæ were in almost all cases those of the *Stegomyia;* whenever I was in doubt, I took samples to my office to hatch out. Mr Newstead, to whom I brought back the adults for corroboration, informs me that the 129 specimens were all *Stegomyia fasciata*, with the exception of 2 specimens of *Culex pruinosus*. After examining another series of adult mosquitos bred by Dr J. G. Wood, of Freetown, from larvæ collected in odd receptacles, Mr Newstead reports as follows:—

Kroobay, Freetown, in rock pool, 31. viii. 10:—
3 specimens of *Scutomyia sugens*, Wied.
Pulteney Street, Freetown, in cask, 25. viii. 10:—
2 specimens of *Stegomyia fasciata*, Fab.

Vals Showrooms, Water Street, Freetown, 27. viii. 10, in old tank :—

3 *Stegomyia fasciata*, Fab.

Dandas Street, Freetown, in tin and rock pool, 24. viii. 10 :—

3 *Stegomyia fasciata*, Fab.

George Street, off Upper Brook Street, 14. viii. 10 :—

1 specimen destroyed, 1 *Stegomyia fasciata.*

Percival Street, in tin, 25. viii. 10 :—

2 *Stegomyia fasciata*, Fab., 2 specimens destroyed.

Waterloo Street, in foul tin, 26. viii. 10 :—

3 *Stegomyia fasciata*, Fab.

Note.—*Scutomyia sugens* is very nearly related to *Stegomyia ;* for the distinctions, see p. 277.

As the result of the examination of a third series sent by Dr J. G. Wood, Mr Newstead reports :—

John Lane, Freetown, outskirts, in pool, 9. ix. 10 :—

3 *Culex invenustus*, Theo.

Fergusson Street, Freetown, in rock pool, 7. ix. 10 :—

1 *Pyretophorus costalis*, Loew.

Ascension Town, Freetown, in pot, 10. ix. 10 :—

1 *Stegomyia africana*, Theo.

Kroobay, Freetown, in canoe, 31. viii. 10 :—

1 *Culex* sp. (?), badly damaged.

Deborah Street, Freetown, in cask, 6. ix. 10 :—

1 *Stegomyia fasciata*, Fab.

Soldier Street, Freetown, in old pot, 15. ix. 10 :—

1 *Stegomyia fasciata.*

Adelaide Street, Freetown, in lily pot, 3. ix. 10 :—

1 ♂ *Stegomyia sugens*, 1 badly damaged *Culex* sp. (?)

Vals Showrooms, Water Street, Freetown, in old tank, 29. viii. 10 :—

2 *Stegomyia fasciata*, 2 *Stegomyia sugens.*

Benjamin Lane, Freetown, in cask, 9. ix. 10 :—

3 *Stegomyia fasciata.*

The above data sufficiently indicate that *Stegomyia fasciata* is by far the most prevalent mosquito found in artificial collections of stagnant water. The reports show that in the course of a few weeks the percentage of houses infested has been reduced from 44 per cent. to 7 per cent., and that it is possible without any large expenditure of money to reduce it still further.

Sierra Leone Protectorate.—I was enabled to examine both Bo and Kenema, which are towns in the interior on the railroad. At Bo, in the native town, I found the common mosquito to be *Stegomyia fasciata ;* it was breeding in barrels and in all collections of stagnant water retained in cans, bottles, or odds and ends.

In Kenema, which is a wonderfully clean native town, there is a pipe supply of water, and there are few barrels or odds and ends. I found *Stegomyia*, however, in a large rot-hole in a tree and in a barrel used by builders.

Bullom country.—After making a sanitary tour of inspection of this part of Sierra Leone in February 1909, the medical officer in charge reported :—

"*Stegomyia fasciata* swarms in the grass fields and is also common in the coast towns ; this being so, if yellow fever once got a hold of the country, it would spread rapidly right up to Port Lokko, if not further."

There can be no doubt that the common mosquito breeding in artificial water-containers in Freetown is *Stegomyia fasciata.* This finding is in conformity with what we know of yellow fever in the Colony, and supports the view that the disease is endemic.

2. *The "Stegomyia" in the Gold Coast Colony*

On my arrival in June 1910 in the Gold Coast Colony, I immediately set to work to obtain an estimate of the prevalence and numbers of *Stegomyia* in Secondee, and at the same time I

addressed a circular letter to medical officers throughout the Colony, asking for information under the following heads:—

1. A return showing the number of houses and yards in which *Stegomyia* have been found.
2. The nature and approximate number of water-holding receptacles in each house.
3. Any reports or traditions of cases of yellow fever in the port.
4. The nature and extent of any anti-larval measures which may have been carried out, such as removal of odd receptacles, screening water-containers, oiling, bush-clearing, draining or fish-stocking.

Previous to my arrival, the senior sanitary officer had instituted a vigorous removal of all larva-breeding receptacles from the yards, and the screening of all large water-containers. He estimated that, at the commencement of operations, in all probability every house was breeding larvæ; that in other words, the *Stegomyia* index was 100 per cent.

On my arrival I went through the town with the senior sanitary officer, and the assistant medical officers, and a house to house inspection yielded the following results:—

Eight hundred and forty-two houses were examined, and in 162, larvæ were met with; yielding therefore an index corresponding to 23 per cent.

The following are the figures:—

House to House Inspection of the town of Secondee, from the 29th of June to the 4th July 1910.

	Houses visited.	Larvæ found.	Percentage.
Accra Town . . . . (Dr Muggliston)	267	29	19
Esikadu . . . . . (Dr Fraser)	135	49	37
Lago Side . . . . . (Drs Croley & Goodbrand)	376	86	22
Business Area	64	1	1·55

Stegomyia survey of Accra.

On the 7th July 1910, the principal medical officer reported:—

Number of houses inspected, 729.

Number of houses where larvæ or *imagines* of *Stegomyia* were found, 477.

In conjunction with Drs Garland, Rice, and Beamish, I myself made a house to house inspection, and although the inspectors had already been through the town, nevertheless I found a vast number of breeding-places. I examined 80 houses. In these I found 404 receptacles of all kinds containing water; earthenware pots were most abundant. Of the 80 houses, larvæ were found in 61, the number of infested receptacles being 138. I hatched out many of the larvæ, and Dr Graham confirmed my diagnosis of *Stegomyia fasciata.* In addition to finding very large quantities of empty tin cans, it was noted that there was an excess of bush all over the town, which might effectively conceal other such tins and prevent the water in them from evaporating.

Cape Coast Castle, July 1910.—The report of the medical officer states that "out of 15 European bungalows examined larvæ were found in 13. Larvæ were found in all native houses without exception."

In a hurried examination which I made with the medical officer on the 6th July, I found a very bad state of affairs. In 30 houses, or their yards, there were 32 earthen pots, 18 barrels, and 3 wells, and larvæ were found in 17 of the 30 houses; index 57 per cent. The *Stegomyia* was the prevailing species.

Axim.—The medical officer reported that he had personally inspected 63 houses and found larvæ in 6, in every instance they were those of the *Stegomyia;* index 9 per cent. Another report stated that 1136 houses had been inspected and in 50 larvæ were found; index 4 per cent.

Elmina.—The report of the medical officer gave the *Stegomyia* index as 33 per cent.

Saltpond.—The report of the medical officer stated that 275 houses had been examined and larvæ found in 78; index 28 per cent.

Kita.—The medical officer reported the *Stegomyia* present in all houses; index 100 per cent.

Ada.—The medical officer estimated the percentage of *Stegomyia* as 6 per cent. In an examination of houses which he made he was unable to detect the *Stegomyia* amongst the larvæ.

Tarquah.—The medical officer reported the index as 10 per cent. He also stated that of 136 rooms inspected, in 14 the larvæ or the *imagines* of the *Stegomyia* were found. On the 24th June I visited the town, and in an examination of 40 houses in the miners' village I found *Stegomyia* larvæ in 23. In my opinion the prevailing mosquito in Tarquah is *Stegomyia fasciata.*

Obuassi.—I inspected 100 houses in this district and found 191 receptacles. Larvæ of the *Stegomyia* were found in 55 of the houses; index 55 per cent.

Kumasi.—The medical officer reported that in an examination of 520 houses larvæ were found in 48; index 9 per cent. On the 2nd July I myself made an examination of the 48 houses and found the larvæ of the *Stegomyia* breeding in 8.

3. *The "Stegomyia" in Senegal, Ivory Coast, and Dahomey*

Bouffard pointed out the prevalence of the *Stegomyia* in the Upper Senegal and French Niger territory in 1906. Ribot and Le Moal also drew attention to the widespread distribution of this species in Senegal, and describe the various anti-*Stegomyia* measures. From the history of yellow fever in that country, it is evident that this must be the prevailing mosquito of the towns, and that it is widely distributed, reaching as far as Dioubeba in the Sudan. In a more recent paper Bouffard draws attention to the essentially endemic character of yellow fever throughout Senegal. He has investigated the trade routes, both by road,

FIG. 42.—The Brigade of Dust Carts for the removal of odds and ends liable to act as breeding-places of the *Stegomyia*. Barbados, 1909. (From *Mosquito or Man?*)

[*To face p.* 294.

rail, and water, between Koulikaro and Timbuktu, Bamako and Sikasso, Segu and San, and between Segu and Kutiala, and finds the *Stegomyia* in all centres of population. This observer insists on the essentially domestic character or the mosquito, and that it is not as a rule found beyond a radius of 100 metres from the dwelling houses. Le Moal gives the distance as 250 metres. He concludes that the mosquito is abundantly distributed throughout all the towns and villages, and that it breeds in the various receptacles which I have already described, and also in all puddles near houses after rain. For the safety of the white man, he insists upon segregation and anti-larval measures. Le Moal describes the *Stegomyia* as abundant throughout Senegal, especially at St Louis and Gorée Islands, also in Konakry and at Grand Bassam on the Ivory Coast.

4. *The "Stegomyia" in German African Colonies*

According to Otto and Neumann, the mosquito is present in Togoland, Cameroons, and in German and British East Africa (Ollwig). It is still doubtful whether it is present in German South-West Africa.

The German authorities are fully alive to its significance, and have introduced strict anti-stagnant water ordinances for Togo, in 1910. Much more accurate information is still wanted as regards its distribution and prevalence in the West and East African colonies, and it is to be hoped that definite information will soon be forthcoming.

5. *The "Stegomyia" in Southern Nigeria*

In company with Drs Pickels, Laurie, and Tynan, of Lagos, I made a thorough house to house inspection of 100 houses, selected in representative parts of Lagos, including the poorest, lower middle class and white trading classes. The houses in the poorest part of the town are veritable rat-traps, dovetailed into one another, and abominably overcrowded for the amount of ground space; they are in consequence very dark, and in the

course of all my experiences I have never yet found so many receptacles of all kinds containing stagnant water, or containing such an immense number of *Stegomyia* larvæ. Altogether in the 100 houses and yards there were 489 collections of stagnant water; these consisted of 339 earthen pots, 32 wells, and the remainder of buckets, barrels, and odds and ends. Earthenware pots, therefore, vastly predominated over all other water receptacles. I found larvæ in 252 receptacles; and as the houses are packed closely together, I am of opinion that it is understating the percentage if it is placed at 100 per cent., for unquestionably each house was infected with *Stegomyia*, breeding in its own yard or room, or in the adjacent yard or hut. Specimens of adult *Stegomyia* were present in the houses in far greater numbers than I have seen them in any other part of the Tropical World. The larvæ were in the vast majority of cases those of *Stegomyia fasciata*. To make sure, however, I bred out numerous batches, which were submitted to Dr Graham, who confirmed my diagnosis, and also found *Culex tigripes*, *Culex hirsutipalpis*, and *Culex albovirgatus*, Graham. Dr Graham had already drawn attention to the prevalence of the *Stegomyia*, and its danger from the point of view of yellow fever. He regards this mosquito as the common species of the Coast towns. From the reports made by the sanitary inspectors and furnished to me by Dr Tynan, there is no doubt that *Stegomyia fasciata* is the common mosquito of Lagos, and is breeding in immense numbers in that town. In the Central Province of Southern Nigeria, Dr MacDonald considers it to be the commonest mosquito found breeding in water-containers. From a report furnished by the medical officer of Sapele, the *Stegomyia* index in that locality is 13 per cent. The medical officer at Warri reports that before cleaning-up operations mosquito larvæ were present in every compound. The medical officer at Forcados reports the *Stegomyia* index as being 2 per cent. The senior medical officer of the Eastern Province (including Bonny and Calabar) reported in July 1910

FIG. 43.—The Anti-*Stegomyia* Brigade (Bridgetown, Barbados, 1909) collecting odd water receptacles and rubbish. (From *Mosquito or Man?*)

[*To face p.* 296.

that every native house and compound contained water receptacles, and the larvæ usually present were those of *Stegomyia fasciata.* The medical officer at Opobo also draws attention in a report to the universal distribution of the *Stegomyia* in Southern Nigeria, and the danger arising from this fact were yellow fever introduced.

In conclusion, we may reasonably assume that, like Lagos, the prevailing house mosquito of the towns in Southern Nigeria is *Stegomyia fasciata.* This species has also been found at Brass, Akassa, and Bonny, by Mr J. J. Simpson, and at Degema, by Mr A. H. Hamley.

Antilarval measures.—During this year, 1910, increased energy has been shown in cleaning up the compounds, removing odd receptacles, and in screening tanks and vats; fines have also been inflicted for neglect of these precautions.

Far more systematic anti-mosquito work must be undertaken before it can be said that yellow fever is not endemic, or that there is no danger from importation.

6. *The "Stegomyia" in other Parts of West, East, and South Africa*

Mr Guy Marshall has furnished me with the following list of additional places where the *Stegomyia calopus* has been reported [1]:—

NORTHERN ANGOLA: San Salvador (*Dr Mercier Gamble*).

THE ISLAND OF PRINCIPE (*Dr Ansorge*).

BRITISH SOMALILAND: Zeila (*Dr A. J. M. Paget*).

BRITISH EAST AFRICA: Mombasa (*J. D. M'Kay*).

NYASALAND: Somba (*Dr H. S. Stannus*) and Blantyre (*Dr J. E. S. Old*).

NATAL: Durban (*Dr Christophers*).

[1] [These records are based on specimens contained in the British Museum collection or received by the Entomological Research Committee. Mr M'Kay notes that at Mombasa this mosquito is common and troublesome.—GUY MARSHALL.]

No information, however, is forthcoming as regards the distribution and abundance of the insect. It is to be hoped that careful reports will be drawn up upon these important points.

Destruction of *Stegomyia fasciata*

In the preceding pages I have sketched the wide distribution of the *Stegomyia* in West Africa, and its significance is obvious, for it explains why, for the last hundred years at least, yellow fever has been common on the coast. It explains the outbreaks of yellow fever for this year (1910), and it warns us that if West Africa is to be still further developed in connection with its great potential mining, oil, and other industries, it will be necessary to eradicate the *Stegomyia* or face the certainty of the disaster and panic which will ensue from outbreaks of yellow fever.

The existence which I have endeavoured to show of the *Stegomyia* in overwhelming preponderance in the Coast towns and in many of the interior towns of West Africa, also goes far, in my opinion, to explain the very high mortality rate amongst Europeans in the past. This high death-rate has as a rule been attributed to malaria, in one or other of its many forms. But I think, not only from the evidence of the wide distribution of the *Stegomyia*, but also from the evidence of hospital case-books and the experience of both English, French, and German medical authorities, that a very considerable proportion of the death-rate may, with a high degree of probability, be ascribed to yellow fever; that, in fact, the disease has been often overlooked. In other words, when the yellow fever cases are taken out, the malaria death-rate on the coast is not unlike the malaria death-rate of all other malarial countries; and it seems probable that the deadly reputation of West Africa has to a large extent been due to mistaken diagnosis, to neglect of fumigation after cases of yellow fever, and above all, to the absence of any attempt to reduce the vast numbers of the

FIG. 44. A Heap of Odd Water Receptacles, collected out of yards. Barbados, 1909. (From *Mosquito or Man?*)

[*To face p.* 298.

Stegomyia. Therefore, if West African development is to proceed in security, it is necessary to lose no time in organising methods to combat this mosquito.

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PART VI

PROPHYLAXIS

FIG. 45.—Using Steam from the Fire Engine to destroy Mosquitos in Sheds, Outhouses, and Railway Carriages.
New Orleans, 1905.

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CHAPTER XIX

PLAN OF CAMPAIGN

THIS subject can best be studied from the two standpoints as follows:—

(*A*) *Where there is reason to believe that yellow fever is endemic.*

1. Segregation of non-immunes, partial or complete, town planning.
2. Screening:—
   - The bed.
   - The verandah.
   - The house.
3. Systematic mosquito destruction.
   - Removal of breeding-places.
   - Screening of water cisterns.
   - Oiling, infliction of fines.
   - Drainage.
   - Bush clearing.
4. Education.
5. Quarantine administration.

(*B*) *Where yellow fever has broken out.*

1. Removal of all non-immunes outside the infected area. Deflection of the traffic outside the infected areas.
2. Provision for the isolation of all cases, or suspected cases.
3. Provision for contacts.
4. Early notification.
5. Fumigation.

6. Emergency mosquito measures.
   - Removal of receptacles.
   - Oiling.
   - Screening.
   - Drainage.
7. Education.
   - Lectures.
   - Meetings.
   - Drainage.
8. General organisation of the medical forces.

A

1. *Segregation of Non-immunes*

Segregation or protection of those who arrive for the first time in any country where yellow fever is endemic, is a self-evident method of protection. It would be barbaric or uncivilised not to adopt this fundamental method of self-protection.

If yellow fever is endemic, the virus must be kept up by the inhabitants whoever they may be—blacks, Indian-Spanish, or Creoles. Therefore the first measure of safety is to live in some quarter away from the inhabitants.

The nature of the new arrival's business may be such as to make it necessary for him to transact it during the day in the midst of the houses of the inhabitants. During the night, however, he can usually find sleeping quarters at a little distance away. It is a well-known fact that sleeping outside the native town does confer a very great measure of safety.

Therefore the rule should be wherever possible, to reserve one portion of the town for special quarters for the white traders, in which reservation no natives should be allowed to dwell.

In the future all West African Coast towns should be planned out with this segregation in view.

Failing complete segregation, the next thing to do is to provide sleeping accommodation outside the native town. This

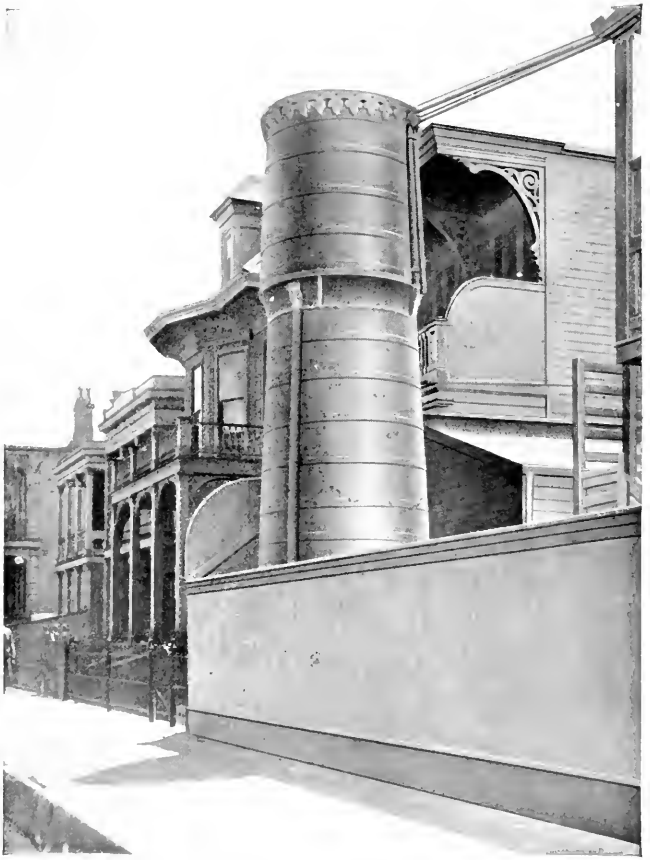

FIG. 46.—A very carefully screened House Water Cistern. New Orleans, 1905.

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can in a very large number of instances be readily arranged. Officials and merchants must keep in far better health if they can sleep away from their business premises in a place secure from infection and exposed to healthy breezes. The small amount of time occupied in going to and fro to their sleeping quarters, is more than counterbalanced by increased vigour and aptitude for work.[1]

2. At the present day it is hardly necessary to insist upon the mosquito net; it is now universally adopted, and the man who does not do so is regarded rightly as a crank.

Every bedstead in the tropics should be provided with a properly made net, one that is an absolute protection against the mosquito. Personally, I prefer the form erected over a square frame under which a bedstead and a small table can readily fit. Very excellent, however, are the small mosquito nets fitted to camp beds. They are exceedingly comfortable, and with one, one can travel anywhere and sleep with the assurance of absolute safety from infection. There is no doubt that during the last ten years the use of the mosquito net has become universal, and it is to this reason that we must ascribe in very large measure the decrease of both malaria and yellow fever.

Sometimes it is possible to screen the whole of a house, or portion of the verandah. If house screening is resorted to, it must be very carefully looked after to see that no holes are formed, and that mosquitos cannot possibly gain access inside. If this is not done the screening may turn out worse than useless. The same measure of security can be obtained by the use of a simple portable screened chamber, which one can erect in a few minutes over a sofa or couple of chairs and a table. It is a great comfort to be able to rest in the afternoon free from the risk of infection, that is, if one's house is near native houses.

I wish, however, to emphasise, that the greatest security will

[1] For many years sleeping out of town was the rule in Rio and Santos during the yellow fever period of a few years ago.

be attained by placing the house at a safe distance from the native town, on some advantageous site, free from bush and freely exposed to every breeze.

3. *Mosquito Destruction*

This is a measure which strikes at the root of all the evil, and affords security to all alike, be they white, black, or yellow-skins. Fortunately, public opinion has begun to recognise that this is a comparatively inexpensive and practical measure, and shows a return almost immediately in increased comfort and health. It is, moreover, a measure in which everyone, man, woman, and child can co-operate. The work divides itself into the following divisions.

(1) Removal of odd breeding-places, such as tins of all kinds, bottles, calabashes, broken and disused crockery, etc., etc. This is work which everyone can help to do. To help it effectually the Health Authority must employ more sanitary inspectors and more dust carts. But it is for each householder to see that the rubbish is removed to a place where it cannot do any harm.

(2) *Screening.*—If the town is not fortunate to possess stand pipes, then it is very essential to see that all proper water receptacles, such as barrels and cisterns, are suitably protected with wire gauze, or otherwise made mosquito proof. This is absolutely essential, and if not seen to, the traders' houses often become the worst offenders.

(3) *Oiling.*—Of course the better plan to deal with pools and defective storm water drains is to fill them in and do away with them; but to quickly deal with them, the simplest plan is to sprinkle every week a little kerosene oil over them. The householder should do this; it is most effective and cheap.

4. *Experiments upon the Culicidal Action of Kerosene Oil*

The most economical, least dangerous, and most readily procured of all culicides, are the various preparations of

FIG. 47.—The Oiling and Screening Gang, ready to start for the day's work. New Orleans, 1905.

FIG. 48.—The Screening Gang at work securing the Cisterns. New Orleans, 1905.

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petroleum. This oil can be used on a large or small scale, and it can be either sprayed or poured on the water. For all stagnant collections of water in pools or lagoons it is excellent, and its culicide action remains long after such collections of water have been treated. It must be recollected that under a powerful tropical sun, the oil evaporates in a comparatively short period, and that therefore the treatment must be renewed every few days. I made some experiments this year in order to determine how soon the effect of the oil passed off. My experiments consisted in placing in a large tub of water, of one square yard superficial measurement, a number of the larvæ of the *Stegomyia*. Then I poured on to the surface 1 oz. of kerosene oil, and exposed the tub either in the bright sunshine, or kept it under shade. I timed the starting of the experiment, and the time when the larvæ ceased to move.

Experiment I. — Surface covered with 1 oz. of oil and exposed to powerful sun.

Stegomyia larvæ placed in water at 12 noon; at 3 P.M. all larvæ dead, and most of the oil evaporated.

Experiment II.—To determine rate of evaporation. At the end of three hours the oil had evaporated, leaving but a faint smell and producing only a slight oil stain on blotting-paper.

Experiment III.—Half an ounce of oil poured on the water. Larvæ placed in the water at 12.30 P.M.; and all dead at 1.30 P.M.; at 5.25 P.M. the oil had evaporated, with the exception of a faint odour and very slight iridescence. I then placed fresh *Stegomyia* larvæ in the tub without adding any additional oil, and at 6.30 the larvæ were dead. It appears that the small quantity of dissolved oil has a culicide action.

Experiment IV.—Half an ounce of oil poured on to the surface of water in the tub, and larvæ placed in the water at 1.40 P.M. in bright sunshine; at 3.15 P.M. the larvæ were very sluggish but still alive; the petroleum had evaporated from the surface. These few experiments show that in bright sunshine the oil film rapidly evaporates, but that the oil which remains

in solution still continues to exert a culicide action. Therefore it may be concluded that 1 oz. of oil to the square superficial yard is sufficient to kill mosquito larvæ, but that the water should be treated once a week in order to avoid all risk of the survival of larvæ.

(4) *Drainage and filling in.*—Every householder could assist the local authority in this respect. He could look after the proper grading of his own garden or yard, and see that all depressions are filled in. That his roof gutters do not sagg and that if they do, that holes are drilled in them at intervals of about 3 feet. It is the duty of the local authority to look after the roads and the side drains. The proper grading of road levels and curves, and the best forms of storm water drains to employ in the tropics are matters now well understood (see *Health Progress in the West Indies*).

I would advise in those cases where towns are about to embark upon drainage schemes, to first ascertain what can be done at once, for a very little outlay, in the way of getting rid of all water-holding rubbish and redundant water storage vessels and bush. In other words, not to embark upon elaborate schemes until it is seen what can be done by a little individual enterprise upon the part of each inhabitant.

If finally a scheme is decided upon let it be comprehensive for both the present and future requirements of the locality, and so planned that it can be undertaken in parts as occasion arises.

The costly mistake is too often made of putting in drains which in a few years not only do not drain, but actually form miniature lakes. It must also be remembered that if drains are constructed they must be kept in proper repair, and due allowance must be made for that purpose in the estimates.

Canalisation.—In the case of mangrove swamps, very often the most efficient and economical treatment is to canalise them (see my Honduranian Report). By this means the water is deepened and the breeding of fish is favoured. Sometimes also by these means it is possible to get the sea-water to enter; in

FIG. 49.—Old Style of filling in a Swamp by means of Hand Labour. Gold Coast, 1910. (From *Mosquito or Man?*)

FIG. 50.—Modern Method of filling in a Swamp by means of a powerful Sand Pump. Lagos, 1910.

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this case a daily tidal rise and fall is maintained, which acts as an excellent drainage system (see British Guiana in *Health Progress*). By canalisation it is also possible to make the margin of the swamp sharp and free from weeds. It will be remembered that it is at the margins of swamps where mosquitos breed.

Filling in.—By this method the nuisance is altogether abolished. In my Honduranian Report in 1905, I advocated the use of sand pumps for this purpose. I have since seen them at work filling in swamps in Southern Nigeria, and there is no doubt that it could in time alter the health conditions of many places in West Africa and elsewhere. It is efficient and cheaper than hand labour.

It must not be forgotten, in filling in lagoons and swamps, that as they are the natural drainage places of the locality and therefore at the lowest level in the locality, that provision must be made to take away the water which previously drained into the lagoon. This can readily be done by reserving an ample central drain in the middle, and into which by means of weep holes the water can readily drain from both sides.

Fish stocking.—Following upon the natural immunity from malaria which Barbados has experienced, owing to the fact of the presence of small fish, known as millions, *Geradinus pœcilloides*, fish stocking has come into practice and is a useful auxiliary method of defence.

Anti-mosquito ordinances and infliction of fines.—In my work on *Health Progress and Administration in the West Indies*, I have brought together the laws which have been framed against mosquitos in the West Indies. In the present volume I have summarised some of those which are in use in West African Colonies (see Chapter XXI.). There is not the least doubt that these laws, when enforced, bring about an immense improvement; and it is further a fact that if these laws are rigorously enforced without favour against rich and poor alike, that there is little or no opposition from the public. The immediate returns, in the

form of greater comfort and health soon convinces the public that these measures are wise and for the general good of the community.

Pipe-borne water supplies.—Where a community can introduce a pure wholesome water supply laid on to each house and to stand pipes in the roads, one of the first steps in the getting rid of mosquito-carried diseases is taken, for a tap water supply does away with the necessity of storing any water upon the premises; therefore there is no question of screening cisterns or barrels. Furthermore and of still greater importance is the getting rid of the horrible surface drinking wells which are found all over the tropics, and which not only breed but become contaminated with all kinds of pathogenic bacteria and parasites.

Bush clearing.—Regular systematic bush clearing is an absolute essential in any tropical town, overgrown bush causes as much harm as puddles and marshes. It prevents proper evaporation and drying of the ground; it shuts out breezes; it gives cover to all kinds of tins and rubbish which breed mosquitos, and it serves as cover for mosquitos. Therefore bush clearing regulations should be inserted in all anti-mosquito by-laws. In getting rid of bush, each householder could assist. At the present time all over the tropics, yards and gardens are allowed to become choked by useless weeds and bush of all kinds. The Health Authority should rigorously insist upon bush clearing.

4. *Education*

Educational prophylaxis is rightly regarded in all civilised countries as a very great factor in the progress of sanitation. Real progress is only made in proportion to the intelligence and progress of the community. Therefore every endeavour should be made to educate the people by training the children in the schools, by teaching the adults by means of popular lectures and lantern demonstrations. The support of the clergy of all denominations should be secured, as they have an excellent machinery ready to hand for teaching the public.

FIG. 51.—Too much Bush. This photo shows very clearly the great disadvantage of surrounding the dwelling-house by excessive bush. Georgetown, Demarara. (From *Mosquito or Man?*)

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5. *Quarantine Administration*

Whether yellow fever is endemic or is only liable, owing to the abundance of *Stegomyia*, to break out if the disease were imported, quarantine regulations have a very practical value. A quarantine station both for the isolation of yellow fever cases and for the observation and detention of suspects should be provided. A ship calling at a port where yellow fever is endemic, has non-immunes on board, and these must be protected on the principle of segregation.

Non-immunes must not be allowed ashore into the town, and the ship must be moored at a safe distance from shore so as to avoid *Stegomyia* coming on board and infecting the non-immunes. Lighters coming alongside must be previously treated with kerosene, and if closed ones, *fumigated.*

Every passenger coming on board should be supplied with a health certificate by the medical officer on shore.

B.—In those Cases where Yellow Fever has broken out

Protection of the Non-immunes

The first precautionary steps to take when yellow fever breaks out in a locality, is to secure the safety of the non-immunes. This is done by removing them to a place outside the infected area; it is in fact emergency segregation.

A temporary camp has to be erected if there are no existing buildings which can be used for the occasion. If the epidemic shows signs of progressing, it is imperative for all non-immunes to live both night and day in the segregation camp. In less severe cases sleeping away from the infected area at night-time affords a great measure of security. The plan of removing at once the non-immunes has been adopted with signal success this year, 1910, at Secondee, and also at Bamako in 1906.

Deflection of the Traffic

In order to interfere with trade as little as possible, it may be advisable to deflect away from the infected town or quarter all through traffic. A new temporary port can be established for the steamers, and a temporary railway terminus can be fixed upon, so as to avoid running the trains or passengers into the infected town.

This plan was adopted with great advantage at Secondee (see Chapter XXIII.). Goods should be packed during the day-time in sheds freely exposed to currents of air. The greatest care should be taken that the *Stegomyia* is not breeding in the vicinity, and from time to time the sheds should be steam fumigated. Having made arrangements for the safety of the non-immunes, the next step is to strictly supervise the care of the infected, so that they cannot become a source of infection to others. For this purpose it is necessary to establish screened emergency wards, rooms or an hospital in a convenient place to which all cases or suspected cases can be removed and carefully nursed and watched. In those cases where it is inadvisable to remove the patient, the room in which the patient is kept must be most carefully screened, and the remainder of the house must be fumigated.

When a case of yellow fever is reported, or where any suspicious cases come to light, the invariable rule is to at once place the patient under a mosquito net, so as to avoid any further chance of infection. If an emergency hospital is established, it avoids a great deal of worry and loss of time. The patient can be removed at once under a mosquito net or in a screened ambulance, and then the house in which the patient resides can be thoroughly fumigated in every part; this is very difficult to accomplish if the patient remains in the house.

Upon the thoroughness in detecting early cases or suspected cases, and in applying fumigation, will depend the cutting short of the epidemic.

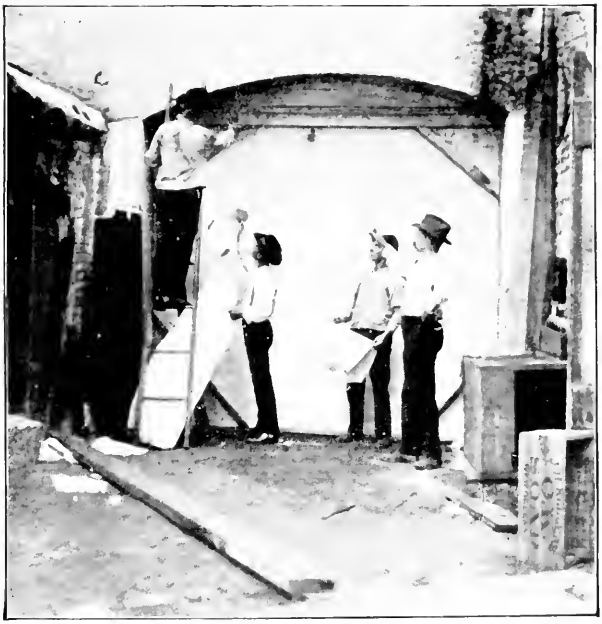

FIG. 52.—The Papering Gang at work, rendering rooms tight before Fumigation. New Orleans, 1905.

FIG. 53.—A Carefully-papered Outhouse, ready for Fumigation. New Orleans, 1905.

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Provision for Contacts

It is often a matter of great practical difficulty to prevent relations and visitors getting access to a yellow fever patient. Yellow fever is constantly spread by non-immunes visiting a patient, and then getting bitten by an infected *Stegomyia.* For the same reason wakes must be strictly forbidden. When a patient is suspected of having yellow fever, none but the nurse and medical attendants should enter the sick room.

When a person becomes infected with yellow fever in a particular house, it is reasonable to suppose that others also in the house may have been bitten by the same infected *Stegomyia.* Therefore, all residing in the house, or who have visited at the house, should be kept under careful observation until the incubation period is well over. Provision should therefore be made for contacts at an early stage in the epidemic.

Early Notification

Nothing in yellow fever prophylaxis is so insisted upon as this, nevertheless, and in spite of all warnings, nothing is so much neglected. Not an epidemic passes without a case of yellow fever being declared in the third, fourth, or fifth day of the illness, that is to say when all the harm will have been done owing to failing to rigidly isolate during the infectious period, and failing to exterminate infected *Stegomyia.* Medical officers appear always to be reluctant to alarm the friends of the patient by declaring yellow fever. It is, however, in the very best interests of the patient and of the community to take no risks from the very commencement of the illness.

Fumigation

It is only the trained medical man who can thoroughly appreciate the significance of complete fumigation. Laymen cannot understand the risks from the bites of infected *Stegomyia*, nor the ease with which infected *Stegomyia* hide in the house in which yellow fever has occurred.

Therefore fumigation must be carried out under the direct supervision of a medical officer. Sulphur is the common fumigating agent used all over the world, and is one of the very best materials. The fumes can be generated in a pot, by burning sulphur, or in a Clayton apparatus. Sulphur vapour, however, tarnishes all brass and copper work and all bright steel.

The following are the directions for the use of various fumigating materials.

1. *Sulphur.*—Allow 2 lb. of sulphur to 1000 cub. ft. Use 2 pots, place them in a pan containing 1 in. of water to prevent damage, and set fire to the sulphur by means of spirit. *Duration.*—Three hours.
2. *Pyrethrum.*—Allow 3 lb. to 1000 cub. ft. and divide amongst 2 or 3 pots, using the same precautions as with sulphur. *Duration.*—Three hours.
3. *Camphor and Carbolic Acid.*—Equal parts camphor and crystallised carbolic acid are fused together into a liquid by gentle heat. Vapourise 4 oz. of mixture to each 1000 cub. ft.; this can be done by placing the liquid in a wide shallow pan over a spirit or petroleum lamp; white fumes are given off. To avoid the mixture burning, the fumes should not come in close contact with the flame of the lamp. *Duration.*—Two hours.

It must be borne in mind that it is by no means easy to seal effectively a bungalow in the tropics, and much more difficult to seal a thatched hut of a native. All that can be said is that it has been done and can be done with patience and plenty of assistance. Plenty of paste and all the paper that can be found in the town should be brought together, and every chink closed. Failing paper, recourse may be had to cheap cotton, cloth, or material of that nature. Sometimes sails or deck awnings can be used to wrap round and cover a small hut or a verandah. The details of sealing, however, must be left to the intelligence and energy of the medical officer in charge. The medical officer should have a free hand, and the funds at his disposal should

FIG. 54.—Generating the Sulphur in the Clayton Apparatus. Secondee, 1910.

FIG. 55.- Photo showing the Clayton Sulphur Apparatus di-infecting a house in Secondee, 1910.

[To face p. 314.

not be stinted. It is far more economical to commence at once to face the worst and to prepare accordingly. Yellow fever prophylaxis cannot be scamped.

Emergency Mosquito Measures

The outbreak of yellow fever is always a good reason to redouble the efforts to get rid of mosquitos. For this purpose it is the rule to form *clean-up clubs*, and to organise *mosquito brigades*, and to set apart *clean-up days*. In this way a yellow fever epidemic is a blessing in disguise, for a town is far purer after a visitation of this disease.

For the same reason public lectures can be increased, hand bills and pamphlets circulated and affixed in all public places. At this stage the clergy can be of great use, for as a rule, they are amongst the earliest to suffer by the disease, and are therefore anxious to help.

An outbreak of yellow fever is also a reason for organising the medical forces of a district, and the opportunity should not be lost of bringing all together to wage war on the mosquito.

CHAPTER XX

EARLY NOTIFICATION AND NOTIFICATION FEAR

FROM the symptoms which I have described under "experimental yellow fever," "inflammatory fever," "bilious and remittent fever," it is very clear that yellow fever must be a disease which, in its milder and more common forms, is not easily recognised. Indeed Carroll states that "their experiments show that genuine yellow fever may be so mild in character that no man, no matter how extensive his experience may have been, would dare to diagnose it as such unless he knew the disease to be prevailing at the time."

The young medical officer should therefore remember that his professional skill need not be criticised should he diagnose as a case of yellow fever one which subsequently proves to be another disease. In the very best interests of the community, it is much safer when there is the least doubt to regard the case as one of yellow fever and treat it as such.

In West Africa in future far greater hesitation will be necessary before a case is assumed not to be one of yellow fever. The outbreak in the year 1910 did not come from the clouds or have a spontaneous origin, it must have been preceded by other cases which passed unobserved, and this is happening all the time—it is universal experience in endemic countries. In other words, the medical man must in the future ever have before him the possibility of yellow fever as long as the *Stegomyia* is abundant. This is a golden rule, and the

presence of the *Stegomyia* should be made the test in all doubtful cases.

In all civilised countries liable to yellow fever, it has been held that it is the bounden duty of the medical officer to notify all suspicious cases; indeed "suspicious of yellow fever" is regarded as a notifiable diagnosis in Cuba. It is the only way to attain security, and ultimately to get rid of the disease.

It is a melancholy fact to have to record, that early diagnosis in the case of yellow fever, and indeed of other infectious diseases, is the exception rather than the rule. The young medical officer is unquestionably apt to shrink from making a diagnosis which will focus attention on him, and perhaps bring him into ridicule should his diagnosis not prove correct.

All young medical officers should, however, remember that they have the greatest living authorities on their side upon the question of having no trace of hesitation in diagnosing a case, when there are reasonable grounds for suspicion.

I have repeatedly been told by medical men, after the passing of an outbreak of yellow fever, that had they at the beginning of the outbreak the knowledge of the disease which they subsequently possessed, they would have diagnosed far more cases at the commencement of the outbreak than they did; and on going over their case-books they were able to point out very numerous instances of mistaken diagnosis. In discussing the outbreak of yellow fever in Barbados, 1909, which I was called upon to investigate, I wrote as follows:—

The Diagnosis of the Early Cases

"The question naturally arises: Do the notified cases in any epidemic represent the true total of all cases? In my opinion, the answer here is the same that has to be given in all these outbreaks: that in all probability there were many other cases which were not correctly diagnosed. There was a divergence of opinion amongst the medical men as to whether the cases were yellow fever or not, and the press was not slow

to make use of this want of unanimity during the outbreak; cases of illness were diagnosed as gastric influenza, epidemic jaundice, malignant jaundice, and dengue. Of course these diagnoses were made in absolute good faith by competent medical men; but for all that, there may have been mistakes, and some of the cases might have been yellow fever. I gathered, from my conversations with the medical men, that this was indeed probably what did occur. I mention these difficulties because they invariably happen in practice, and because they demonstrate the absolute necessity of a medical head in the Colony, who, by virtue of his position, can, when he deems it advisable, bring about the information of a friendly consultative committee of the medical men of the Colony to act as a jury in medical questions affecting the welfare of the Colony."

Notification Fear

On looking over the history of yellow fever in any country, it becomes clearly obvious that great confusion has arisen over what should be called yellow fever or not. Countries where yellow fever is old established in its endemic form have argued that yellow fever is only a disease of the newcomer, and that as far as the residents are concerned, they do not get it; at least in that particular severe form.

Some countries like Cuba would not diagnose yellow fever unless black vomit was present. In other words, it became customary in some parts of the tropical world not to diagnose yellow fever unless the exceptional and severe symptoms were present. The mild, commoner form passed under a variety of names as we shall see later. It was essentially of the nature of an "acclimatising fever," peculiar to the place, not imported. Probably it was in those days assumed to be miasmatic, and produced by the altered conditions in which the new arrival found himself. This fever was looked upon as the necessary tax for coming to a tropical country. Those who got it became "salted" and "old coasters," and were those who subsequently

openly ridiculed any idea that yellow fever could possibly be present amongst them. From time to time, however, when large bodies of newcomers arrived, epidemics did occur which could not be concealed, and the narratives of the survivors of the terrible symptoms, fostered an exaggerated idea of the very deadly and horrible nature of yellow fever. Yellow fever soon became to be regarded as an awful and mysterious disease, which paralysed enterprise and commerce. As a natural result of this there arose stricter quarantine regulations, and as a consequence of this stricter supervision, there arose a natural disinclination amongst merchants and others to declare yellow fever if they possibly could avoid doing so.

This reluctance has been universal, and exists at the present day.[1] There appears, as it were, to have been a universal conspiracy to deny the presence of yellow fever, both from the point of view that it was of no account, only attacking the new arrival; secondly, from the fact that quarantine authorities were insisting on rigid regulations; and thirdly, that, as its name caused such a panic amongst the public, the better and more business-like policy was to say nothing about the disease, either deny its existence, or return it as some other disease, or make light of it. I am convinced from many investigations and from the careful analysis of innumerable outbreaks of yellow fever, in different parts of the world, that this attitude has prevailed.

The evidence furnished from West Africa shows that it certainly exists there.

Looking at yellow fever in the light of modern discoveries, the dread of declaring the existence of this disease appears foolish in the extreme, as one of the greatest authorities on yellow fever has said there ought not to be any more hesitation about its notification than about a case of measles. The

[1] Bérenger-Féraud especially blames the British race and speaks of the invincible reluctance of the English to acknowledge that they could by any possibility have yellow fever in their colonies.

abnormal fear produced by its diagnosis is only the result of ignorance. To-day the means of controlling and stamping out this disease is if anything more easy of execution than in the case of measles.

In addition to the admittedly great difficulty encountered in making a diagnosis, the physician has often to put up with difficulties of another sort, namely, these brought about through prejudice or commercial interests.

It seems very strange that there should be obstacles in the way of the diagnosis of yellow fever; but such is the case. In all the outbreaks which I have had to investigate I have encountered them, and writers upon yellow fever have, time and time again, drawn attention to this point.

I encountered it in a particularly gross form in Barbados last year (1909), and I was the means of drawing attention to it, and I venture to hope that I may have been able to stop it. It arises primarily from the press and mercantile community, and these agencies, in their turn, slowly but surely influence those in authority as well as the younger members of the medical profession.

There is an absolute notification fear. I have known personally medical men who have been persecuted because they dared to notify yellow fever. I have examined the correspondence in other cases, where the medical officer making the diagnosis has been promptly sat upon, and where in consequence the opinion is secretly held by the profession that it was better not to diagnose yellow fever. This year, in Africa, the press called into serious question the diagnosis of the reputable medical authorities of the Colony, and stoutly upheld and liberally praised those who diagnosed "bilious remittent fever."

Press vilification is common all over the world, and it therefore becomes all the more incumbent upon those who are in administrative authority to uphold the decision of their medical officers. I will give three examples of notification fear in West Africa.

In 1884, a Dr Davies, practising in Freetown, reported a case of yellow fever. Upon receipt of this report, the acting principal medical officer sent him a letter, asking him the grounds upon which he made the diagnosis, and if "he considered the case of yellow fever which he had had in his practice to be contagious or not?" In a letter to the Governor the acting principal medical officer stated in his opinion that Dr Davies had made a grievous mistake in his diagnosis of yellow fever.

In 1903, a Cape Coast doctor, Dr Barker, wrote the acting principal medical officer reporting a case of yellow fever, and stating that it was the fifth case. The medical man who made this diagnosis was Dr Rome Hall. The acting principal medical officer wrote as follows to the Colonial Secretary :— "I regret to say that I am obliged to take the alarmist statement made by Dr Rome Hall with a large amount of doubt."

In 1910 there is an improvement as regards the majority of the medical officers, who all have had the courage of their convictions, and did not hesitate for a moment. A few held, however, that the disease was the well-known "bilious remittent fever," and the press did not hesitate to openly abuse those who diagnosed yellow fever.

The following extracts from West African Coast papers, serve to illustrate this extraordinary notification fear :—

The Alleged Outbreak of Yellow Fever in Secondee. Extract from the "Gold Coast Leader," 1910.

"Since we asked the question in a recent issue of this paper, whether or not it was true that yellow fever existed in Secondee, events have transpired there of great importance which throw a flood of light upon the situation, and, before setting down the facts, we may at once relieve the mind of the public by stating that there are very strong grounds for believing that, in all probability the cases which the hospital authorities labelled as

yellow fever and treated as such were not cases of yellow fever at all."

The same spirit of interference of the press in matters in which they can have little real knowledge is seen in the following extract from the *Sierra Leone Weekly News* of 4th January 1910:—

"Nevertheless there is a disposition on our part to beg leave to doubt as to whether the cases that have happened are cases of real yellow fever, or of what may be termed malignant malaria. We remember something of this kind occasioning much loss of life among the European portion of our community to have happened in the year 1884, when Rev. E. P. Sparks, Colonial Chaplain, and Mons Criquet, died, and the community was then plunged into much sorrow on their account. But the expert judgment of the day attributed the deaths of these Europeans, not to yellow fever, but to what they called malaria of a malignant type.

"May it not be so in the present case? We cannot of course speak confidently on the subject, not being experts, but we have always thought that a case of actual yellow fever is invariably associated with black vomit. We rather lean to belief in the malarial nature of the disease, because the present season of the year is easily conducive to malarial developments. We are just passing from the dry to the rainy season and, in the passage, we have had a few showers which, with the recurring sunshine acting upon drains, etc., may easily bring to pass any kind of malaria."

The 1884 outbreak which is referred to in the above was proved to be one of typical severe yellow fever by the Commission, appointed by a former Governor, Sir Arthur Havelock. There can be no doubt that the wish of the press is to call all fevers malarial. It has the merit of not leading to quarantine or producing a scare. But it has a disastrous effect in the lives of the whites.

In my opinion, the injury which articles of this nature are

likely to produce in the minds of the poorer and more ignorant classes is very great. A real injustice is done to the sanitary reputation of the colony, and the good name of the colony in other directions is also bound to be affected.

It is earnestly to be hoped that a more healthy, honest, and mutually co-operative spirit will prevail in future, and that the press will do all in its power, by means of reasoned articles, to show the world how, with a comparatively small outlay of money, the unquestionably unsavoury reputation of the Coast for disease is quite unnecessary and can be put an end to, to the great advantage of commerce and happiness.

The statement which I have made in my work, *Health Progress and Administration in the West Indies*, upon the obstacles to early notification, will bear repetition in this place, because they are applicable to what has happened this year, 1910, in West Africa. I wrote under the heading of "Early Notification: Timidity, Prejudice, and Opposition":—

"As in my British Honduras Report, so in this one, I seriously direct attention to the difficulties of early notification.

"These difficulties have been encountered in an aggravated form in British Honduras and in Central and Southern American ports generally. There is no question in my mind that a considerable amount of odium is liable to be incurred by a medical officer who has the courage to declare a case of yellow fever.

"This unsatisfactory state of affairs is often owing to the fact that there are usually to be found medical men who, without having themselves examined a particular case, nevertheless make up their minds that it is not yellow fever, but some other simple disease, of which they have had in their practice many cases. This gives the press an opportunity, if so minded, to vilify the first medical officer who notifies, and even to hint that he knows nothing about the disease, whilst at the same time lauding the particular medical man who has given the opinion which is more palatable, and therefore probably more in accord with the views of the newspaper.

These tactics constitute a veiled form of intimidation, and are destructive in the long run to the reputation of any colony for fair play and honesty.

"It is with very great regret that I am obliged to state that there was considerable opposition encountered in Barbados from a particular section, directed to thwarting and ridiculing those who were acting in the very best interests of the community, by insisting upon early notification of suspected cases."

CHAPTER XXI

ANTI-MOSQUITO ORDINANCES

WITHIN recent years an attempt has been made to grapple with the mosquito nuisance in West African colonies, just as I have shown has been done in the case of the West Indian colonies (see *Health Progress and Administration in the West Indies*, 2nd edition).

Given active medical officers and sanitary inspectors, a great deal can be done at a comparatively small cost, as I have witnessed this year in many places on the coast. But in order to accomplish this, the medical officer has to take upon himself the functions of sanitary inspector and labourer. In other words, he has to do the cleaning-up in large measure himself. His example in this respect is of immense benefit to the Colony; for the European merchants and the natives soon follow suit, and begin to realise that it is not beneath their dignity to sweep and garnish their back yards and compounds.

If the obvious and easily removed or remedied breeding-places were first done away with, an immense reform would be accomplished for a very little expenditure of money. It would, moreover, pave the way for the other improvement schemes, such as water supply and drainage. For a coast town to proceed with elaborate drainage schemes, whilst each compound is left to swarm with easily removed breeding-places, is action which displays wanton disregard of money, as well as ignorance. With a little energy and a little kerosine an immense

breeding-ground can be readily removed, and a corresponding improvement in the health guaranteed.

In Sierra Leone, a special ordinance was passed to deal more specifically than in the old Act of 1905 with mosquito larvæ.

No. 16 of 1910

An Ordinance to Amend the Public Health Ordinance, 1905

Be it enacted by the Governor of the Colony of Sierra Leone, with the advice and consent of the Legislative Council thereof, as follows:—

This Ordinance may be cited as the Public Health Amendment Ordinance, 1910.

Notwithstanding anything to the contrary contained in the Public Health Ordinance, 1905 (hereinafter called the Principal Ordinance), where mosquito larvæ are found by the Sanitary Authority in any collection of water or in any well, pool, channel, barrel, tub, bucket, or any other vessel in any premises, the occupier or owner in occupation of the premises on which the nuisance arises shall be liable on summary conviction to a fine not exceeding Twenty shillings for each offence, whether any such notice requiring abatement of nuisance or nuisance order, as is in the Principal Ordinance mentioned, is or is not served or made upon him.

This Ordinance shall be brought into operation by the Governor-in-Council by Order from time to time, and it shall be lawful for the Governor-in-Council by such Order to apply this Ordinance or any part thereof to any town or village or part of a town or village of the Colony, with or without conditions.

Nothwithstanding the provisions of Section 3, this Ordinance shall, until otherwise ordered by the Governor-in-Council, apply to the City of Freetown, subject to the proviso that no proceedings under the provisions of section 2 hereof shall be taken in respect of mosquito larvæ found by the Sanitary Authority in any well, pool, or channel in rock or earth till the same have been ordered by a further Order of the Governor-in-

Council: And subject to the further proviso that nothing in this section or in this Ordinance shall prevent any proceedings being taken in respect of the nuisances liable to be dealt with summarily under the Principal Ordinance or for the penalties imposed by the Ordinance, on account of the existence of such nuisances.

Southern Nigeria.—A special mosquito destroying ordinance was passed 4th August 1910.

It shall not be lawful for any owner or occupier to allow at any time the presence on his premises of any receptacle for water containing mosquito larvæ, or to allow any water to be kept on his premises for a period exceeding three days without the receptacle containing the same being emptied and cleaned, unless such receptacle is properly protected or screened, to the satisfaction of the Sanitary Authority, from the access of mosquitos, nor shall such owner or occupier allow on his premises any reasonably preventable conditions which may, in any way, be favourable to the breeding of mosquitos.

It shall be lawful for the Sanitary Authority to recover from the owner or occupier of any premises the expense of any measures carried out on his premises under the provisions of this Ordinance, but if it is satisfied that such owner or occupier is not in a position to pay such expenses, to pay all such expenses itself.

All expenses incurred by the Sanitary Authority in carrying out, with respect to any premises, the provisions of this Ordinance, shall and may be recovered in a summary manner before a Police Magistrate or District Commissioner, anything in the Supreme Court Ordinance to the contrary notwithstanding.

Any person who obstructs the Health Officer, or any Medical Officer specially appointed by the Governor for the purpose, the Sanitary Engineer, Sanitary Inspector, or any person duly deputed in writing by the Health Officer to carry out the provisions of this Ordinance in any act authorised by this Ordinance shall be liable to a fine not exceeding twenty-five pounds, or in default thereof to imprisonment not exceeding three months.

(i.) Any person who contravenes any of the provisions of Section 4 of this Ordinance shall be liable to a fine not exceeding five pounds or in default to imprisonment not exceeding one month.

Gold Coast Colony.—Under Section 18 of the Infectious Disease Ordinance, 1908, regulations were made to deal with the outbreak of yellow fever in Secondee. They were issued 17th May 1910, and the clauses dealing with mosquitos are as follows:—

The Senior Sanitary Officer, Medical Officer of Health, a Medical Officer, the Commissioner of the Western Province, District Commissioner, or any person authorised by any one of them, may enter upon any land, house, or premises in Secondee for any of the following purposes:—

(i.) To inspect all water receptacles therein and destroy, repair, or otherwise deal with such as are not mosquito proof, or form or may form breeding-places for mosquitos, and treat the water therein with kerosene.

(ii.) To fill up or otherwise deal with all pools, holes, wells and other places, where there is standing water which are or may become breeding-places for mosquitos.

(iii.) To make such alterations to the roof or gutters of any house or building to prevent stagnant water remaining therein.

(iv.) To clear and clean such premises of all rubbish tins, bottles, and undergrowth.

(v.) Generally to take all such steps as may be necessary to destroy and prevent the breeding of mosquitos upon any such premises.

The provisions of this Order shall be published by beating of gong within the said Municipal Area by the chiefs of Secondee. Made by the Governor-in-Council this 17th day of May 1910.

The Gambia.—The Public Health Ordinance of 1910 has been modified to lay greater stress upon mosquitos, insects, and parasites as follows:—

(*a*) All collections of water, sewage, rubbish, refuse, ordure, or

other fluid or substance, and all other conditions which permit or facilitate, or are likely to permit or facilitate, the breeding or multiplication of animal or vegetable parasites of men or domestic animals, or of insect or other agents which are known to carry such parasites, or which may otherwise cause or facilitate the infection of men or domestic animals by such parasites.

(*b*) Any collection of water in any well, pool, channel, depression, excavation, barrel, tub, bucket, or any other vessel, and found by the Board to contain mosquito larvæ.

(*c*) Any cesspool, privy, urinal, dung-pit, ash-pit, found by the Sanitary Authority to contain mosquito larvæ shall be nuisances liable to be dealt with summarily under this Ordinance.

The occupier or owner of any premises shall keep such premises free from all articles, bottles, whole or broken, whether fixed on walls or not, old tins, boxes, calabashes, earthenware vessels, shells, or any other articles which may retain water, and so become the breeding-place of mosquitos. Any occupier or owner of any premises failing to comply with the provisions of this section shall be liable to a fine not exceeding twenty shillings.

Any person who shall keep on any premises any collection of water in any well, barrel, tub, bucket, tank, or any other vessel intended for the storage of water without providing them with covers so constructed as to prevent the ingress of mosquitos into the same, shall be liable to a fine not exceeding twenty shillings. If a person shall fail to comply with the provisions of this section he shall, after notice received from the Sanitary Authority to comply therewith, be liable to a further fine not exceeding twenty shillings a day during his default.

On the receipt of any information respecting the existence of a nuisance liable to be dealt with summarily under the preceding sections (A, B, and C) of this Ordinance, the Board shall, if satisfied of the existence of such a nuisance, serve a notice on the person by whose act, default, or sufferance the nuisance arises or continues, or if such person cannot be found, on the occupier or owner of the premises on which the nuisance arises, requiring him to abate the same within the specified

time by such notice, and to execute such works and do such things as may be necessary for that purpose, and if the Board think it desirable (but not otherwise) specifying the works to be executed.

The Board may also by the same or another notice, served on such occupier, owner, or person, require him to do what is necessary for preventing the recurrence of the nuisance, and, if it think it desirable, specify any works to be executed for that purpose, and may serve that notice notwithstanding that the nuisance may from the time have been abated, if the Board consider that it is likely to recur on the same premises provided that where mosquito larvæ are found in any collection of water, or in any well, or pool, channel, barrel, tub, bucket, tank, or any other vessel, or in any bottle, whole or broken, whether fixed on a wall or not, tin, box, calabash, shell, or any other article, the Board itself may abate the same, and may do what is necessary to prevent the recurrence thereof.

All cesspits shall be so screened as to prevent the ingress of mosquitos, or alternatively shall be covered with oil or other approved larvicide at least once a week. The occupier or owner of any premises to which any cesspit not treated in accordance with the provisions of this section is attached shall be liable to a fine not exceeding twenty shillings.

The Board may by a general order prohibit:

(*a*) the making of holes, pits, or excavations, for the purpose of taking earth from them or of storing rubbish or offensive matter therein;

(*b*) the digging of cesspools, tanks, wells, or pits, without special sanction.

If any such excavation, tank, well, or pit is made after the publication of any such order, and without the permission required thereby, the Board may by written notice require the owner or occupier of the land on which the same is made to fill up the same with earth or other material approved by them.

If default be made in complying with such notice the Board may cause the work to be executed and half the expenses thereby incurred shall be paid by the owner and half by the occupier of the land.

THE ANTI-MOSQUITO DECREE, TOGOLAND, 1910

1. All receptacles for storing water must be furnished with a mosquito proof covering, or they must be emptied every four days at least, in order to prevent the development of mosquito larvæ. Instead of screening, a larvicide like petroleum may be added to the water. Boats may be treated in a similar manner.

2. Odd receptacles in which water may collect such as tins, bottles, etc. (boats and "Einbauma" excepted), are to be kept in such a manner that water cannot collect in them; pools of water must not be allowed to remain for longer than twenty-four hours. The decrees respecting deposits of refuse remain unaltered.

3. On the request of the local authorities waste ground and other uncultivated plots are to be freed from bush, which may serve as cover for mosquitos.

4. The Medical Officer and the Inspector employed by the local authorities are authorised to inspect grounds and localities for the purpose of sanitary surveyance at any hour of the day, alone, or accompanied by the Sanitary Inspectors.

The Sanitary Inspectors alone are authorised to do so only on certain days and at certain hours which are to be fixed by the "Medical Officer," or the local authorities and which must be made public. The official notification appears quarterly in the *Amtsblatt*, and at least once a week before the beginning of the period in question.

The owners of the lots have to submit to the required measures necessary for the sanitary surveyance.

5. Non-natives failing to comply are liable to a fine not exceeding 100 marks, or two weeks' imprisonment. In the case of natives, however, the fine will be according to the decree of the Governor of 22nd April 1896 (*Kol. Blatt.*, p. 241).

In cases of failure to comply with clauses 1 or 2, fines will be imposed:

(*a*) On the owner of the receptacles named under clause 1, or on anyone who has not complied with the conditions of clause 2.

(*b*) If the occupier (*a*) cannot be found out, or his punishment is not possible for some other reason, then the

owner of the grounds on which the nuisance has occurred is liable to prosecution.

(*c*) If the owner of the property cannot be found or his punishment is not possible for some other reason, then the agent of the owner or the administrators are liable to prosecution.

6. This decree comes into force on 1st July 1910.

Enforcement of anti-mosquito laws.—The preceding laws and regulations are very good on paper, but unless they are backed up by daily inspections by the sanitary inspectors and medical officers, and unless the inhabitants who may offend are quickly brought to book for offences under them, they are of course, useless. During the outbreak of yellow fever, 1910, the senior sanitary officer in Secondee took summary action with good effect, and fines were likewise inflicted. In Nigeria the passing of the Ordinance was viewed with great disfavour, by one section of the press, which indulged in the following mediæval garbage, by way of comment on the Ordinance. From *The Nigerian Times*, 5th July 1910:—

"But we might ask the question whether the Government realises that the task, the performance of which it has undertaken under this Ordinance, is one which is really superhuman. First of all the Government ought to be aware that mosquitos do not breed, nor do they propagate in the sense that we understand the propagation of species. The larvæ are generated by some atmospheric action on water—fresh-water especially—under certain climatic conditions and influences, more particularly at certain seasons, operating on the oxygen or some other of the constituents of fresh-water; and the larvæ develop into mosquitos. We do not profess to give this as a scientific description, nor would we allege that the statement is founded on accurate scientific knowledge. The statement is nevertheless an accurate description of certain processes which are to be seen going on under ordinary observation daily. So that whether the 'receptacle in which water is stored is properly protected or screened from the access of mosquitos,' the larvæ

can and do oftentimes generate in the water so protected or screened, and develop into mosquitos on any occasion of a contact for a short period with the influences of the external atmospheric action."

I have repeatedly pointed out the existence of this mediæval superstition in the newspapers of British tropical colonies. It demonstrates that ignorance of simple biological facts is still a nightmare to overcome in West Africa. Moreover, if the ignorance of simple facts is so obvious in this direction, we may be certain that ignorance in the other directions which make for the progress and welfare of the colony is equally great.

In my opinion, formed as the result of my various investigations in tropical countries far more stress requires to be laid upon educating the people both young and old. In Sierra Leone, thanks to this far-seeing initiative of Sir Leslie Probyn, a great deal has been accomplished in this direction. In the Gold Coast Colony the late Sir John Rodger has also sown good seed.

Prosecutions under the Mosquito Ordinances

In *Freetown*, from April to July, thirty-nine convictions were obtained for having larvæ on premises and for other offences under the Health Act; fines to the total of £11, 6s. 7d. were inflicted.

In *Secondee* from May up till June 1910, thirty-two convictions were obtained and fines inflicted to the total of £39.

In *Accra* there were several convictions and fines from 10s. to £1 were inflicted.

CHAPTER XXII

TOWN PLANNING AND SEGREGATION—MINING AND RAILWAY WORKS

SYSTEMATIC inspection of the sickness and mortality returns of the West African colonies, which are published in the annual medical and sanitary returns of the various colonies, shows most unmistakably that the merchant and non-official classes are at a disadvantage compared to the official classes both as regards sickness and mortality rates. These points are well brought out in the following returns prepared for Secondee on the Gold Coast :—

Year.	Died.	Invalided.	Death-Rate.	Invaliding Rate.
OFFICIALS.				
1906	1	21	1·8	39·6*
1907	1	1	0·7	3·9
1908	0	11	nil	5·9
1909	0	10	nil	16·12
NON-OFFICIALS.				
1906	3	11	40	148·0
1907	3	10	18	62·0
1908	3	12	22	88·0
1909	2	7	13·3	46·6

* Three included from up country.

Commenting upon these figures the senior sanitary officer of the Gold Coast states as follows :—" A study of the compara-

FIG. 56.—Coolie Ranges on each side of Wide Road, Trinidad.

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tive death and invaliding rates of the European officials and non-officials in Secondee from the years 1906 to 1909 affords an interesting object-lesson on the value of segregation, when it is borne in mind that the officials are segregated, the non-officials not.

Invaliding rate of Officials per 1000 . . .	16·38
Invaliding rate of Non-officials per 1000 . .	86·1
Death-rate of Officials per 1000	0·52
Death-rate of Non-officials per 1000 . . .	23·33

"In a report on Secondee, written in April 1910, I stated that 'experience has taught us that Europeans who reside in West Africa unsegregated from natives, have a high sickness and death-rate.' This experience has been added to by the recent outbreak of yellow fever in Secondee."

"When one reflects that probably not one of the valuable lives sacrificed in the recent outbreak would have been lost had the European merchants lived like the officials, segregated from the natives, the value and paramount necessity of practising segregation in this country is impressed in one's mind. I am aware that public opinion is not yet ripe for the consideration of compulsory segregation; but I look forward to the day when it will be, and when the fact will be recognised that the natives of West Africa are so many reservoirs of disease to which they themselves are comparatively immune, but which, when mosquito-borne to the Europeans, are capable of producing fatal results. Under present conditions it is as dangerous to live unsegregated from the natives in West Africa as it is to live in the immediate vicinity of a small-pox hospital in England, but the former practice is tolerated, the latter not. In the meantime it will be necessary to largely increase the number of scavengers employed and the sanitary staff, in order to wage a constant war on mosquito-breeding vessels and areas. I advise, too, that the special anti-larval powers given to the sanitary authorities in Secondee during the outbreak of yellow fever be made to apply all over the Colony."

With the statement contained in the preceding paragraphs I heartily agree.

The cause of the ominous difference between the two classes is not far to seek. Principal medical officers have over and over again drawn attention to it. Governors have also done their best to impress upon the mercantile classes that this difference does exist. But in spite of all warnings little endeavour is made to remedy the evil. The reason for this is of course not far to seek. The medical officer and the administrator points out that as the result of the most recent and well-established researches in medicine, there can be no question that certain diseases like malaria and yellow fever are endemic amongst the native races, and that in consequence the first measure of protection which the newcomer should naturally take, would obviously be to sleep in some locality removed from the native houses.

On the other hand, a short-sighted financial policy on the part of the mercantile firms, argues that it would place the white traders at a disadvantage if they resided at a distance from their place of business.

It will perhaps be remembered that precisely the same arguments were held in Europe from the Middle Ages up to the nineteenth century. Merchants in olden times lived over their shops, no doubt to keep guard over their goods and money. But the practice had to be given up because it was obviously insanitary.

At the present day in West Africa it is not necessary for the business clerks and managers to reside in business houses in the midst of native houses. During the *daytime* the business can be transacted in the store, but at *night-time* the clerks and managers could readily, were quarters provided for them, sleep some distance away from the native town.

Observation has taught us that risk of infection during the day is far less than at night. Business can be carried on with comparative safety in the towns during the daytime, because

FIG. 57.—A Road in Port of Spain, properly graded, with well-made concrete side drains.

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daylight and currents of air afford a very considerable measure of safety against mosquito-carried diseases.

On the other hand, it has been proved in innumerable instances that those traders who have lived on their business premises amongst the native houses, and especially those who have been obliged to live in badly constructed houses and on the *ground floor*, have almost without exception contracted some form of endemic disease. Surgeon-general Blair drew attention to this fact in Georgetown, British Guiana, and during the recent epidemic (1910) of yellow fever along the West Coast, it has been most observable that the white residents who lived in the midst of the native inhabitants were the first to contract yellow fever, whilst those who lived some distance away were not affected.

Thus the Hill Station in Freetown afforded complete protection to its inhabitants. On the other hand, it is very significant that in Freetown itself the first whites to become infected with yellow fever were the Syrian traders living in the dense native quarters of the town.

These traders live, as is well known, with their families in small houses wedged in amongst the native houses. Their sleeping and living rooms are often on the ground floor, or at most only slightly elevated from the ground level.

The *Stegomyia* is breeding everywhere in immense numbers immediately around them, and can readily gain access to their sleeping and living rooms. Therefore the fact that the small Syrian traders first demonstrated the existence of yellow fever in Freetown, is precisely what one would expect in a place where we have every reason to believe yellow fever is endemic.

Had it broken out at the Hill Station or in the military quarters placed in well-elevated sites above the town, it would have been a matter of very great surprise. It did not do so, and, as has been demonstrated on countless occasions in the past, segregation afforded the necessary protection.

But in spite of this fact being fully accepted all over the tropical world as a well-established axiom in yellow fever, firms still pay little attention, and will not take the opportunities held out to them by Colonial governments to select suitable sites, removed a short distance from the native houses.

The Gold Coast Government announced in 1908 that it was prepared to lease land close to Accra for a term of fifty years at a very reasonable price for the erection of bungalows, built on an approved plan.

It was stipulated that the only natives to reside on the premises were to be the personal servants of the residents.

The bungalows and grounds were to be subjected to periodical inspection by the medical authorities.

Thus both at Accra and Freetown every inducement has been held out to the white non-immune to reside outside the danger zone; but exceedingly few have taken advantage of these offers. Many still reside in the closest proximity to the native houses, and criticise the administration because their sickness and mortality rate is high.

The question of town planning is now likely to assume a practical form in view of the increasing prosperity of West Africa.

This will afford an excellent opportunity to bring about segregation from the very outset, by reserving, as has already been done at Bo in the Sierra Leone Protectorate, a portion of the town for the white traders.

At the same time care can be taken that the streets are of suitable width, and that none of the glaring disadvantages of existing native towns are repeated.

In conclusion, I would place segregation of the whites (non-immunes) in the fore-rank of all prophylactic measures at the present time in West Africa. It is practicable and can be carried out at once.

Drainage operations and anti-mosquito measures generally require a little time and organisation before results can be

seen. This is readily understood by those who have practical knowledge of West African towns.

The merchant at home in England does not live in the slums, why should he expect his clerks to do so in West Africa, where they are exposed to far greater risks owing to the prevalence of endemic diseases.

The saving in invaliding pay would alone compensate for any supposed disadvantage of sleeping a short distance from the native quarters.

CHAPTER XXIII

YELLOW FEVER PROPHYLAXIS IN WEST AFRICA, 1906 AND 1910

BEFORE discussing in detail routine yellow fever prophylaxis, I wish to draw the reader's attention to what was done this year, and also in 1906 to prevent the spread of yellow fever. It is a very practical lesson and serves as an admirable guide for future occasions.

In the first place, owing to a variety of circumstances on the West African Coast, the medical men were not on the lookout for yellow fever. When on 12th May the medical officer at Secondee was convinced that he was face to face with yellow fever, owing to the fact of the occurrence of three cases exhibiting the clinical features of yellow fever, not a moment of time was lost. The greatest praise is due to all the medical officers, for their exceedingly prompt and fearless action in this connection.

On 13th May the Port of Secondee was declared infected.

On 14th May the senior sanitary officer arrived to take charge of the preventive measures, and brought with him a Clayton sulphur apparatus and a supply of sulphur.

Two important movements were set on foot, both of which were based upon the principle of segregation of the non-immunes.

Step 1, taken on 15th May, consisted in deflecting the traffic from the infected town of Secondee to another point on the coast 11 miles distant, where the ships could call. Secondee is the principal port for the mining centres, and is the point of

FIG. 58.—The "Bee Hive Destructor." A very simple, efficient, and cheap form of destructor, constructed out of "swish." Secondee, Gold Coast.

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departure of the railway system, and under ordinary circumstances there is a constant flow of non-immune whites to and from the mines to the steamers. Therefore the senior sanitary officer deflected the railway passengers at a place on the railway 9 miles above Secondee, and arranged for their transport to a small village on the coast, especially prepared for their reception 11 miles distant along the coast from Secondee. The non-immunes were safe from the risk of infection in this village, and were most carefully supervised. It served as an observation or detention camp for travellers in transit, and there being no yellow fever there, the ships called, and trade with the mining centres was inconvenienced as little as possible.

Step 2 consisted on 15th May in issuing instructions to prepare temporary sleeping accommodation for all those white traders in Secondee who had not a residence outside of the town area. A temporary camp was chosen on an elevated site at a reasonable distance from the town. On the same day a public meeting was called of the residents, and the nature of the outbreak and the preventive measures contemplated were fully explained. At the same time, all Europeans residing in the infected business area were notified that from 5 P.M. on 17th May, Europeans would only be allowed within the infected area between the hours of 7 A.M. and 8 P.M. In other words, the non-immunes were obliged to *sleep* outside the infected area. After these immediate emergency steps were taken, the senior sanitary officer proceeded to organise the sanitary forces at his disposal, to enrole volunteers, and to proceed to execute the well-known *anti-Stegomyia* measures of larval destruction, and the systematic fumigation of all houses in the infected quarter. Before proceeding to describe these steps, it is interesting to compare the steps which the French medical authorities took in 1906, on the outbreak of yellow fever at Bamako, in Senegal.

1. The first step consisted in an official notification of the danger.

2. Secondly the medical men were summoned together to discuss the situation.

3. Sanitary forces were organised, and a raid made upon the breeding grounds of the *Stegomyia.*

4. A temporary camp was constructed at a distance from the infected town, where the non-immunes were obliged to retire from sunset to sunrise. This was enforced for twenty nights. Goods and provisions for the use of the camp were packed during the daytime.

Is is very noteworthy, therefore, that both the French and English medical authorities put the removal of the non-immunes in the first line of defence. This is, in my opinion, the right course. Remove to a place of safety the vulnerable, *i.e.*, enforce temporary segregation until all the houses in the infected quarter are fumigated, and all infected *Stegomyia* are destroyed. In both instances absolute success followed these measures. Where these precautions are not taken—I can speak with experience—yellow fever lingers on. To continue with the prophylactic measures which were undertaken at Secondee—

On 18th May, mosquito brigades were organised. The emergency temporary buildings erected consisted of:—

1. A mosquito-protected hospital for Europeans.
2. Accommodation for European contacts.
3. Mosquito-proof hospital for natives.
4. Buildings for Syrians.
5. Buildings for segregating natives wishing to leave Secondee.
6. Buildings for segregating Europeans wishing to leave Secondee. Sixty-one Europeans were segregated for six days in this camp which was placed outside the infected area. Altogether 144 separate buildings were fumigated by the Clayton apparatus, or by simply burning sulphur. The closed waggons on the railway were fumigated before loading. The preventive measures adopted are summarised as follows by the senior sanitary officer: —

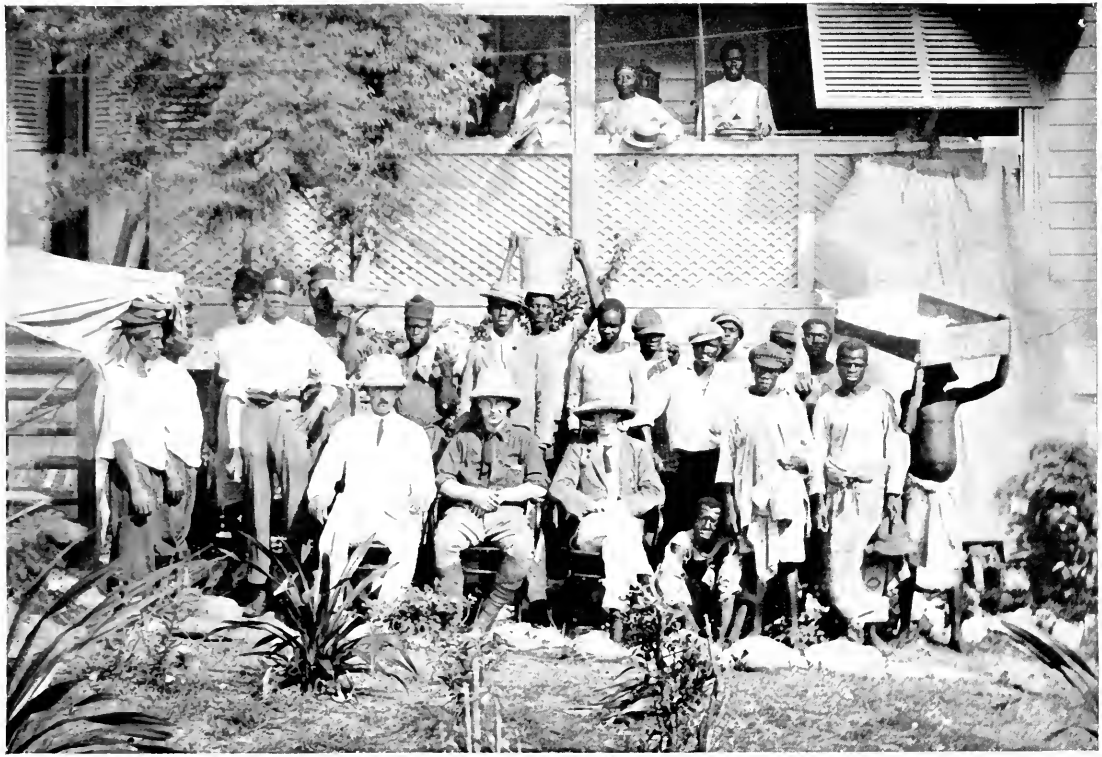

FIG. 59.—Yellow Fever Outbreak, Secondee, 1910. The Medical Officers and their Papering and Fumigating Gang.

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(1) "The evacuation of every infected bungalow. These bungalows were sealed up, and each of them was fumigated with sulphur, and afterwards Claytonised.

(2) "The evacuation of the infected area, by Europeans, between the hours of 5 P.M. and 7 A.M. This continued for a month, and no European was infected after the evacuation order was put in force on the evening of the 17th May. It was a drastic measure, which, however, I submit was justified by the prompt checking of the outbreak which followed its enforcement.

(3) "The fumigation with sulphur gas of every house in business area and of every European bungalow outside it."

(4) "The perforation of all gutters, a hole being punched in each lineal yard of guttering. It is a matter of common experience to find a mosquito-proof barrel or tank full of mosquitos on the wing, a fact which is explained by the supposition that they lay their eggs in the gutters, and that these or their larvæ are subsequently washed down into the tank or barrel. If the tank or barrel be mosquito-proof, no harm results, as the mature insect is unable to find an exit, but if the receptacle is not mosquito-proof it is otherwise. I believe if this simple precaution were generally adopted, it would be the means of destroying the potential breeding-places of innumerable mosquitos."

(5) "Gangs were sent round collecting all tins, bottles, or other receptacles liable to breed mosquitos.

(6) "The whole town was divided up into mosquito brigade areas, each of which was in charge of a European, who went round with a small gang. At first the people were warned, and the larval breeding vessel was merely oiled or upset, but afterwards full advantage was taken of the special anti-larval powers—the power to destroy —passed in Council on the 17th of May, and any vessel found to contain larvæ, when no honest attempt had been made to screen it, was destroyed. Barrels were emptied, turned over, and their ends were staved in with an axe. It may be argued that this is a drastic

treatment, but it is the only way. If it is carried out, people will soon begin to take trouble and make an effort to keep their water receptacles free from larvæ; and when they are found to be so doing, they should be assisted by carpenters employed by the Government, as was afterwards done in Secondee."

"I am convinced that we shall never be able in this Colony to free the Coast towns of *Stegomyia*, until this power to destroy larval breeding vessels has been conferred upon every medical officer and district commissioner in the Colony.

"I am informed that such powers are given in the Togo-country."

Notices which were issued by the Senior Sanitary Officer

1. *Notice upon importance of early diagnosis, Secondee, 21st May* 1910.—The following telegram was sent to medical officers at Axim, Chama, Tarkwa, Cape Coast, Saltpond, Winneba, Addah, Quittah, Akuse, and to the senior medical officer, Coomassie:—

The early diagnosis of yellow fever is of the utmost importance, and the presence of albumin in the urine is one of the commonest symptoms of the disease. Every medical officer, therefore, should get ready a small portable urine testing apparatus, and take it with him whenever he is called upon to see a case of illness, in order that he may be able to test at once for albumin.

2. *Precautionary measures to be taken in Gold Coast ports.*—The following wires were sent 18th May 1910, to the medical officers at Chama, Cape Coast, Winneba, Saltpond, Quittah, Addah:—

"You should take the following precautions to prevent an outbreak of yellow fever and to meet such an outbreak:—

"(1) Select sites for isolation hospital for Europeans, a

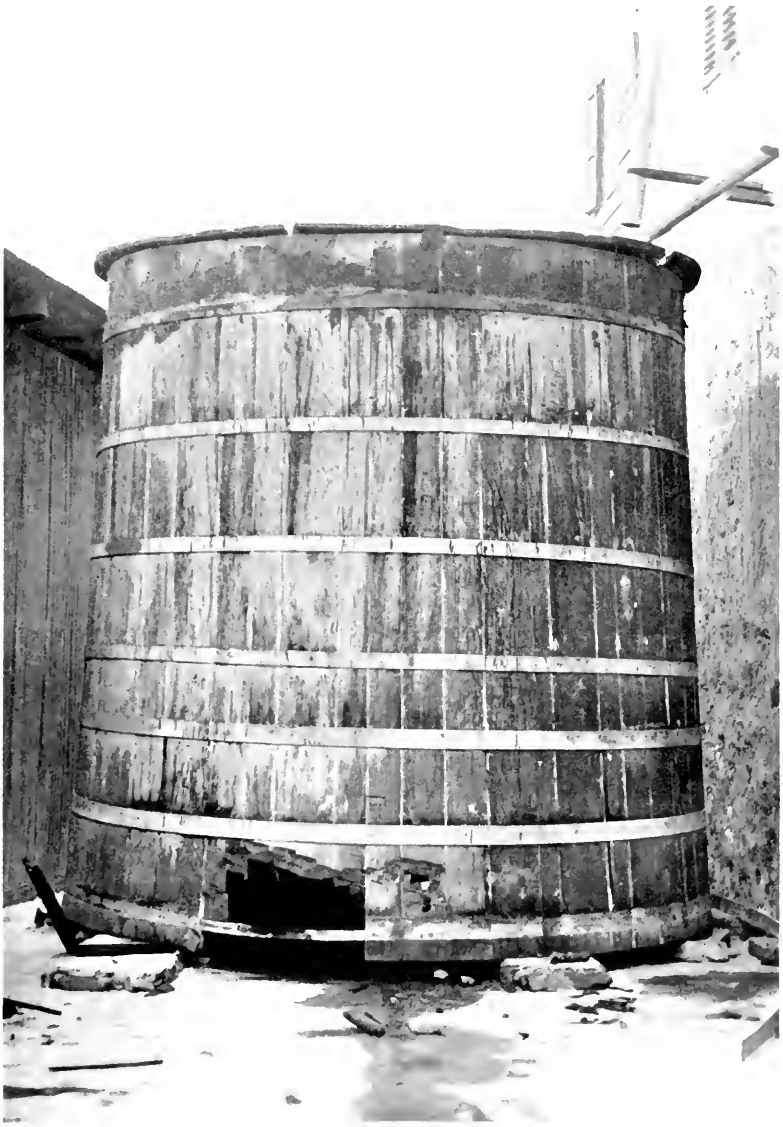

FIG. 60.—A Large Rain-water Vat, forcibly broken by the Health Authority because the owners failed to screen it, after proper warning.
Secondee, 1910.

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contact camp for Europeans, an isolation hospital for Natives, a site for temporary housing of Europeans who may have to be evicted from an infected area.

"(2) Post-mortem all suspicious cases of death among Europeans or Natives.

"(3) Organise Mosquito-Brigades under European Supervision.

"(4) Have all gutters perforated, tanks made mosquito proof, and take measures generally for the destruction of mosquito larvæ."

A sum of £500 was allocated to the above ports as well as to Secondee and Axim to take these steps to prevent an outbreak.

3. *Public Notice upon the Danger of Breeding Mosquito Larvæ, issued by the Sanitary Board of Secondee*

"This disease, yellow fever, is conveyed by the mosquito. The breeding-places of these mosquitos are in any receptacle of stagnant water.

"The rule as to the screening of tanks, water butts, etc., will be enforced with the utmost rigour.

"Any person on or near whose premises bottles, tins, or other receptacles that hold water, such as imperfect guttering, liable to breed mosquitos will be severely dealt with.

"All bush and weeds must be kept cleared around the house."

On 16th May mosquito brigades were organised and put in charge of competent officials and volunteers and apportional districts."

4. *Notice to Natives*

Any Natives wishing to leave Secondee should apply to the Provincial Commissioner for permission to reside in the Native Segregation Camp beyond No. 2 Lagoon.

After five days' isolation there they will be given Medical Certificates and allowed to leave Secondee.

The Camp will be ready for occupation on Monday, the 23rd of May.

Notice sent 21st May 1910 to the European Traders and Educated Natives in Secondee

The fumes from burning sulphur kill mosquitos; therefore kill all the mosquitos on your premises by adopting the following procedure, fumigating each room in turn:—

1. Measure the cubic capacity of the room.
2. Shut all the doors and windows and paste paper or cloth over every aperture, close the keyhole with paper or cloth.
3. Take some sulphur, 2 lb. for every 1000 cub. ft. of space in the room. Place the sulphur in a tin and the tin in a basin containing water to the depth of 1 in. Pour a little methylated spirit over the sulphur, set fire to it, and leave the room, closing the door after you.
4. Keep the room closed for three hours.
5. Afterwards sweep the ceiling, walls, and floor of the room, and burn the sweepings to ensure the destruction of any mosquitos that may be simply stupefied.
6. Sulphur may be obtained at the Town Council Office any morning from 8 to 9 A.M.

5. *Notice sent by the Senior Sanitary Officer to all Ministers of Religion and Teachers in Schools*

The town of Secondee being infected with yellow fever it is at the present time especially important that the fact that this disease is solely caused by the bite of a mosquito should be impressed upon every member of the native community, and I shall be grateful if you will do your utmost, both in the pulpit and out, to educate the public in this matter. It is especially important also that instruction should be given on this subject in all schools. You have already been supplied with literature giving the salient causes and the methods of dealing with them.

6. *Notice to Prevent the Spread of Yellow Fever by the Destruction of the Mosquito*

Inside the House

(1) Keep all windows and doors open to allow in air, light, and wind: three enemies of the mosquito.

(2) Do not let beds or other articles of furniture rest against

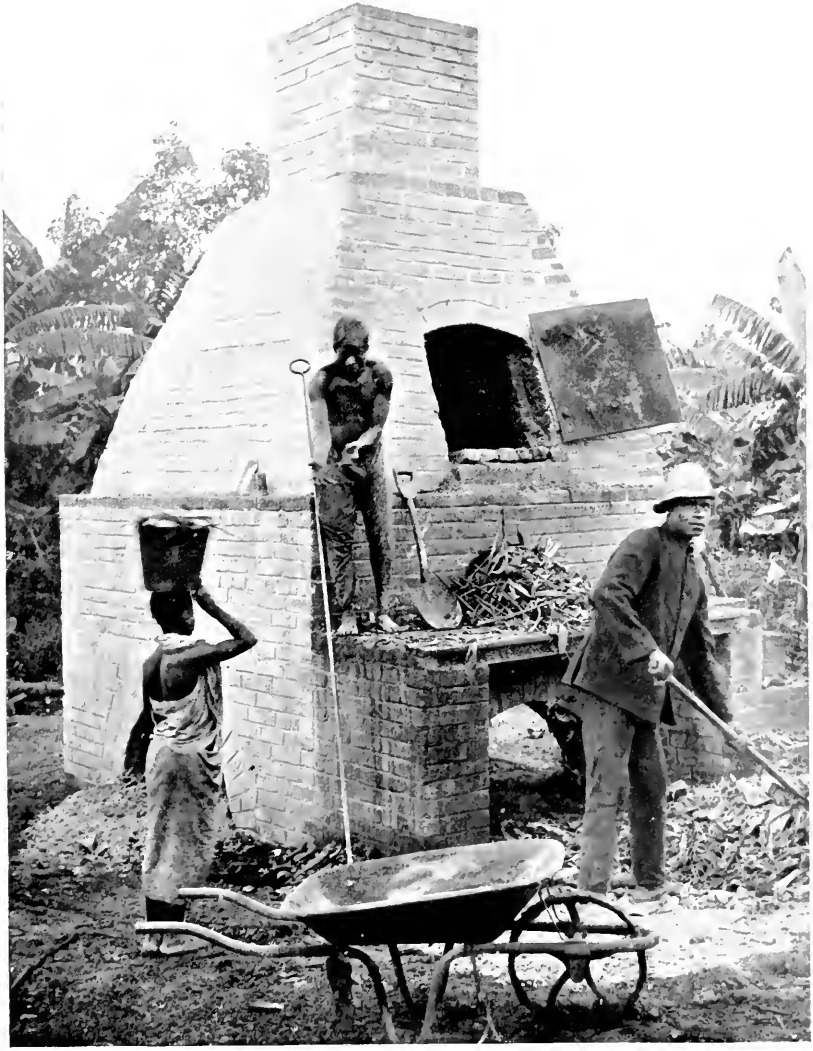

FIG. 61.—A Substantial Incinerator. Coomassie, Gold Coast.

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the wall for mosquitos to lurk behind. All such articles should be pulled out at least 18 inches from the wall. All clothes hanging in wardrobes, or on the wall, and all books on bookshelves should be shaken freely every day to dislodge mosquitos.

(3) Water in flower vases should be changed daily.

(4) The practice of standing the legs of tables, cupboards, meat-safes, etc., in tins of water to prevent the ingress of ants should be discontinued.

(5) All vessels in the house, or compound, capable of containing water should, unless actually in use, be turned upside down or destroyed, and those in use should be made mosquito-proof.

Outside the House

(6) All gutters should be perforated to the extent, at least, of one hole to each lineal yard of guttering.

(7) No depressions in the ground, ungraded ditches or other conditions likely to favour the breeding of mosquitos should be permitted inside the compound or in the vicinity of any house.

(8) All long grass, weeds and banana trees near to houses should be cut down.

In addition to the preceding excellent measure, which could be carried out without any delay, permanent reclamation work was started by taking in hand the filling up of one of the lagoons in Secondee, where it was shown *Stegomyia* was in the habit of breeding.

Owing to the deflection of traffic above Secondee to the temporary Port of Chama, comparatively little disturbance of the shipping took place.

The senior sanitary officer and those who worked with him did not escape the usual odium and criticism which is invariably poured on those who have the courage to declare a port infected with a disease like yellow fever. In the end, however, as is also usual, the action of the sanitary staff receives its full praise.

Yellow Fever Prophylaxis in Freetown, 1910

The conditions obtaining in Freetown differed from those in Secondee. This port contains a large population of indigenous coloured inhabitants, a garrison high up on one of the hills, and a very beautiful hill station at a considerable distance from Freetown, which is the residential suburb for the majority of the white official classes. The traders live in the better class houses in the town, but comparatively close to native streets. The smaller white traders, the Syrians, live in the midst of the native houses, in small poorly constructed dwellings. Both the garrison and the officials in the hill station were very effectively segregated. The better class traders lived in larger and better houses, not completely segregated, however. The Syrians with their families lived amongst and just as the native residents. The necessity for making provision for the non-immunes was therefore not necessary, as this already to a large extent existed. Attention was therefore directed as in Barbados in 1909 to bringing about a permanent anti-mosquito reduction in Freetown, by increasing the visits of inspection, by inflicting fines, and by educating the public generally. Indeed the method of procedure was the counterpart of that practised last year in Bridgetown, Barbados. The course of the outbreak is highly instructive. In the first place, all those who lived out of the infected town escaped free. Those who by the nature of their business were obliged to live in the town suffered to a variable extent; those who, like the Syrians, were in the worst part of the town, in the midst of the native population, were most heavily affected. Thus the segregation, which already existed in Freetown, accomplished what the emergency segregation at Secondee and Bamako likewise succeeded in doing. Yellow fever was declared 15th May 1910. Immediately a very vigorous anti-*Stegomyia* campaign was organised. The following very useful notices were issued. They may serve as

guides in the future; many of them are of local application, for it must always be remembered that each country has its own special nuisances:—

1. *Notice: a Cleaning-up Day Appointed*

"It is from want of thought more than from want of energy or effort that so many old useless tins, bottles, pans, calabashes, and other things are not removed to the refuse bins by occupiers from their premises, yards, and compounds.

"In order that persons may be frequently reminded of their duty in this respect, the *Mayor* and *Councillors* of this City hereby *declare* and *proclaim* the day of *Wednesday* in each week to be a special "Cleaning-up Day," and we enjoin all inhabitants to pay special attention on that day to ensure that all useless old vessels are removed to the *refuse bins*, and none allowed to remain on their premises.

"It is not intended that this duty should be neglected on other days; but it is hoped that when a special *reminder day* is observed every week, it will assist the public in preventing accumulations in their compounds, yards, and premises by frequently reminding them of the duty of removal."

2. *Notice*

The public is hereby warned that Section 4, Sub-section (3) (*d*) of "The Public Health Ordinance, 1905" (No. 15 of 1905), reads as follows:—

"Where mosquito larvæ are found in any collection of water, or in any well or pool, channel, barrel, tub, bucket, or in any other vessel, the sanitary authority may themselves abate the same, and may do what is necessary to prevent the recurrence thereof.

"Notice is hereby given that in order to prevent the recurrence of mosquito larvæ in barrels, tubs, etc., in which mosquito larvæ are found, measures will be taken by the sanitary department, which may include removal of the vessels, or removal of their bottoms or such portions of them as will prevent the recurrence of the above-mentioned nuisance."

3. *Notice to Householders and Occupiers*

"Collect all house and yard rubbish, empty or broken bottles, tins, calabashes, and useless vessels of all kinds, and vessels you do not require to use, and place them in a heap in one place in your compound or yard near the entrance."

4. *Notice*

"To enable occupiers of lots to fill in pools and depressions in the ground, the Government is providing broken stone in heaps at convenient places without any charge."

5. *Notice: Broken Bottles on Boundary Walls*

"Break away the glass till only short sharp pieces are left in the cement, then no glass will be left on the wall in which water can remain, and no 'wrigglers' can live, and no mosquitos can breed there.

"Why not help to stop quarantine?"

6. *Notice: Bottle Borders*

"are not beautiful, and the deeper the hollows in the bottoms of the bottles are, the more rain-water they hold, and the worse they are, as they breed mosquitos from 'wrigglers' in the water. Do away with bottle borders, and place stones there instead, and when the 'rains' are over, whitewash them.

7. *Notice: Why Not Help to Stop Quarantine?*

"There are many persons in Freetown who keep Ojuoro Water in calabashes, pots, etc., because they think it makes the water good for washing sore eyes, but mosquito larvæ live in the water and breed into mosquitos. The same happens in water with kola leaves in it.

"A better eyewash can be bought from any druggist, made up of Boracic Acid, 1½ drachms dissolved in 6 oz. of water. Anyone unable through poverty to pay druggist price can obtain it for three pence at the Colonial hospital. This eyewash will keep good for a very long time if the bottle is corked.

"Throw away the Ojuoro plants and water, and help to get rid of quarantine!"

Sulphur fumigation.—Houses in which cases of yellow fever occurred were fumigated.

Health in Mining and Railway Construction Camps

We know from past experience, the great loss in men and money which has occurred only too often in connection with industrial enterprises in the tropical world, and especially in West Africa, ever since the ill-fated industrial mission to Bulama.

It is not unreasonable to expect that both private companies and Governments should from the outset make all rational preparations to prevent disease. We know from innumerable reports the success which has attended engineering enterprises, like those at Panama and in Mexico, where sanitary precautions have been taken.

I have therefore thought it of advantage to emphasise those precautionary measures which, in my experience, demand consideration at the outset of every enterprise in a tropical country. The measures group themselves under the following heads:—

1. *Segregation.*—It must always be remembered that the white man is in a position of disadvantage compared to the black man. We know that the latter is the host of the virus of malaria, yellow fever, sleeping sickness, and probably of other diseases, and that to a varying degree he is immune to these diseases. On the other hand, we know that the white man from Europe is absolutely non-immune on arrival, and that if any of these parasites gain access to his blood he is incapacitated from work. Therefore, knowing that the native carries in his blood stream the infecting virus, and that in all probability the carrier of the mosquito is at hand in abundance, it follows that the first thing to arrange for is the *segregation of the non-immune.*

The non-immunes must be provided for in bungalows separated by at least, a quarter of a mile from the dwelling places of the immunes, *i.e.*, the natives. This primary pre-

caution should never be omitted. It is an advance along the line of least resistance, and experience has demonstrated it is a certain line of defence.

It is only common sense to protect the white labourer, who must from the outset be at a certain disadvantage.

With regard to the construction of the bungalows, there are very many exceedingly good models. The points to aim at are exposure of the bungalow to the prevailing currents of air; the sleeping rooms should face the prevailing currents. For this reason also elevated sites, and sleeping rooms raised above the ground-level are essential. Mosquitos shun currents of air.

For the reasons above, all bush should be cut close down to the ground; nothing should impede the currents or give shelter to mosquitos.

In the next place, the bungalow should be so constructed as to give the maximum shade and protection from the sun rays; for that purpose the thatch roof, in addition to the iron roof, as devised by Sir Leslie Probyn in the Sierra Leone Protectorate, is most useful and simple.

In order to still further minimise the risk of infection, the men must be compelled to sleep under mosquito nets, and, where possible, part of the verandah or a living room should be made mosquito proof. The chief factors which are to-day reducing the sickness and mortality rates from malaria and yellow fever over the tropical world are sleeping under mosquito nets and segregation.

2. *Mosquito destruction.*—A defensive measure of easy execution provided that it is set about intelligently, bearing well in mind what is to be aimed at. The end desired is the destruction of those mosquitos proved to be harmful to man, and which in consequence, will in every probability be found breeding close to him. In other words, what should be aimed at is the extermination of those mosquitos which breed in and immediately around the dwelling-places of man. Were this

simple precaution taken the major proportion of the fevers of West Africa would disappear.

For this purpose every article which can by any possibility hold water should be removed from the compounds, roads, and waste places. Every week a tour of inspection should be made; all compounds examined, and all odds and ends removed —tin cans of all kinds and discarded bottles and broken crockery are exceedingly common in all mining camps. Notices should be pasted up warning the workmen of the danger of leaving the articles lying about. All these odds and ends should be buried in suitable places.

Next to the removal of odds and ends should come *bush clearing*. All compounds, roads, and waste places should be kept absolutely free from weeds and bush of any description. The bush around every village or camp should also be kept well down by repeated clearing. The bush obstructs the current of air, facilitates the breeding of mosquitos, and conceals all kind of insanitary material.

If there is no pipe-borne water supply, and cisterns and barrels have to be used for storage purposes, these must all be securely screened by copper gauge. This can be done at little expense.

Treatment of pools, marshes, etc.—If these form in or immediately around encampments, they will give rise to the breeding of dangerous mosquitos; where possible, they should be filled in; but failing that, they should be oiled once a week with kerosene. None of the precautions which I have enumerated above are costly, nor require expensive administrative measures to carry them out. They are common-sense methods, and an intelligent foreman could do all that is required.

Pure water supply.—In view of the liability of all engineering and mining camps to water-carried diseases, care should be taken from the outset to lay down a pure pipe-borne water supply where this is possible. The greatest care must be taken to ensure the purity of the source. If it is a stream or spring

no house should be allowed near it. Wells are a source of grave danger in any workman's camp, unless they are very deep (artesian); shallow wells should be rigorously excluded. Until a pure water supply is assured, directions should be issued that all water must be boiled; and boiled water should be stored in convenient receptacles for the use of the workmen.

Disposal of excreta.—The costly and disastrous experience of ankylostomiasis has taught a salutary lesson upon the imperative necessity of the careful removal of night soil, and the rigorous prevention of depositing fæcal matter in the bush. Proper trenches should be constructed, and the excreta should be covered over so that flies cannot get to them.

In connection with *railway construction* the indiscriminate formation of burrow pits should be prohibited. When on the Gold Coast, I investigated the sickness and mortality rate amongst the men employed in the construction of the railway system, and there is no question that the amount of sickness was very high, and that a great proportion of it could have been prevented.

In the mining centres also there have been mysterious and very fatal outbreaks of what were without doubt mosquito-carried diseases, and in all probability of the nature of yellow fever and malaria. This could readily have been avoided by the use of intelligence and the expenditure of very little money.

Choice of a medical officer.—Where, as is usually the case, mining and exploration companies employ their own medical officers, I would strongly urge that preference should be given to those candidates who possess, in addition to the usual medical qualifications, the Diplomas of Tropical Medicine and of Public Health.

CHAPTER XXIV

QUARANTINE ADMINISTRATION

Extracts from the International Sanitary Convention of American Republics

Signed October 1905 ; *Ratified* 1906 ; *Proclaimed* 1909

Article 1.—Immediate notification by the government in whose territory an authentic case of yellow fever has occurred to the other governments.

Article 2.—Information to be furnished to the other governments upon the yellow fever situation, such as place of origin, number of cases, deaths, etc. Distribution of *Stegomyia fasciata.* Prophylactic measures taken.

Article 3.—The above information to be directed to the diplomatic and consular agent in the infected capital of the country, and to officials charged with the public health of the several countries.

Article 4.—Provides for regular weekly report of progress of the fever to the various governments, including in detail the precautions taken to prevent the extension of disease :—

1. Measures of inspection, isolation, and disinfection.
2. Measures taken to prevent the exportation of disease or infected mosquitos on departing vessels.

Article 5.—Strongly recommends making it obligatory upon each government to declare the first case.

Article 6.—Relates to the organisation of a service of direct information between the chiefs of administration upon the frontiers.

Article 7.—Information of a first case of plague, cholera, or yellow fever does not justify against a territorial area when it may appear, the application by other countries of the prescribed measures of defence.

"Upon the occurrence of several non-imported cases of plague, or a non-imported case of yellow fever, or when cases of cholera form a focus, the area is to be declared infected."

Article 8 limits the restrictive measures to the affected region or area, and defines what is implied by "area." But the narrowing down of restrictive measures to a particular area in a country will depend upon the measures taken by the government of the infected country to confine the fever to the particular area, and provided also that Article 1 has been faithfully complied with.

"When an area is infected, no restrictive measure is taken against departures from this area, if these departures have occurred five days, at least, before the beginning of the epidemic."

Article 9.—That an area should no longer be considered as infected, official proof must be furnished :—

1. That there has been neither a death or a new case of yellow fever for eighteen days after isolation, death, or cure of the last case, but each government may reserve the right to extend the period;
2. That measures against mosquitos have been executed.

Note.—"By the isolation in the case of yellow fever is understood the isolation of the patient in an apartment so screened as to prevent the access of mosquitos."

The following articles deal with the defensive measures adopted by other countries against an infected territory :—

Article 10.—The government of each country is obliged to immediately publish the measures which it believes necessary to take against departures either from a country or from an infected territorial area.

Article 11 states that there is no merchandise itself capable of transmitting yellow fever; it only becomes dangerous when it harbours mosquitos.

Article 12.—No merchandise or objects shall be subjected to disinfection on account of yellow fever, but the vehicle of transportation may be subjected to fumigation to destroy mosquitos.

Article 13 states that if merchandise is properly protected, transportation through an infected territory should not debar its entry into the country of destination.

Article 14.—That merchandise should not be prohibited if it were shipped at least five days before the beginning of the epidemic.

Article 15 provides for the authority of the country of destination to fix method and plan of disinfection for destruction of mosquitos, the disinfection to be carried out so as to cause the least possible injury to merchandise.

Article 16.—In case of yellow fever, postal parcels are not to be subjected to any restrictions or disinfection.

Articles 17 *and* 18 relate to merchandise requiring disinfection, more especially in the case of plague.

Article 19.—*Baggage.*—There shall be no disinfection of baggage on account of yellow fever.

Article 20.—*Classification of ships.*—A ship is considered as infected which has plague, cholera, or yellow fever on board, or which has presented one or more cases of plague or cholera within seven days, or a case of yellow fever at any time during the voyage.

A ship is considered as suspected on board of which there have been a case or cases of plague or cholera at the time of departure or during the voyage, but no new case within seven days; also such ships as have lain in such proximity to the infected shore as to render them liable to the access of mosquitos.

The ship is considered indemne which, although coming

from an infected port, has had neither death nor case of plague, cholera, or yellow fever on board, either before departure, during the voyage, or at the time of arrival, and which, in the case of yellow fever, has not lain in such proximity to the shore as to render it liable, in the opinion of the sanitary authorities, to the access of mosquitos.

Article 32.—Ships coming from a contaminated port which have been disinfected, and which may have been subjected to sanitary measures applied in an efficient manner, shall not undergo a second time the same measure upon their arrival at a new port, provided that no new case shall have appeared since the disinfection was practised, and that the ships have not touched in the meantime at an infected port.

When a ship only disembarks passengers and their baggage, or the mails, without having been in communication with terra firma, it is not to be considered as having touched at a port, provided that in the case of yellow fever it has not approached sufficiently near the shore to permit the access of mosquitos.

Article 33.—Passengers arriving on an infected ship have the right to demand of the sanitary authority of the port a certificate showing the date of their arrival, and the measures to which they and their baggage have been subjected.

Article 34.—Packet boats shall be subjected to special regulations, to be established by mutual agreement between the countries interested.

Article 35.—Without prejudice to the right which governments possess to agree upon the organisation of common sanitary stations, each country should provide at least one port upon each of its seaboards, with an organisation and equipment sufficient to receive a vessel whatever may be its sanitary condition.

When an indemne vessel coming from an infected port arrives in a large mercantile port, it is recommended that she

be not sent to another port for the execution of the prescribed sanitary measures.

In every country, ports liable to the arrival of vessels from ports infected with plague, cholera, or yellow fever should be equipped in such a manner that indemne vessels may there undergo immediately upon their arrival the prescribed measures, and not be sent for this purpose to another port.

Governments should make declaration of the ports which are open in their territories to arrivals from ports infected with plague, cholera, or yellow fever.

Article 36.—It is recommended that in large seaports there be established :—

(*a*) A regular medical service and a permanent medical supervision of the sanitary conditions of crews and the inhabitants of the port.

(*b*) Places set apart for the isolation of the sick and the observation of suspected persons. In the *Stegomyia* belt there must be a building or part of a building screened against mosquitos, and a launch and ambulance similarly screened.

(*c*) The necessary installation for efficient disinfection and bacteriological laboratories.

(*d*) A supply of potable water above suspicion, for the use of the port, and the installation of a system of sewerage and drainage, adequate for the removal of refuse.

Article 46.—Ships infected with yellow fever are to be subjected to the following regulations :—

(1) Medical visit (inspection).

(2) The sick are to be immediately disembarked, protected by netting against the access of mosquitos, and transferred to the place of isolation in an ambulance or a litter similarly screened.

(3) Other persons should also be disembarked if possible, and subjected to an observation of six days, dating from the day of arrival.

(4) In the place set apart for observation there shall be screened apartments or cages where anyone presenting an elevation of temperature above 37·6° C. shall be screened until he may be carried in the manner indicated above to the place of isolation.

(5) The ship shall be moored at least 200 metres from the inhabited shore.

(6) The ship shall be fumigated for the destruction of mosquitos before the discharge of cargo, if possible. If a fumigation be not possible before the discharge of the cargo, the health authorities shall order either :—

(*a*) The employment of immune persons for discharging the cargo ; or

(*b*) If non-immunes be employed they shall be kept under observation during the discharge of cargo for six days, to date from the last day of exposure on board.

Article 47.—Ships suspected of yellow fever are to be subjected to the measures which are indicated in Nos. 1, 3, and 5 of the preceding article ; and, if not fumigated, the cargo shall be discharged as directed under the sub-paragraph (*a*) or (*b*) of the same article.

Article 48.—Ships indemne from yellow fever, coming from an infected port, after the medical visit (inspection) shall be admitted to free pratique, provided the duration of the trip has exceeded six days.

If the trip be shorter, the ship shall be considered as infected.

Article 49.—All persons who can prove their immunity to yellow fever to the satisfaction of the health authorities, shall be permitted to land at once.

Article 50.—It is agreed that in the event of a difference of interpretation of the English and Spanish texts, the interpretation of the English text shall prevail.

West Indian Intercolonial Quarantine Convention, 1904

The following are extracts of the Quarantine Regulations adopted by the Convention, and which bear upon yellow fever :—

Infected place means any place where any infection or contagious disease exists ; provided that the place shall not be regarded as an infected place because of all existence thereat of imported cases of such disease, or because of the occurrence of a single non-imported case.

Infected ship means a ship on board of which a case or cases of infectious or contagious disease are present, or have occurred within a period of seven days previous to the date of arrival of the ship, except in the case of smallpox, when the period shall be twelve days.

Suspected ship means a ship on board of which a case or cases of infectious or contagious disease have occurred during the voyage, or during the stay of such ship in the port of departure, but on board of which no fresh case has occurred within a period of seven days previous to the arrival of the ship, except in the case of smallpox, when such period shall be twelve days.

Healthy ship means a ship which, although having come from an infected place, has had on board no deaths from, nor any case of, infectious or contagious disease, either before leaving the port of departure or during the voyage, or on arrival.

Observation means isolation of passengers, either in a proper station provided for that purpose or on board ship, prior to their obtaining free pratique.

Surveillance means that passengers are not isolated. They receive free pratique at once, and are allowed to proceed to their place of destination (the proper authority of which must be informed of their arrival), there to undergo medical supervision.

By Article 3 a place has ceased to be regarded as infected if the health officer is satisfied :—

(*a*) That there has been no new case of yellow fever within six days.

(*b*) That measures have been taken with a view to the destruction in the infected locality of mosquitos on and near the infected premises.

By Article 5 infected ships shall be dealt with as follows :—

(*a*) The sick shall, as soon as possible, be removed from the ship and isolated.

(*b*) The other persons on board shall be permitted to land, and be kept under observation, or subjected to surveillance.

(*c*) When observation is resorted to, the period shall not exceed six days in the case of yellow fever.

(*d*) When surveillance is resorted to, the period shall be the same.

(*e*) In applying these measures, the date of the last case and the condition of the ship to be taken into account.

(*f*) In the case of yellow fever, measures shall be taken to secure the destruction of mosquitos and their larvæ on board.

By Article 6 suspected ships shall be dealt with as follows :—

(*a*) The passengers and crew subjected to surveillance during a period which shall not exceed six days in the case of yellow fever. The period to date from the arrival of the ship.

(*b*) That measures should be taken to secure the destruction of mosquitos and their larvæ on board. When such measures as the health officer may have deemed necessary in accordance with the provisions of this article have been carried out, such ship shall immediately thereupon be admitted to free pratique.

By Article 7 healthy ships shall be admitted to free pratique immediately on arrival, irrespective of the nature of their bill of

health. They may, however, at the discretion of the health officer, be subjected to the measures specified in paragraph (*f*), Article 5, and the passengers and crew may be subjected to surveillance during a period of six days in the case of yellow fever. The period of surveillance shall date from the departure of the ship from the infected place.

By Article 10 a ship shall not be regarded as having called at a place if it has merely disembarked passengers and their baggage, or mails, without having been in communication with the shore.

Ships from an infected place which have been disinfected shall not again be subjected to sanitary measures on their arrival in another port, if in the opinion of the health officer of such ports the measures applied were effective, unless a fresh case of infectious or contagious disease has occurred on board since disinfection, or unless they have again called at an infected place.

By Article 12, where measures of observation or surveillance are prescribed, the health officer may exempt from their application any person who is, in his opinion, immune to the infectious or contagious disease on account of which these measures are applied.

By Article 13, where these regulations provide that a person may be permitted to proceed to his place of destination subject to surveillance, the health officer, before granting such permission, must be satisfied that it is reasonably probable that the person to whom it is granted will duly comply with the conditions of surveillance, and permission, if granted, shall be upon the following conditions:—

(*a*) He must satisfy the health officer as to his name, intended place of destination, and his place of residence thereat.

(*b*) He must agree to present himself, and shall present himself for medical supervision during the prescribed period, and he may be required by the health officer to

deposit a sum not exceeding two pounds, which may be forfeited if he fail to present himself.

(*c*) The place must, in the opinion of the health officer, be conveniently situated for the medical supervision.

If the health officer is not satisfied as herein required, or if the person fails to comply with paragraphs (*a*) and (*b*) hereof, the health officer may detain him under observation, or direct him to proceed to a specified place, and there remain under medical supervision during the prescribed period.

In the latter case, the provisions of paragraph (*b*) hereof may at the discretion of the health officer be applied to such person.

In the case of a healthy ship the measure authorised by the foregoing proviso must not be applied to passengers who have not embarked or gone ashore at the infected place, and it should not be applied to those passengers who embarked or went ashore at the infected place, if circumstances of their stay there afford reasonable evidence of their non-infection.

By Article 14 merchandise shall be only disinfected when in the opinion of the health officer it is infected, provided that in the case of yellow fever merchandise shall under no circumstances be liable to disinfection or prohibition.

By Article 16 nothing in these regulations shall render liable to detention, disinfection, or destruction, any article, forming part of any mail, other than a parcel mail, conveyed under the authority of the postal administration of any government, or shall prejudicially affect the delivery in due course, of any such mail, other than parcel mail to the post office.

By Article 17 when any port within the colonies is an infected place, measures shall be taken to prevent the embarkation from such port of any person showing any symptoms of infectious or contagious disease. To this end every person taking passage on a ship leaving such port shall be examined by the health officer immediately before the departure of the vessel. Such examination shall, as far as practicable, be made by day on the shore. Measures shall be taken to prevent mosquitos, in the

case of yellow fever, from gaining access to ships. When access of mosquitos to the ship cannot be prevented, measures should be taken immediately before the departure of the vessel to secure the destruction of the mosquitos on board. The health officer shall give to the master of the ship a certificate stating in detail the measures taken.

By Article 18, when, in the case of a healthy ship from a port which is an infected place, the health officer at the port of arrival is satisfied that the measures certified in Article 17 have been efficiently carried out, such ship shall be exempted from the measures specified in Article 7. Provided always that if the period specified in that article, and dating from the departure from the infected place, shall not have been complete, the passengers and crew shall be subjected to surveillance of such duration as is necessary to complete the period.

Comparisons between Articles of the West Indies and the Pan-American 1905 *Sanitary Congresses*

Both conventions agree that:—

(1) A central quarantine authority; (2) observation stations (quarantine stations) and isolation hospitals; (3) abolition of fees; and (4) compulsory notification are essential.

Definition of:—

Infected place.—Both conventions agree in regarding imported cases as not causing a place to be declared infected, Also that a single first non-imported case does not cause a place to be declared infected (Article 7, Pan-American Convention).

Infected ship.—Here there is a difference between the two conventions. By the West-Indian Conference the ship is infected if a case of yellow fever has occurred within seven days previous to date of arrival. In the Pan-American Convention a ship is infected if yellow fever has occurred at any time during the voyage (from last port). This is no doubt necessary, as, if there are mosquitos on board, the infection may

be transmitted to them from the patient, and twelve days afterwards and onwards for a considerable time they may be able to transmit the disease.

Suspected ship.—This also differs. In the case of yellow fever the ship which is regarded as suspected by the West-Indian Convention would be classed as infected by the Pan-American. By the latter authority a suspected ship is one on which, although no case of yellow fever has occurred at any time during the voyage, nevertheless has lain in such proximity to an infected shore as to render it liable to the access of mosquitos (*vide* Article 20).

Healthy ship.—By Article 20 of the Pan-American Convention, it is only when a ship has had no case of yellow fever during the voyage, and has not lain in such proximity to the shore as to render it liable to mosquito infection, that it is declared "indemne."

Observation, surveillance.—Both conventions agree upon the absolute necessity of proper observation stations, and isolation stations. There does not appear to be anything in the Pan-American Convention dealing with surveillance. I think that the question of surveillance should be favourably considered under certain circumstances.

Observation.—The definition of the term is similar in both conventions.

When a place ceases to be infected.—Here there are also substantial differences. By the West-Indian Convention there should not have been a new case of yellow fever within six days. By the Pan-American not until eighteen days after isolation, death, or cure. Both agree that anti-mosquito measures must have been adopted.

How infected ships are to be treated.—With regard to the treatment of the sick, both agree that they are to be removed at once. But the Pan-American Convention is very precise upon how the yellow fever patients are to be isolated and removed. These very important instructions are omitted in

the West-Indian Conference. With regard to the other passengers, both conventions agree that the passengers should land and be kept under observation (in quarantine) for a period of six days. The Pan-American Convention indicates how the observation station is to be protected. The West-Indian Convention also allows a period of six days' surveillance.

Place where the infected ship is to be moored.—The Pan-American Convention lays down that ships must be moored at least 200 metres from the inhabited shore. In the West Indian there is no mention of this.

The fumigation of the infected ship.—Both agree that this must be done.

Suspected ships.—As there are differences in the definition of suspected ships as regards yellow fever, in the case of the two conventions, strict comparison cannot be made. Were, however, the definition the same in the Pan-American Convention the treatment would nearly coincide in the two cases, except that six days' surveillance is substituted for six days' observation by the West-Indian Conference.

Healthy ships.—Here there is a difference. By the West-Indian Conference the ship is admitted to free pratique immediately, irrespective of the nature of the bill of health, and discretionary powers are given the health officers to fumigate and to exercise surveillance of passengers and crew. By the Pan-American Convention the trip must have exceeded six days, and the ship must have been medically inspected before departure.

Ships which have "called" at intermediate points.—Article 10 of the West-Indian Conference and Article 32 of the Pan-American agree that vessels are not regarded as having called at a place if they had not been in communication with the shore (see Article 32), and only disembarked passengers, baggage, and mails.

No necessity for second fumigation.—In the same article there is agreement about non-necessity of second fumigation, pro-

vided that no new cases shall have appeared, or that the first fumigation was carried out efficiently.

Immunes to be exempted.—The exceptional position of the immune is recognised by both conventions; compare Article 49 (Pan-American), and Article 12 (West Indian).

Treatment of merchandise.—Both agree that merchandise be not disinfected: Article 12 Pan-American Convention, and Article 14 West-Indian Convention. But in Article 12 (Pan-American) it will be seen that the vehicle of transportation may be fumigated.

Mails.—Both agree are not to be fumigated.

Baggage.—Both agree are not to be fumigated.

Departure of passengers from an infected area.—There are differences here between the precautionary measures to be taken according to the two conventions. By the Pan-American Convention no restrictive measures are to be taken if the departures have occurred at least five days before the commencement of the epidemic. The West-Indian Conference provides simply for the examination by the medical officer immediately before the departure of the vessel.

Regulations affecting passengers travelling under surveillance.—There is nothing in the articles of the Pan-American Conference dealing with permission being given to travellers to proceed to their destination, as is the case with the West-Indian Conference in Article 13.

Prevention of access of mosquitos to ship.—In Article 17 of the West-Indian Conference it is stated that measures are to be taken to prevent mosquitos gaining access to the ships, and if this is not possible, that measures "should be taken" to secure the destruction of the mosquitos on board immediately before the departure of the vessel. Nothing is said as regards proximity to shore—a point laid stress upon by the Pan-American Conference.

Note.—Cessation of infection. Very considerable difficulty has been found to occur in practice over Article 3 of the West-

Indian Convention. That is to say, to determine when a place shall cease to be infected. I have repeatedly pointed out that this will entirely depend upon the degree of reliance which can be placed upon the anti-mosquito measures which the infected country has undertaken. For as mosquitos may long remain infected, it is obvious that only the most careful fumigation will get rid of any infected *Stegomyia*. Experience in many places in recent years shows that isolated cases of yellow fever frequently appear months after an outbreak was supposed to have been stopped.

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