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MARTIN.

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A Monograph

BY

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E. & S. LIVINGSTONE,

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TO

LOUIS WYATT, Esq.,

WHOSE ACQUAINTANCE I MADE ON THE GOLD COAST, AND TO WHOM
I AM INDEBTED FOR MUCH KINDNESS;
ALSO FOR THE
HIGH RESPECT HIS THOROUGH KNOWLEDGE OF LIFE AND
CIRCUMSTANCES IN HOT COUNTRIES EVER CLAIMS;
AND TO THOSE WHO
HAVE TO REMAIN MANY PERILOUS MONTHS IN SUCH REGIONS,
THIS VOLUME IS

Respectfully Dedicated.

PREFACE.

THE greater portion of this work formed the substance of a Paper read before the Royal Medical Society of Edinburgh some years ago; and it is published with the kind permission of the Society.

In bringing the subject of my dissertation before the profession generally, I make no apology on the score of the paper dealing with a disease practically unknown within our shores, and therefore one we are not likely to see and study for ourselves during our years of preparation here.

I content myself with treating of a disease of vast importance to the populations within and near tropical zones, whether of the Old World or of the New; and to those who travel to distant parts, whether as members of the profession or in any other capacity, who will encounter there the disease in some form or another.

EDINBURGH, *November 1891.*

YELLOW FEVER.

IN these days of germ research a fever of this kind must be of commanding interest to scientific workers, who are seeking to ascribe to such organisms the very origin of epidemic diseases; and, as a consequence, therein must lie the secret of their cure, if not also of their prevention.

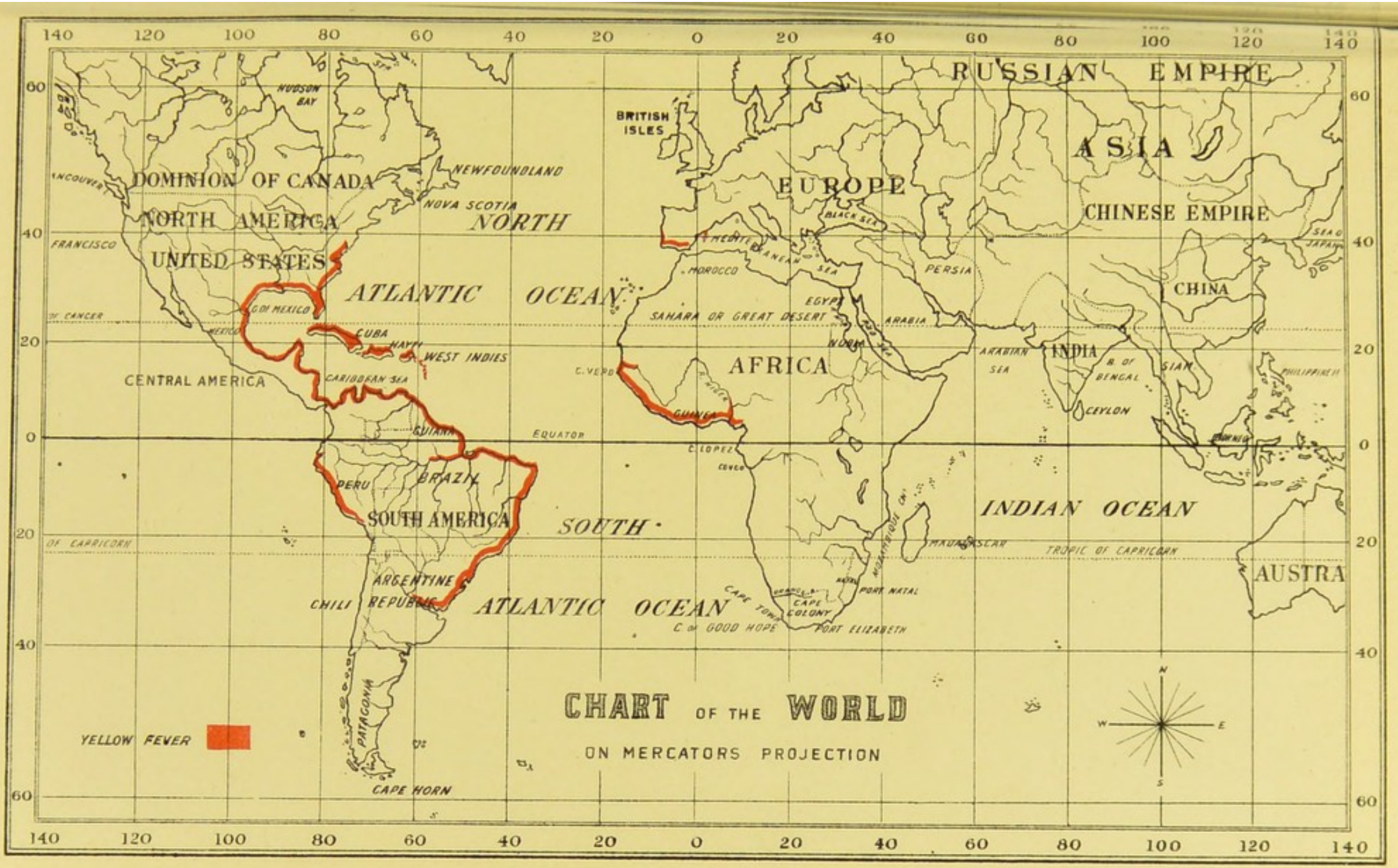
Definition.—The definition of yellow fever is shortly as follows:—It is an epidemic fever, malignant in its kind, being either continued or paroxysmal, and characterised by yellowness of the skin and conjunctiva, together with hæmorrhages from various surfaces and orifices, and occurrence of the fatal "black vomit."

Nomenclature.—The nomenclature is as follows—It was known in Latin as *febris flava;* the French call it *fievre jaune;* the Germans, *gelbes fieber;* the Italians, *fiebra gialla;* the Spanish, *el vomito;* and for a local name it has *vomito amarilla* and *calcutura*—for example, in Central America, *vomito prieto.*

Distribution. — Yellow fever has a somewhat limited distribution, and on the map of the world, with Mercator's projection (see Map), you see that it is only found within a comparatively small section of the habitable globe, occurring on the sea-coasts and seaboard. The imaginary lines on the earth's surface

which comprehend the area are 45° N. Lat., 10° S. Lat., 97° W. Long., and 2° E. Long. It is thus almost entirely confined to the tropics, and to one hemisphere of the globe. It does not exist where the temperature of the atmosphere is below 72°. It has not been found much above 4000 feet from the level of the sea, a high altitude being inimical to it. It abounds where there is a dense population, and in low-lying foul foreshore harbours, where the water is stagnant and the wharves are undrained and filthy, especially where the sun is in the tropic of Cancer.

Historical and Geographical.—Yellow fever is said to have been the "golden fever" which attacked the followers of Columbus, when he visited the Spanish Islands, about the year 1492 (see "Historia General" of Ovido). That it was already known to the natives of those islands there would seem little doubt (see "Rouchoux"). Tradition says it was brought from Siam, in Asia, by a fleet which touched at Martinique. The first great epidemic of which we have record is that which visited Barbadoes in 1647 (see "Lignon"), and the disease has appeared there every fifteen to thirty years since. The epidemic of 1691 was a very fatal one. In 1651 a great plague visited Barcelona and Spain, which proved to be yellow fever. The English army during the conquest of Jamaica, in 1655, suffered very much from the fever. In 1721 it was at Barbadoes, in 1729 at Carthagena. In 1762 and 1793 there were epidemics at Philadelphia: the latter year is memorable, it was in the West Indies at Dominica, and Barbadoes; it was at Grenada, and in the north at Charleston and Philadelphia. Next year it was at Guadaloupe, Martinique, St Lucia, and St Domingo, where 6000 perished out of 10,000


RUSSIAN EMPIRE
ASIA
EUROPE
CHINESE EMPIRE
BRITISH ISLES
HUDSON BAY
NEWFOUNDLAND
NOVA SCOTIA
DOMINION OF CANADA
NORTH AMERICA
NORTH
ATLANTIC OCEAN
UNITED STATES
BLACK SEA
MEDITERRANEAN SEA
MOROCCO
PERSIA
CHINA
SEA OF JAPAN
SAHARA OR GREAT DESERT
EGYPT
ARABIA
RED SEA
NUBIA
AFRICA
ARABIAN SEA
INDIA
BAY OF BENGAL
SIAM
PHILIPPINES
CEYLON
BORNEO
CUBA
HAYTI
WEST INDIES
GULF OF MEXICO
CENTRAL AMERICA
CARIBBEAN SEA
C. VERD
GUINEA
GUIANA
EQUATOR
C. LOPEZ
PERU
BRAZIL
SOUTH AMERICA
SOUTH
INDIAN OCEAN
MADAGASCAR
TROPIC OF CAPRICORN
AUSTRA
ARGENTINE REPUBLIC
CHILI
ATLANTIC OCEAN
CAPE TOWN
CAPE COLONY
PORT NATAL
C. OF GOOD HOPE
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inhabitants. The 82nd and 92nd Regiments lost over 800 rank and file. It was at New York, Newhaven, Baltimore, and Charleston. Humboldt tells of it being at Vera Cruz the same year. In 1794–96 it was at Dominica, being brought by emigrants from Martinique; the English sailors suffered first, then the emigrants, then the new comers to the island; the attendants and physicians were not so much attacked. In 1799, Trotter describes it being in the West Indies. It was at Gibraltar and Tangiers, at Cuba, Cette, and Genoa. In 1800, it was at Cadiz and Port St Mary; at Xeres, where 10,000 perished; and at Seville and Malaga. At Barrio di Santa Maria 7000 perished, and the air is described as being "stagnant and vitiated, and the very animals died." In 1801 yellow fever was at Cadiz, Seville, and Medina Sidonia. It was also at Martinique and Dominica. The epidemic of 1804 was very fatal at Gibraltar: the garrison lost 864 out of 3500, and the inhabitants 4864 out of 15,000 to 16,000 souls. It was also at Malaga. In 1811 it was at Carthagena in Murcia. In 1812 Alicante suffered, as also Cadiz, but the 500 out of 700 recruits who were smitten there were from Alicante. In 1814 it was partial in Gibraltar. From 1817 to 1818 the West Indies suffered. In 1819 and 1822 there were epidemics at New York. The year 1819 saw pestilence all over its area. It is to be noted in these last years that there had been much war with all the miseries attending it. Gibraltar suffered from yellow fever in 1821, but the epidemic soon died out. It was in Sierra Leone, Gambia, and Gorée in 1823, 1829, 1837, and 1847. In 1850 there were a few cases at Corfu. In 1856 it was at Lisbon, where it destroyed 5652 of the inhabitants. In that year

it raged at Newcastle in Jamaica, and it was also at Charleston. In 1837 and 1838 it was in British Guiana. In 1842 at Guiaguill on the Pacific coast of South America, 3° S. Lat., and again in 1852. In 1847, at New Orleans. In 1849 and since, at Bahia and Coast of Brazil, and it was noted that the collier vessels suffered most. In 1850, on the coast of Brazil, and in 1856 at Cayenne, where it appeared in the months of February and March. 1858 was the year of the outbreak at St Thomas. In 1828 there was an outbreak at Gibraltar, and Dr Hesmen describes "the fatal sheds," which the men locked up while they were absent during the day. The years 1859 and 1866 are memorable at the River Gambia at Bathurst, when half the European population died of it. In 1855 and 1859 it was at Sierra Leone, when the town suffered. In 1870 there was yellow fever at Havanna. In 1871 there was fever at Buenos Ayres, Paraguay. 15,000 fell victims in South America in 1871. 1879 was the year of the great epidemic at New Orleans and Memphis, U. S. A., where there were 12,000 deaths. There are, at the same time, historical accounts of outbreaks on board-ship, and the names of the "Kant," "Scout," "Bann," "Icarus," and the "Eclair," are often referred to. The "Icarus" lost 39 men out of 110, in the Gulf of Mexico; and I shall say something of the others again. These are but instances of the epidemics which have prevailed, and which literature has placed on record.

It may thus be noted how the scourge of yellow fever is found within definite areas, although it has reached Spain in Europe; it invariably follows in the train of commerce, and it is to be remarked how it has augmented in recent decades.

Clinical History and Symptomatology. — Yellow fever is a disease of the warmer months of the year—viz., June, July, August, and September. It attacks the patient in the night or early morning, when he becomes unwell, complaining of coldness, shiverings, or rigors. There has been a period of incubation from two to twelve days, and now the symptoms begin to develop themselves. There is first a kind of reaction from the coldness, and the patient will be suffused with a glow of warmth. Then he complains of headache. There is a slight injection of the eyes and conjunctivæ, hot skin, pulse 92, pains in the orbits and loins, stupor; tongue white in the middle and red at the tips and edges; thirst, nausea, and epigastric uneasiness. This is the congestive stage. From day to day the symptoms increase; there are more severe pains in head and loins; there are flushes, cold sweats, the eyes are bloodshot, suffused, and "ferrety;" the face of the patient is striking, being a compound of fear, anguish, and astonishment. The temperature is now about 107°. There is jaundice and yellowness of the skin and conjunctivæ, and the pulse is rapid and full; there is suppression of urine. There are head symptoms, the voice and movements are tremulous, the speech is interrupted by sobs (singultus); there is increased distress about the epigastrium, and a tendency to vomit, which soon commences, at first white and acid, then suddenly supervenes the "black vomit," which is deoxydized and disintegrated blood, and the scene is quickly closed if the case is to terminate fatally. The whole occupies typically a space of seven days. Death may, however, be speedy, occurring on the third or fourth day, with black vomit, and a bad case has gone on till the fourteenth day. The case terminates by a

crisis, and if on the fifth or sixth day there occurs a discharge from the bladder and bowels, the patient is relieved, and passes into convalescence. There is, however, often a deceitful remission before the black vomit. The suppression of urine and bad head symptoms, insomnia, delirium, etc., invariably lead to death. Dr La Roche says "it is a disease of a single paroxysm," but some authors have noted periodicity. There is invariably the appearance of albumen in the urine, and when that is so, quinine has no power in the treatment of the disease.

The Types of the Fever cannot be stated at less than six :—1st, Mild and incomplete—no severe symptoms. 2nd, Complete, dangerous and of long duration. 3rd, Very dangerous and short; the voice and movements are tremulous, and death takes place on the third day. 4th, Yellow fever, whose chief characteristic is pain in the epigastrium and vomiting. 5th, Yellow fever, with critical symptoms predominant, as hæmorrhages, petechiæ, etc. 6th, Yellow fever, with cerebral symptoms.

In taking up a type—*e.g.*, the 2nd (complete)—there is often a history of exposure to the sun and rain; colds, sweats, flushes, pains in head and loins set in; on the second day, frontal headache; on third, fourth, and fifth day, there is an increase of symptoms, with jaundice; on seventh day, eyes markedly blood-shot; and on ninth day, black vomit, cold sweats, and death terminates the case.

The occurrence of the black vomit and the adynamic stage is *not incompatible* with continuance of muscular power, intellectual activity, self-possession, and courage (see La Roche, Philadelphia, 1835).

Cause.—The disease is set up by a specific poison which enters the system, and which is carried everywhere by the blood-stream. The blood is polluted, and therefore the morbid changes in the abdomen. The organism seeks to throw off the deleterious substance developed in the body, and in so doing the liver, the stomach, with the intestine and the kidneys, are specially taxed. The nervous centres are morbidly impressed, and through the sympathetic the diseased phenomena with their modifications arise, and these are essential to the manifestations of the fever. In different individuals they vary in violence, and likewise under different circumstances. The phenomena of the fibrile action can be explained. For example, the muscular contractions. There is accumulation of carbonic acid in the blood and muscles, and the nerve centres and spinal cord are thus stimulated. There are phenomena arising from implication by all the systems; but there is an organ excepted, which does not seem to play a part, namely the spleen. It would seem that the process is altogether too rapid and violent for the spleen to be affected in the way of retrograde metamorphosis. We shall subsequently see how the functions of the liver and other abdominal organs are so disturbed. The nature of yellow fever then is pestilential; and there is first a phase of reaction against the poison, which is infectious; and second, a phase of depression and adynamic ataxia, when the bodily organism wages such unequal strife, and is alas so quickly overpowered.

Pathology.—There is first the congestion of organs brought about by the development of the poison within the body. There may be hæmorrhages from various organs and surfaces, and petechiæ are

common. There is epistaxis, also intestinal hæmorrhage, and in the female hæmorrhagic discharge from the uterus.

The Yellowness of the Skin is not altogether, in some cases not at all, connected with biliary obstruction —the gall bladder and ducts being free. It is said to be due to a condition similar to that of a bruise; the corpuscles of the blood are broken up, there is colouring matter in the serum, the amount of which is small. The fibrine is below the normal standard; there is a certain amount of defibrination, and there is some bile superadded. The liver is engorged; there are functional modifications, and the elimination of bile element is arrested—this is irrespective of textural changes, for these come after, if at all. There is an alternation in the hæmatin of the blood corpuscles, and the hæmatin explains the phenomenon; the serum of the blood becomes yellow, and no cause of biliary absorption is exhibited. The elements of the bile are not merely separated from the blood by the healthy liver, but there is a further process in the liver, not as in the kidney, where the elements of the urine are separated.

The Hæmorrhages are not due to vomiting; black vomit is blood altered by hydrochloric acid. The blood is greatly deoxyginated, and, as Dr Parkes has pointed out, it is greatly defibrinated.

The Blood is dark and of the consistence of molasses; it does not coagulate, and when the crassamentum appears it is as if it were undergoing solution at the base. The serum varies in colour. It contains colouring matter and disintegrated corpuscles. The amount is small. The odour is characteristic, and Davy has shown it to be acid. Chassaniel has shown

that there is much urea in the blood in the third stage. I have already referred to the symptoms of urea poisoning at the approach of death. The fibrine is below the normal standard. The colour of the skin would seem to have to do with the black vomit. The face is lemon-chrome colour, and the colour overspreads the body, the discoloration first beginning at the forehead. The tendency to hæmorrhage has already been noticed, and it may be from the skin, nose, eye, ear, mouth, tongue, gums, lips, stomach, uterus, bladder, and rectum. It is altogether as though the body were under great internal pressure. In the blood of yellow fever, dumb-bell bacteria have been seen (see page 34).

From the stomach there are three vomits—first mucus and bile; second, white vomit alone, which is markedly acid; third, black vomit. This may occur on the third or fourth day. There are at first snuff-coloured brown streaks, as if accidentally marked.

When the black vomit is acted upon by caustic potash (KHO), it is neutralised and there is a flocculent sediment, most likely the other ingredients besides the hydrochloric acid,—viz., albumen, alkaline bases, phosphates, iron,—and over and above, sulphuric acid and chlorine are met with. When treated with carbonate of potash (K_2CO_3), there is effervescence, and ammonia is given off. I may here mention the elements of normal blood: the blood cells and plasma of venous blood contain, in addition to water and organic solids, inferior quantities of chlorine, anhydrous sulphuric acid, phosphates, phos. pentoxide, sodium, potassium, calcium phosphate, magnesium phosphate, and oxygen —iron being excluded. Thus there are most of these elements in black vomit. This is important, for as

I shall presently show the blood in the stomach is not due to rupture of vessels.

When one examines the blood from the stomach microscopically, one sees much columnar and glandular epithelium, and some few spherical and corrugated cells, one-half the diameter of blood discs, colouring matter brown, amorphous, spores, torulæ, and other extraneous matter, but no blood globules. The colour of the discharge is that of venous blood.

Macroscopically, the mucous membrane of the stomach is often deep brown. Microscopically one sees tubular glands and epithelium in a granular condition, and impregnated with brown colouring matter, as well as granules pretty free from it. One also sees the vessels with entire blood corpuscles. Thus it is a true, though morbid, secretion which is vomited.

There is a condition of cloudy swelling in the cells, and the same occurs in other organs. The stomach is occasionally contracted, the mucous membrane is thickened and friable, and sometimes patches of congestion are seen here and there, or they may be confined to one end only. The duodenum is usually implicated. In the ilium, colon, and jejunum also, there is much columnar epithelium shed. The fluid called the black vomit, as well as being secreted from the tubular glands of the stomach, can in like manner be secreted by the kidney. Various parts of the intestine can discharge dark material, and it is seen opposite Peyer's glands to be of a dark brown or reddish hue. Many glandular organs are thus seen to throw off a fluid like "black vomit."

Liver.—This organ is nearly always pale yellow, nankeen, buff, or straw-coloured, and when washed it does not give out colouring matter, nor is there bile

in the ducts. As has been said, the jaundice is not altogether a biliary congestion. The organ is enlarged and congested, weighing from 3 lbs. 12 ozs. to 4 lbs., and is of diminished firmness and cohesion, being friable and otherwise abnormal. It may be fawn-coloured, yellowish, or greyish-brown; or again, it is sometimes like the rhubarb or nutmeg liver. Oil, blood, and bile may be found in it. When there is no oil it has a nutmeg liver appearance under the microscope; when there has been tenderness during life, it is found olive and green coloured. The peritoneal covering is opaline and adherent; pus may also be formed on the surface. The gall bladder is found to contain a quantity of bile; and a notable amount finds its way down into the intestines. The liver is not strictly speaking *inflamed* in the disease.

Microscopically.—The lobules are distinctly seen with their cells, and that of normal shape. The portal vein with its branchlets can be seen between the lobules, and between the branchlets, layers of connective tissue; and between them and the lobule an opaline substance, interposing between the vein and the accompanying artery and duct. The small vessels are covered with nuclei and granules, and by their connective tissue with granulation matters, and exudation. Bile ducts are at times opalescent, and do not seem to be pervious. In these cases there is jaundice. The connective tissue with the lobules is to be noted—a columnar network lying between the tubules of the cell containing network, and towards the margin of a lobule. The cells are deep-brown coloured. Bile is at times seen in a lobule; the nuclei are not always distinct, as if between the tubes of cell containing network. The cells are frequently without oil

globules or very small mesially. The liver is often fatty with oil globules, which are contained in the diameters of the tubes. The cells are frequently altogether without oil globules—which are always attached to and imbedded in the hepatic cells. There are hepatic cells in the hepatic ducts, as pointed out by Wharton Jones, and these may also exhibit the changes referred to. The deposition in the liver is physiological and altogether unconnected with the disease. There is granular matter in the portal branches, and there is an active exudation into the parenchyma of the organ. The minute bile ducts are embraced and closed against the passage of the bile. It is to be noted that in this disease, the yellowness is not seen at the very first. Bile is found in the urine and in the serum effused within the cranium. I have stated that the yellowness is due to capillary effusion and destroyed blood, something after the manner of a contusion. The cloudy swelling in the hepatic cells may lead to fatty degeneration.

Kidneys.—The kidneys are congested, and weigh from 5½ to 8 ozs.—intermediate portion, light greyish yellow. Pyramids contain tube casts as milky-looking matter. Sometimes interstitial effusion is met with.

Microscopically, the epithelium of the convoluted tubules is granular; the outlines of the cells are for the most part indistinct, and the nuclei difficult to detect. It is thus readily seen how urine is obstructed. The cells are sometimes tinged with hæmatin, the pigment often covering the epithelium as minute granules at intervals along the tube. The capillary vessels by the side of the tubes are always covered with nuclei. When cleared by acetic acid the nuclei are seen to be granular. Compare this condition with that of

cloudy swelling and the similarity is seen to be striking. Loops of malpighian vessels are similarly affected, and appear quite distinct on the larger vessels in the interior of the tuft. The capillaries are thus tumid or dark grey. The connective tissue around seems to be generally swollen, and contains clear or opaline exudation and often granular matter, with much exudation. Around are straight and convoluted tubes, with granules, oil, and pus. The whole are clouded and indistinct. The exudation on the parenchyma of the organ, and catarrh of its mucous surfaces must also be noted. The passage of the urine is prevented. This usually occurs about the fifth day, and the secreting power may be entirely destroyed.

Urine.—The excretory process is hindered by the rapid degeneration and desquamation of the tubular epithelium, and by the tubules being greatly clogged with minute organisms. Dr Porcher, in his "Careful Microscopical and Chemical Examination of the Urine during the Epidemic of Yellow Fever in Charleston, 1856," proved the constant absence of urea and uric acid. To this failure of elimination he attributes some of the dangerous symptoms—*e.g.*, coma, and indications of brain torpor in the later stages.

The urine voided during the illness is found to contain many abnormal products, the most noticeable among these are albumen, globulin, and casein. In testing for urates, it is found that when the urine is treated with acetic acid a precipitate forms, which precipitate is dissolved at a low temperature, and this treated with carbonate of soda gives uric acid. The following table of tests has been devised to discriminate between the albumen, casein, and globulin.

	Heat.	Nitric Acid.	Acetic Acid.	Sodium Carbonate.
Albumen,	√ at 140°	√	o	o
Globulin,	√ at 200°	√	o	√
Casein,	no precipitate	√	√	√

These tests have been found successful in examining the urine in yellow fever (Lawson on "Yellow Fever," 1859), and the above mentioned substances may occur and yet there be no blood in the urine. Collins, writing from Barbadoes, 1848, gives cases with albumen in the urine; this was on the fifth day, and when the albumen was copious casein disappeared. Gallic acid is given to reduce casein, and waxy casts are formed. It was remarked that when the liver was involved there was jaundice, but the urine had less albumen. The urea was also deficient: that would point to defective elimination, if not to less tissue metabolism (increased). Creatin is copious. Examined on a slide, there are seen needle-like crystals, and also small rhomboid plates — hexagons with the angle removed — the needles present a swelling in the middle of the original hexagon. The prolongation of the needle is triangular, and there is a nacreous appearance peculiar to creatin. It is soluble in the following substances—water, ammonia, nitric acid, hydrochloric acid, acetic acid, but not in alcohol. There is likewise creatin, and the peculiar dendriform masses of navicular crystals. It is removed by ether. There is tyrosin, and this is not removed by alcohol. There is uric acid frequently, when the urea is diminished and the chlorides are absent. The disappearance of the chlorides and the reduction of urea is to be noted. Hippuric acid is copious. There is desquamation of the bladder on the fourth day, followed by that of the kidney. Suppression of urine is most to be

feared on the fourth and sixth days. From the third to the fifth day there is diminution of the quantity of urine passed, and it may be only 12 ozs. in twenty-four hours. The colour varies from 4 to 7 of Vogel's scale. The specific gravity 1008–30 at 60°. It is acid and has sediment. The scaly epithelium from the bladder is noted, and the day after granular tube casts from the kidney. They become hyaline on the sixth day. Waxy casts are early seen. These are not soluble in acetic acid. The urine may be dark with hæmatin or globules. The hæmatin is with the epithelium of the casts with which it is secreted. The blood denotes local depletion. The urine is often coloured deeply by bile. Uræmatine, uroxanthine, urhodine, and uroglaucine have been detected as some of the pigments. It has been noted that when these pigments are less in quantity the epidemic is less severe; and that we can understand, for the poison is acting with less virulence, and exercising a less destructive influence upon the blood. I should have noted further the vicarious elimination effected by the stomach, that there is much hydrochloric acid in the stomach, and the chlorides are less in the urine.

The *Alvine Evacuations* are feculent at first, with an admixture of mucus and a melanotic matter of disagreeable odour. Then follows the "caddy stool" which is liquid, light coloured, depositing a grey sediment containing crystals of phosphates (triple), uric acid, and amorphous masses of black opaque matter (see Blair's Report, Demerara, 1856). There is afterwards a clear mucous stool with broken up epithelial matter and myriads of epithelial granules, either uncoloured or variously tinted yellow or green or black, with blood. This is contemporaneous

with diminished urine and "black vomit." The elements of blood may be copious. There is a want of brown colouring matter, supplied by the colon. Note the light-coloured evacuation with the black vomit. The secretion of the colon should be excited if possible, for it makes the stool more natural and helps the patient. The blood may be like the black vomit. High up the gut the mucous membrane of the ilium and colon and jejunum is congested, swollen, and easily abraded. The following differentiates the chief "black vomits:" that from the stomach has mucus, small granules, and granular cells; that from the intestine, columnar cells in abundance; and from the colon, casts from its tubular glands.

The Head Symptoms have been noted, and consequently changes in the brain and its membranes are expected. The vessels in the membranes are full; the pia mater is opaline from interstitial effusion; yellow serum is found at the base of the brain and in its ventricles. If the brain is sliced, the red points seen are very numerous. Its consistence is less firm than usual. Under the microscope one sees large exudation corpuscles, numerous in the white matter near the surface of the hemispheres, and small vessels and capillaries covered with nuclei — which are most probably the swollen nuclei of endothelial cells, for they are closely set. (Compare with kidney and liver.) It is difficult to see the exudation, but exudation is present, and exudation corpuscles. These are characteristic of stasis. Amyloid bodies which are exudation products, are seen with firmness and with fracture, and under pressure flattened. It is rare to have softening of the upper part of the cerebellum. [One case is cited, that of Corporal Brownlow—7th August 1859—with severe

head symptoms, and delirium followed by death, no yellowness, as post-mortem appearances proved.] You are in a dilemma as to the treatment. You seek to stop the fever, for every paroxysm increases mischief to the brain, and if you are not cautious with your remedies you are very likely to increase the cerebellar affection.

Heart and Blood Vessels.—Heart pale and soft, cavities distended, they contain blood and lymph coagula, the serum is yellow; sometimes coagula are seen extending up into the pulmonary artery and forming moulds. The coagula are largest when the liver is very severely affected; less when kidneys, intestinal canal, and brain are implicated. Fatty heart is not a consequence of the disease. Yellowness of the serum is due to bile. There are indications of urea in the serum of the brain.

Lungs.—Sometimes healthy, sometimes show acute active disease. Congestion, with bloody mucus in the tubes, accompanied with pneumonic congestion may be seen. Pneumonic condensation of a portion of the lung, of greyish-yellow colour, and surrounded by congestion is met with. The air cells are seen filled with epithelial cells in a granular condition. There is no exudation into the pleura. (See Lawson on "Yellow Fever," 1862.)

Acclimatisation.—Yellow Fever certainly shows a considerable preference for Europeans. American Indians and Coolies are said to be attacked by it, but some authorities affirm that the negro race is immune. At any rate, according to Fenner, they do not suffer from the worst effects of it. The Chinese, too, seem to suffer very slightly from it. Dutroulau said "The chances of immunity," in a stranger population,

"appear to be always in direct proportion to the length of residence at the headquarters of the disease; but no acclimatisation is acquired except by those who have lived through a previous epidemic without leaving the country, and who have been more or less impregnated by the epidemic principle, and most of all by those who have survived an attack of yellow fever."

I do not intend to go fully into these questions, but it certainly appears that strangers who arrive from a high latitude are far more predisposed to yellow fever than those inhabiting lower latitudes. This has been well shown by Barton, who, in giving a table of the deaths per 1000 by the epidemic at New Orleans in 1853, shows that it was as follows:—

Native Creoles, ...	3·58
Strangers from South America, . .	6.14
" " Middle States of Union, .	30.69
" " France, ...	48.13
" " Netherlands, . . .	328.94

Cause.—The disease is caused by living organisms which require heat for their vitality. A strepto-coccus has been discovered. The cocci ·6 to ·7μ. occur in chains, or aggregated in masses, with distension of the vessels of the kidney and liver (Cornil et Babes' "Les Bacteries," 1885). These organisms have been found very numerous in the tubules of the kidney, together with degenerated and desquamated epithelium, to the hinderance of the excretory process.

Differential Diagnosis.—It is only necessary to separate yellow fever from remittents and intermittents as tropical fevers. These also arise from the emanations of the holds of ships—a foul hold in a sailing vessel, or coals in a steamer. When there are heavy rains there is no yellow fever; it is perturbed and

suspended, but remittents and intermittents are not greatly affected. Note how in yellow fever the functions of the colon, kidneys, and liver are suspended: in the colon there is cessation of secretion of colouring matter; in the kidneys there is desquamation, as also of the urinary tubules and bladder. In yellow fever, there is liability to suppression of urine; the alvine discharges devoid of natural appearance; the black vomit supervening when alvine and urinary secretion assume their peculiarities. Blood issuing from the skin, rectum, etc. There is suppression of urine. You note the yellowness of the skin on the fourth or fifth day. There is early active exudation into the liver. Note the critical efforts, and the crisis. A case may begin as remittent, and afterwards develop into yellow fever. There may be typhoid complications. Remittents and intermittents may go on where yellow fever seems to have cleared away.

The following is a table showing the various mortality of fever in :—

WINDWARD AND LEEWARD COMMAND.

	Mortality.
Intermittent, ...	1 in 169
Remittent, ...	1 in 9
Yellow Fever, ...	1 in 2⅓

JAMAICA COMMAND.

	Mortality.
Intermittent, ...	1 in 163
Remittent, ...	1 in 8
Yellow Fever, ...	1 in 1⅓

GIBRALTAR.

	Mortality.
Intermittent, ...	1 in 60
Remittent, ...	1 in 11
Yellow Fever, ...	1 in 1⅔

(Sir A. Tulloch.)

The mortality was one in three of those attacked in Havanna (Europeans), and one-third of 4000 in the Spanish quarters. The mortality is never so heavy on board ship.

The **Prognosis** is never surely favourable: a slow pulse, moderate temperature, and quiet stomach are good signs, more especially if the urine continues fair in amount; but the usual severe phenomena of the disease may supervene and carry off the patient at any time.

Treatment.—The remedies employed are empirical more than anything else. The first principle is to seek to eliminate the poison; second, to check the disorganization of the blood—Sir Ranald Martin says, 'decarbonize the surcharged blood globules and rouse the capillary circulation;" third, to sustain the body in its struggle with the foe. In the first stages calomel should be given in combination with quinine, capsicum, and stimulants. A strong tumbler of hot rum has been known to cut short the disease. Calomel is the first medicine we look to to disgorge the organs:—

℞ Calomelanos, grs. vi–x.
Jalap, grs. x.
Zingiber, gr. iij. M.

Dr King used turpentine in small doses with success in Bermuda in 1843. It was first suggested by Dr Copland, writing in the "Medical Times," 14th April 1855. Given internally combined with oil, and applied externally as well, it is found of great effect: the dose is 20 minims to 1 ounce. John Hunter pointed out how turpentine restrains passive hæmorrhages; it acts as a sudorific and diuretic, and the oil is emolient to mucous membranes.

Give—

℞ Salad Oil, ½ pt., (for mucous membranes).
Sol. Chlor., ʒ j., (antiseptic).
Lime-juice, ʒ ss., (for blood).

or

℞ Quin. Sulph., gr 1, not in vomit.
Myrrh, gr 1,
Camphoræ, gr 1, } (for anxiety and restlessness).

The following medicines, etc., are also given :—

Ammonia Acetatis, Potass. Nitrite, Spiritus Ætheris Nitros, Syr. Scillæ, Hyoscyamus, Chloroform (for stomach), Creasote, Hydrocyanic Acid, Chlorodyne, Nitro-Muriatic Acid—internally and externally (Sir R. Martin), Strychnine (for vagus), Potass. Chloridum, Phosphoric Acid, Lime-water, Beef-tea, Champagne, Ice, Pepsin, Ferri Perchloridi, Secale cornutum, and bland food frequently and in small quantities. The Hydrochlorate of Cocaine, as mentioned by Thorington, is not to be overlooked as a useful adjunct.

They are given according as circumstances arise, and symptoms require to be met, and the intelligent physician will select each or several as required, besides other new remedies to meet symptoms, such as are now used in tropical fevers. Dr Warburg's tincture (1849) might be mentioned.

I give the formula as quoted by Maclean ("Tropical Diseases").

℞ Aloes (Socot.) libram.
Rad. Rhei (E. India).
Sem. Angelicæ.
Confect. Damocratis ana uncias. quatuor (of the London Pharmacopœia, 1746.
Rad. Helenii (S. Enulæ).
Croci Sativi.
Sem. Fœniculi.

Cretæ præparat ana uncias duas.
Rad. Gentianæ.
Rad. Fedoariæ.
Pep. Cubeb.
Myrrh elect.
Camphor.
Bolet. Larcis ana unciam.

The above to be digested with 500 ounces proof spirit in a water bath for 12 hours, then expressed and ℥x disulphate of quinine added; the mixture to be replaced in the water bath till all is dissolved. After filtering it is ready for use.

Ætiology.—Questions relating to contagion, and the disease being purely localised have arisen. I give a mass of evidence relating to the fever on shipboard and land. The origin of the fever is a "specific miasma" which infects the body of the victim, and reproducing itself develops the phenomena of the disease and runs its course. It is generally supposed to be a purely endemic disease—*i.e.*, that it is peculiar to certain localities and places and attacks only persons brought under its influence there. Take for example a copy of the despatch from the Governor of Bermuda relating to the spread of yellow fever in 1856. A Commission investigated the recent epidemic, and state "that the epidemic was not introduced *ab extra*, but originated in the colony itself. It appeared at several points; at two such simultaneously. It did not spread through contagious infection, but through the same causes that had given rise to it. There was an inferred epidemic constitution of the atmosphere, exploding in attacks of the disease, where it encountered the other required elements, coupled with predisposition in individuals, bad sanitary condition in localities, or both." They conclude by saying, "Nothing is known to prevent its recurrence,

but more organization is required for sanitary arrangements, and that quarantine should not be applied to the crews of ships, but to the ships themselves, with yellow fever."

Mr Lawson, Inspector-General of Hospitals, describing an outbreak in Jamaica, 1856, says, regarding the ætiology, "Healthy persons are very liable to the attack. If they returned to a healthy locality they went through the fever, but did not communicate it." Referring specially to the military station at Newcastle, he describes in detail:—"In a cantonment on the narrow crest of a mountain were four ridges, and between them three zones, these alternating with the ridges, and 800 yards apart. The soldiers living in the three zones suffered much from the epidemic, while those living on the four ridges enjoyed perfect immunity;" and he goes on to say, "the immediate cause is some emanation given off in these certain localities," and denies the conclusions of Humboldt, that it occurs at 4000 feet above sea level. He says yellow fever is not contagious, and instances it by pointing out that 38 per cent. of the men who attended the fever cases died, while of those who did not attend, 43 per cent. died." He disproves the high temperature theory, that it only occurs at 80°, and says it occurs 15° lower. He points out how the fever is suspended under heavy rain, and "arises from local causes."

The most frequent months of the fever are August and September. It would seem to be contagious in the following cases—"Two patients were brought from the lower ground on the 17th and 18th September. Two men in the same ward of the hospital were attacked, and it killed forty-five persons

that year. Personal communication," he says, "was not necessary." Regarding the yellow fever at Sierra Leone in 1847, he says, "it was confined to the lower part of the town and was not contagious," and he applies the same to all the epidemics there — viz., 1823, 1829, 1837, and 1847. "A factor of very extreme operation became active from time to time, determining the production in suitable localities of emanations, the immediate cause of the disease, and this noxious influence extended to ships at 150 to 200 yards from the shore. Though quarantine established, yet cases broke out on board." He also remarks how the fever "made its appearance among the shipping at Kingston, and in spite of precautions." Troops go up to the mountains to avoid the fever.

Surgeon-General Maclean in writing upon the epidemic at Bermuda in 1864, dwells upon the very insanitary state of the town and garrison. "There was not even wanting the adjoining churchyard."

The same writers give conflicting evidence regarding the St Domingo epidemic of 1802. M. Bally "is doubtful;" in 1804 he denies contagiousness; in 1821 it is admitted. M. Francois, writing from the Antilles 1811, says "it is contagious and is imported." Havanna is regarded as a centre for the fever. There are accounts of it breaking out among the Indians of the West Indies, and they took little trouble about it, regarding it even "as a scourge of the Deity." The fever is said to arise easiest from volcanic disticts where there is a light turfacious ashey soil, such as is found in some parts of Central America—*e.g.*, Rialego and Panama.

At Corfu in 1850 a few cases, no question of importation to suggest a doubt as to origin. The men

were engaged excavating the soil and working amongst decomposing matters.

Bancroft (Essay, 1811) denies that the virus is brought by human effluvia. He says if it were strictly contagious it could not be generated by filth, putrifying agencies and human life. Sir William Pym affirms it, and disallows the presumed generation of the virus. Decomposition is not quite contagion. These things do not of themselves generate it. Sun and the soil, plus marsh miasmata, and new comers, give rise to yellow fever. He also points out how the contagious powers of yellow fever are destroyed by cold air. Rain and cold put a stop to the Philadelphia epidemic of 1793, and that of Gibraltar in 1810. It is said to break out like the attacks of rabies, glanders, whooping-cough, etc. When it breaks out in several places there must be the production of a similar substance to cause it. It is quite possible it may break out under certain circumstances, and we can infer something similar when we think of the Black Hole at Calcutta, the sweating sickness which followed Richmond's army, the lues with Charles VIII.'s, the malignant fevers of Napoleon's, hospital gangrene, etc. There would seem to be a virus in yellow fever. Rochoux says ("Dictionaire de Medicine"—see "Contagion") Contagion belongs to pestilential fevers and there must be a germ, which germ is easily destroyed. Tweedie ("Cyclopædia of Medicine") says the human body develops a fever principle. Herman writes "Diseases acquire property of propagation by crowding, filth, etc., and tropical heat." Copland says, "Disease is produced by infectious or contagious agents, and may be modified by causes, such as fever epidemic sources, and from animal effluvia, emanation from patients, and these aggravate

the sickness and make it malignant." There may be a marsh yellow fever which is not contagious. Some think that there were two diseases on board the "Kant," "Scout," "Bann," and "Eclair." Sir Wm. Pym shows marsh fevers to occur after June, and it is in July when yellow fever often breaks out. There are proofs of the universality and contagiousness of yellow fever in inoculation, for it has been successful, and one attack protects against another. Fraecocestoro holds to there being a specific virus, and to the contagiousness of the disease. It would appear that there is a poison and a nidus, for these can be destroyed. Compare with other contagious diseases—"Typhus is killed by heat; cholera, by moisture; small-pox, by dry air; yellow fever, by cold.

Quarantine has not been found of much use, and, where internal measures of prevention are properly attended to, it can be abandoned. Quarantine must be 300 yards from the shore, and thirty days must be given to cause the virus to leave the ship. Twenty days must elapse since the cure of the last case or death. It would appear that there is something more than local causes. Instance, the epidemic of 1856 in the West Indies, and around Gulf of Mexico. At the same time there was a spotted hæmorrhagic yellow fever near the Peruvian Andes, and the morbific agency was widely diffused (Dr Smith in the "Edinburgh Medical Journal").

The following is evidence that the disease can be imported. It broke out at Lisbon in 1857, when there were 5000 deaths (13,500 attacked). Ships were coming into port from the Brazils, the Americas, the West Indies, and Africa. So in the outbreak at St Thomas in 1858. The ship "Pavu" came with seven

cases on board, and there ensued "large numbers of deaths of valuable lives," as the account proceeds, "those who make the wealth of the community and give it power;" and it speaks "of the magnitude of the subject," and agrees that "yellow fever is one of the most mysterious diseases flesh is heir to." They account for it by the sewage, drainage at fault, excrementitious filth, debris of food, hot sun and tides—all these causes at work; the noxious effluvia being the very essential of the deadly element. For Government purposes it was not called infectious. We shall shortly see how ships coming from such places have brought the disease even to our shores.

Take, for example, the investigations regarding the yellow fever at New Orleans, some years ago. The fever was brought by a ship from the West Indies. The disease broke out in groups, wherever there was violation of the laws of health, and very insanitary influences at work. There was no case *de novo*. Here was importation and transmission due to human intercourse. It is remarked how the natives in the West Indies fly from a stricken person. The investigations state it is brought by fomites, as cotton bagging, goods, bedding, blankets. They found disinfectants valueless, and prophylaxis was no good; but quarantine, if rigorously carried out, was efficacious. They state (the American observers) that the bodies of those labouring under, or who have died of, the disease, do not give off a poison capable of producing the disease. Frost and cold kill the germ. A refrigerating ship was suggested for reception of invalids. In 1878, 100,000 persons were stricken down, and 20,000 perished, making a net loss to the country of $100,000,000.

I must refer shortly to a few historical cases on shipboard and to further evidence of contagion. The ship "Hankey" from Bulam (West Africa), 19th February 1793, brought the fever to the Island of Granada, where it broke out some days after. There had been fever at Bulam. H.M.S. "Baun" brought the fever to Ascension Island, 1823. She had come from Sierra Leone. The garrison lost 21. The fever attacked persons at Passages in Biscay, on opening her hold. She had come from Havanna. The outbreak at Swansea, 9th September 1865, was the result of infection brought from Cuba by the "Hecla." The infection was traced to Port Royal, due to visits from a diseased ship. The case of the "Eclair" (steamer) 1844 is historical. From December of that year till July 1845 she was watching for slaves off Sherbro, Seabar, and Sierra Leone. Boats were sent up the rivers at these places, and she was six miles off the shore. From 3rd April to 10th June 1845, there were thirteen cases of fever, and ten fatal; the sufferers were men employed in the boats. On the 4th July 1845, at Sierra Leone, several men slept on shore; and they were clearing out the "Albert" of the Niger Expedition. They anchored off the coast till 9th August 1845, and there were fifteen cases, and six deaths. They left the Gambia on the 15th. They were at Goreé on the 16th—21st at Boa Vista, Cape de Verde Islands, and there were five cases of fever on board. Crew landed at the capital town, but because of increase in the number of cases they re-embarked 13th September, and sailed for England 23rd September, and were put in quarantine. There were forty-one cases and twelve deaths on the voyage, and at Motherbank there were nine fresh cases and four

deaths. The fever was set up in Boa Vista, and the soldiers were first attacked, then persons living near the sick. The incubation period was here remarked to be from two to eight days. A merchant taken on board, and who died, was blamed for introducing the fever. These facts speak for themselves. Intercourse with the shore may carry the disease. The "Eclair" lost seventy-four in all.*

I have not space to discuss the durability of yellow fever, its power of diffusion, and its rapidity of propagation. Seclusion and separation kept back the disease, as was proved at Gibraltar in 1813. Yellow fever would seem undoubtedly to be a specific contagious disease and probably due to some subtle micro-organism which thrives and multiplies in the air under favourable circumstances. It may grow and fill the whole air with a deadly miasma, and be like a patch of vegetation, and whoever comes under its baneful influence, receives the poison into his blood, and as he rarely succeeds in throwing it off, he must succumb. It is not a disease of water, like cholera and others, but is more a "fœecal" one, as pointed out by Dr Parkes. It only remains for a scientific committee to investigate the malady, discover the exact organism causing it, and to point out how it can be combatted, at the same time laying down prophylactic measures to be adopted by those who cannot avoid putting themselves in the way of running the risk of the noxious infection.

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* In the case of the "Eclair" it may have been spread through imperfect sanitation, evacuations from some of the patients may have been allowed to remain on some of the clothing, which was not destroyed. So that the theory of contagion from one person to another would not hold.

Since writing the foregoing, I have had an opportunity of visiting some of the haunts of yellow fever, particularly on the Guinea Coast of West Africa, where it has been epidemic at times.

The most common fevers met with there are the ordinary intermittent and remittent malarious fever and the bilious remittent. In this last, the conditions approach a type bordering on the actual phenomena of yellow fever. There is vomiting, which is serious —quantities of blood at intervals accompanying the copious bilious matters ejected; and the depression, along with other signs, makes one dread the actual onset of yellow fever. The diagnosis not establishing itself, however, one does not dread the likely fatal termination one would in actual *causus*. Together with this vomiting there is epigastric tenderness; and the quantities of blood ejected at intervals, though not necessarily changed blood, are themselves two alarming symptoms. Then in those cases of fever, not yellow fever, there are insomnia, delirium, subsultus, sallowness of the skin though no icterus, and a marked heat to be felt, almost pungent. This brings before one's mind a diseased state of the blood, which closely approaches that of yellow fever. The urine voided may be like blood, but it rarely becomes scanty as in yellow fever. A true distinction can always be drawn between yellow fever and the other forms of malarious poisoning, as they never merge into each other so as to become another type of fever. In ordinary malarious fever you will get pain in the region of the stomach and in the right lumbar region, pains in the back and limbs; the patient may be in contortions, and constantly restless; the vomiting continuing, at times bilious, a black

discharge will sometimes be belched up and the patient passes into a faint, if not actual syncope. In other cases yawning, hiccough, and delirium, together with high fever and hot skin, and in a rare instance the patient may be slightly tinged with jaundice; but here, other things being equal, the tendency is to recovery and not to critical fatality or yellow fever. Some patients may also have bloody mucous stools; others, hæmaturea. The potency of quinine, which retains its efficacy in severe malarious cases, is also a strong factor; for the drug is not a specific in yellow fever. A retrocession of the symptoms sets in if the case is not hopeless, as it might be in a person otherwise enfeebled, and then quinine makes itself felt. It is not so in yellow fever.

Bilious remittent and yellow fever have many points in common. The following are points of difference between remittent and yellow fever :—

BILIOUS REMITTENT.	YELLOW FEVER.
Attacks repeated.	One attack.
Remission of paroxysms.	No paroxysms.
Source—All malarious districts.	Source—Contagion, which may be carried beyond malarious districts.
Character never changes.	Character changes with each epidemic.
Hæmorrhages—none.	Hæmorrhages from all the mucous surfaces.
Vomited matters bilious.	Vomited matters contain elements of pure blood.
Jaundice from outset.	Jaundice from third to fifth day.
Spleen often enlarged.	Spleen not enlarged.
Skin colour due to bile.	Skin colour due to decomposed blood.

BILIOUS REMITTENT.	YELLOW FEVER.
Urine — Blood often appears, containing bile.	Urine—No blood, but albumen and casts.
Pulmonary congestions occur.	Pulmonary congestions do not occur.
Pulse keeps pace with temperature.	Pulse does not keep pace with temperature.
Quinine counteracts the disease.	Quinine does not counteract the disease.
Death seldom before the eighth day.	Death may be looked for at the third day or before.

Differential diagnosis between Dengue and Yellow Fever—the following being the essential points.

DENGUE.	YELLOW FEVER.
Pains rheumatic-like.	Pains not flying from joint to joint.
Tongue strawberry.	Tongue coated all over.
Epigastrium not tender.	Epigastrium—great tenderness.
Vomiting not a prominent symptom.	Vomiting a prominent symptom.
Skin covered with a rash, spots disappear on pressure, rash recurrs on fifth or seventh day.	Skin—no rash. Skin yellow.
Urine is normal.	Urine contains albumen.
None.	Hæmorrhages from mucous surfaces.
Insomnia, jactitation, hiccough, absent.	Insomnia, jactitation, hiccough, present.
Convalescence slow.	Convalescence rapid.
Duration, eight days.	Duration, nine to fourteen days.
Relapses occur.	One attack.
Quinine of benefit.	Quinine rarely beneficial.

The following are some of the causes which bring on attacks of fever in malarious countries, where it is known to occur; but different individuals, subjected to the same influences, exhibit different manifestations of disease :—

1. Exposure on the banks of a river after sunset; the miasmata which arise readily poison the system.

2. Lying or resting on the ground after enduring fatigue.

3. Working in the sun's rays without sufficient protection to the head or back.

4. Getting wet feet and not changing after a wetting to dry them. A chill is to be avoided.

5. Exposure to the miasma of newly turned-up soil or disturbed dead leaves and decaying vegetable matter.

6. Exposure to the night air after sunset, if one is unprotected by a roof or head covering.

7. The wind, which blows over marshes and low-lying places, and where rivers are at times in flood, brings much malaria. It has been noticed that in some tracts, such as these, lizards are absent, but what connection exists between the unhealthiness and this fact, I am unable to state.

8. The heavy dews of night are dangerous. It is well not to breathe through the mouth when in the open at such times. The onset of the rains is a fertile time for fever.

These are amongst the most common sources of malarious fever; and much the same applies to yellow fever, particularly at the seaboard where there are human habitations and shipping. The effluvia which comes from decomposing matters close to the tide line, such as collect at wharves exposed to the sun's rays, is a fertile source of yellow fever, and particularly when the epidemic has previously visited that locality or neighbourhood.

Going out the day after recovery is a sure way of bringing back the fever, and it will then be more

apt to assume the remittent type; or yellow fever, if prevalent, may set in.

Of cases on ship-board I cannot speak. If a ship comes to an infected district, and is in communication with the shore, there is liability to infection. It would seem that the air three miles out to sea may be laden with malaria. The mortality from yellow fever is much less at sea, particularly in the naval marine, as statistics show:—

BERMUDA, 1864.

	Population.	Attacks.	Deaths.
Sailors,	98	46	15
Marines,	98	12	3
Civilians,	335	146	10

LEEWARD ISLANDS, ANTIGUA, 1888.

Yellow Fever or other Epidemic Diseases. — Number attacked, 94; of whom 10 died.

AT BERMUDA, 1864.

Difference of Deaths at Hospital and outside, showing the advantages of Marquees over Hospitals—

	No. of Cases.	Deaths.	Ratio per 1000.
Hospital at St George,	291	107	367·69
In marquees and at Ferry Point,	61	17	278·68

AMONG TROOPS AT BERMUDA, 1864–65.

	Number.	Attacks.	Deaths.
Officers,	74	40	14
Men,	1147	511	173
Women and Children,	228	79	15

According to Horton the mortality in yellow fever varies greatly in different epidemics. He says, "Of 134 cases in which the disease occurred, the deaths were 130; of 1739 cases, 1265; of 35 cases, 34; of 20 cases, 19; but in 16,517 cases, 6684 died."

"At St Domingo, in 1795, 700 British officers and 30,000 soldiers died," so says Sir W. Fergusson. In 1804, at Malaga, 11,486 died out of a population of 36,054. In the previous year, 6684 died out of 16,517 persons attacked. In 1812, 118 persons died out of 422 treated at Brimston Hill, St Christophers. At Barcelona, in 1821, hardly a patient survived in the wards under the French Commission. At Seminaria, where French and Spanish Physicians were in charge, 1265 patients out of 1729 died.

Horton gives the following summary of mortality among British troops :—

Windward and Leeward	Intermittent Fever.	Remittent Fever.	Yellow Fever.
Command,	1 in 169	1 in 9	1 in 2½
Jamaica,	1 " 163	1 " 8	1 " 1⅓
Gibraltar,	1 " 60	1 " 4	1 " 1⅔
Malta,	1 " 311	1 " 24	—
Ionian Islands,	1 " 236	1 " 22	—
Upper Canada,	1 " 1143	1 " 11	—
Lower Canada,	1 " 535	1 " 5	—

The rate of mortality for yellow fever on the Island of Boa Vista is as follows :—

Spaniards, Portuguese, and French, -	1 in 2·28
English and Americans, - - -	1 " 1·6
Native Population—Slaves, - - -	1 " 33·4
Native Population—Free, - - -	1 " 14·6

The following were the deaths from yellow fever at Gibraltar :—

	1804.	1810.	1813.	1814.	1828.
Military, and their Families,	869	6	391	114	507
Civil Inhabitants, - -	4864	17	508	132	1170
Total, - -	5733	23	899	246	1677

The following is an instance of increased mortality through yellow fever, which occurred at New Orleans

between 1844 and 1880. Of 19,233 cases of yellow fever treated at the Charity Hospital, New Orleans, the deaths were 9,667 or 50·2 per cent. The total number of cases which were treated in the hospital during this period was 304,213, and the deaths were 43,718 from all causes in the thirty-four years. In the city of New Orleans for the same period the total number of deaths were 242,739, of which 28,739 were from yellow fever; and the total deaths from all forms of malarial fever were 12,413.

A word must be said regarding micro-organisms and yellow fever. Cornil and Babès found chains of diplo-cocci in the capillaries of various organs, in a case of yellow fever; but in other cases which they examined they were absent. Later on, however, Babès found in two cases short rod-like bacilli in the mucous membrane of the small intestine, very similar to the typhoid bacilli.

Domingo Freire regards a micro-organism, which he has named *crypto-coccus xanthogenicus*, or *micrococcus amaril*, as the cause of yellow fever; but Flügge says that his observations are based upon errors. According to Duncan, who has done good service by his investigation of yellow fever—and whose work * I have to thank for what follows—these may be thus briefly summed up:—

"In 1883, Freire reported to the Biological Society of Paris that he had found a specific microbe in cases of yellow fever, and that he had proved his point by experimenting on guinea pigs. He also ascertained that the microbe secretes an alkaloid, which when attenuated, can be used for preventive inoculation. These organisms, Freire has also found in the soil in districts where yellow fever is prevalent.

* "Prevention of Disease in Tropical Campaigns."

"From 22nd December 1884 to 22nd March 1885, or a period of three months, 1109 persons of different nationalities, whose ages ranged from one month to sixty years, have been inoculated in the deltoid region; not one single case was severely attacked, and the very few who had suffered from the fever had it in the mildest form possible. In many cases the process was performed in houses where a few hours before deaths had taken place from yellow fever."*

Again, the results in December 1885, and January and February 1886, are as follows:—

"Among 3051 subjects inoculated in Rio de Janeiro there were no deaths, whereas in the same localities and houses 278 unprotected individuals died of the disease. Lastly, Dr Mastier found in Rio de Janeiro the mortality to be 1·6 per cent. amongst those inoculated, and 13·7 per cent. amongst those not protected."

Drs Giraud and Carmona use inoculations as advised by Freire, and with good results, and when we know that the usual mortality is at least 50 per cent. we cannot fail to appreciate Dr Freire's labours in curbing this fatal disease, and urging the general introduction of inoculation.

It remains to be noticed that Carlos Finlay, of Havana, made experiments with the mosquito in the prevention of yellow fever. He caused a mosquito to sting a yellow fever patient and a person who was to be protected, and the result was that many of the persons thus inoculated remained free from yellow fever though exposed to it; several had slight non-fatal attacks, and only one died of malignant yellow fever, seven months after the inoculation; but in him the inoculation had shown no result. If two mosquitoes are used the attack of fever resulting is very severe. Finlay holds that yellow fever is never

* 1890—In 10,881 inoculations, the death-rate was 0·4 per cent.

found where tropical mosquitoes do not exist, and thinks that they aid very materially in causing its epidemic spread.

Such are some of the attempts to reach the micro-organism of yellow fever and to bring about an amelioration of the disease. Everything tends to show that the workers are proceeding in a right direction and the scientific value of their labours is beyond dispute. With such encouraging results our scientific knowledge of yellow fever will be more and more widened, and as surely as Jenner brought to bay such a terrible foe to human life as small-pox, so we may hope that this malignant scourge in tropical climes will recede and be rendered inert before the advance of the Medical Science in the near future.

EXAMPLES OF BILIOUS REMITTENT FEVER AND YELLOW FEVER.

Case I.

H. C., aged 39 (see Temperature Chart, No. X.).

23rd May.—The patient complains of being sick, and cannot take food. In bed.

24th May.—Still sick. Has headache, diarrhœa, and pain in region of liver. Salts and other remedies tried without effect.

25th May.—Vomiting troublesome; headache persisting, patient feels ill. At 1·30 p.m. retained some Brand's essence of beef. Bowels still loose; enema of brandy administered as a restorative. The following medicines have been tried, but not retained—Chloral hydrati, antipyrin, bromide of potash, calomel. Bismuth retained. Temperature 100·6° and 100·8°; pulse 81; fairly steady, skin moist. Complains of sleeplessness and restlessness.

26th May.—Patient much worse during night. Has to rise in night time; complains of diarrhœa. Vomiting; vomited matters at first clear, afterwards green bilious matter, also frothy matter. Patient goes into a state of contortions and twistings, legs drawn up, and at times great pain. The pain referred to right lumbar region. The breathing affected. 2 p.m.—Temperature, 101°; pulse 90. The pain shifts. Hot cloths applied, afterwards sinapisms. Liq. opii sedative administered. The pain somewhat relieved. Patient very weak; champagne given. Headache persisting, very commanding. 6·30 p.m.—Temperature, 100°; pulse 90. Patient became calmer. Later temperature, 99°. Some little sleep during night.

27th May.—Temperature still 99°. Patient markedly weak and exhausted. Sent home to England.

Case II.

E. T., aged 23 (see Temperature Chart, No. II.).

13th October.—Went down to work this morning, but returned. Felt unwell, took a little wine. After returning from work, lay down. Felt his stomach aching; bowels loose; pain internally; no appetite. At 12·45 p.m., patient vomited first white and clear matter, then a large quantity of dark bloody matter, before this he had vomited several times. The wine he had drank seemed to float on the top. He vomited a curdy-like fluid. Patient passed into a faint, and became unconscious, when he vomited the dark fluid. Eyes turned up, and pupils dilated. The breath smells of blood. Patient felt cold at 1·45; pulse 117; temperature not ascertained. Diarrhœa persisting; fæces very feculent, dark, and watery. Spirit. ammoniæ aromaticus was administered, and the skin bathed with mustard and hot water. Egg and brandy afterwards administered—champagne given. Cramps throughout the attack; palpitation and pain in region of stomach. The vomited matter examined microscopically, contained fibrin, blood discs, fragments of food and peculiar rounded masses. Afterwards patient fell into a sleep; and perspiration setting in, he felt better. The following day he soon began to improve.

In bilious remittent fever the vomiting and stomach symptoms are very marked. The vomited matters may be clear, frothy, and partly bilious. All that is taken being vomited, and bile superadded. Again it may be very markedly bilious, and green like spinach, this continuing for a lengthened period, and distressing patient and physician alike. At other times the vomited matters are thick, mucoid, tawny, and mixed with blood, and soon blood is conspicuous in the ejected matters. The skin all the time remains dry, pungent and hot, being even tinged with jaundice. The tongue is foul, the bowels are loose, and the urine is high-coloured and scanty. Headache is persisting, and the temperature keeps high; the pulse also requires watching. Measures must be pushed to get the skin to act, and it rarely happens that when once this is attained and sweating re-established, the severe symptoms begin to abate, and with proper management the case is brought under, and recovery quickly follows. In some cases the urine will be bloody and "turning" like port wine in the vessel; but this must be considered apart from bilious remittent fever, and does not enter into the subject of this work.

These are some of the worst features experienced in bilious remittent fever; but in no case do you have the severe hæmorrhage, the extreme yellowness, the adynamic symptoms, and sudden collapse of yellow fever; and the administration of quinine at suitable times, and when it can be retained, is serviceable.

Case III.

P. L., Clerk, aged 28.—Has been healthy, excepting several slight attacks of ague, for the five months he has been from England.

First day.—On the first day of his illness he awoke feeling out of sorts; his head ached slightly, and he had no appetite for breakfast. He went to his work, but the headache got worse during the day, and at noon he had to take to bed. He was then attacked by pains in his back and legs. When seen at 8 p.m., the patient complained of rigors, very severe headache, and deep-seated pain behind his eyes. He had pain too in his back, abdomen, and legs, which were often attacked by cramps. He was constipated; tongue coated with dirty-white fur; face flushed, and skin dry and harsh; temperature 100·5°; pulse 80, full and regular. A purge of calomel and jalap was given and a diaphoretic mixture ordered to be taken every three hours.

Second day, 10 a.m.—Patient has had a fairly good night and is rather better this morning; his pains are less; the medicine has acted. Pulse is 78, but the temperature is still at 100°; skin acting slightly. At 3 p.m. the patient was suddenly taken much worse; all the pains returned with great violence; headache and pain in the eye-balls very severe, photophobia, conjunctivæ injected; temperature, 104°; pulse, 110; breath very foul; tongue coated with yellowish fur, with red tip and edges; skin hot and dry, slightly jaundiced.

Third day.—Patient has passed a bad night; bilious vomiting commenced at 5 a.m. and has been severe; bowels not moved; patient is restless and light-headed; very little urine has been passed; temperature, 105·2°; pulse 128, weaker, and irregular. Enema ordered; also hot bath, and a drachm of sweet nitre every two hours. 6 p.m.—Patient is very jaundiced; black vomit made its appearance at 4 p.m. Patient is in great pain; complains especially of pains in the stomach and cramps in the legs; but headache not quite so severe. Only 4 ozs. of dark-coloured urine have been passed; albumen, one-fourth. Patient terribly thirsty, but ejects fluid as soon as it is swallowed. Yellow fever smell noticeable. Hot fomentations ordered to abdomen, and loins to be dry cupped. Acid. hydrocyan. dil. ordered in an effervescing mixture. 10. p.m.—Vomiting and pain less, and patient generally more easy.

Fourth day.—Patient has had a better night, but early this morning vomiting recommenced. There has been slight epistaxis. Patient deeply jaundiced. Temperature, 106°; pulse 140, irregular and very thready. To take Ol. terebinth. 15 ♏; spirit. ether. nit., 1 drachm; mist. camph. 1 oz., every three hours. Cold to the head, gum water to drink, and turpentine fomentations to abdomen. Has not passed water since last note; 3 ozs. were drawn off; albumen one-half. 7 p.m. — Patient much better. Bowels have acted; and vomiting stopped; temperature, 103·4°; pulse 116, rather more regular. Cramps better, but headache still bad; sweating profusely.

Fifth day.—Patient has had a good night, only vomiting twice, but has still a feeling of nausea. Tongue still foul; crop of herpes upon his lips. 7 ozs. of urine were passed during the night; temperature, 101°; pulse 112, stronger. Patient sweating slightly. Brandy, egg, and beef-tea enema ordered.

Sixth day.—Since last note patient has gone on well. All his pains have left him; tongue is cleaning; he has no nausea; temperature, 100·8°; pulse 106. 26 ozs. of urine have been passed; albumen, one-eighth. Brandy and milk and beef-tea ordered by mouth; soap and water enema. Patient is much brighter, and jaundice much less.

Seventh day.—Temperature 99°; pulse 100, regular. Patient is doing well. A bitter tonic ordered. Bowels opened once since yesterday's enema.

Eighth day.—Temperature 99°; pulse 104. Patient feels very weak, but otherwise quite comfortable. Urine, 38 ozs.; albumen, a trace. Jaundice nearly gone. From this date patient progressed favourably to convalescence.

Case IV.

R. M. T., aged 22.—A sailor, belonging to a sailing vessel just arrived from England. He had gone ashore against orders, and been "on the spree" for three days. He was taken suddenly ill in the street with giddiness and vomiting.

On first seeing him at noon, he was found to be dull and stupid; face lived, eyes sunken and glazed. He was covered with a cold sweat; temperature, 97°; pulse 110, very thready and irregular; tongue heavily coated with dirty yellow fur. A hot bath was ordered; after which he was well rubbed with hot cloths, and then with brandy and quinine. He slightly revived, the pulse became steadier and rather firmer in character, but this improvement only lasted for an hour. He then complained of very great pain in the stomach, cramps in his legs, and commenced to retch and vomit. At first almost pure bile was ejected, but in half-an-hour black vomit ensued, and he suffered from bleeding from the nose and bowels. He passed some dark-coloured urine which contained bile and blood. He had had three or four severe rigors, followed by a convulsive seizure which lasted twenty minutes. At six p.m. the patient was in a state of great agony; the pains seemed unbearable, and vomiting was incessant. Turpentine was administered by mouth and by enema, turpentine stupes were applied to the abdomen, and hypodermic injections of ergot were given, but without avail. At 7.30 he had completely collapsed, and died soon after 8 p.m.

POST-MORTEM.—The external appearances showed yellow discolouration of the whole body, with livid patches on the abdomen, and especially on the posterior surface of the body. The abdomen was moderately distended. The stomach and intestines were pale, but covered by livid patches. The liver which weighed 58 ozs., was congested, friable, and bile-stained. The gall-bladder was almost empty. The spleen was slightly enlarged; it weighed 8 ozs. In the abdominal cavity there was about two pints of what seemed to be fluid blood; the kidneys were slightly congested; the lungs were congested; the stomach and intestines contained a considerable quantity of black, tenacious, jelly-like material. Anti-mortem clots were found in the heart, which was softer than normal. The bladder contained a few ounces of blood-stained urine. The head was not examined.

Case V.

S. M., aged 27.—A young Frenchman, who complained of giddiness and fainting fits for the last two days, says that he has slight shivers at times and frontal headache, feels stupid and irritable, and has shooting pains all over the back.

First day.—His face flushed, and he has this morning vomited repeatedly a bitter frothy greenish fluid; this being accompanied by heartburn. The tongue is slightly coated; the bowels have not acted for three days; his skin is cold and moist, slightly jaundiced; temperature, 99·8°; pulse 86, irregular and feeble. He complains of shortness of breath and a slight cough. 10 grs. of calomel were given, to be followed by a saline draught. 9 p.m.—Bowels have been opened, but the patient is not better; he has more oppression and difficulty in breathing, feels colder, and is covered with a clammy sweat. He is greatly pained; his eyes throb, conjunctivæ somewhat injected; the yellow discolouration of his skin is more marked; he is very restless. 15 grs. of Dover's powder were given.

Second day.—Patient had rather a restless night, waking often on account of the cramps, which were very troublesome; he is very flushed; his head throbs; his eyes, which are protruded, water freely; the conjunctivæ are markedly injected. The tongue is covered in the centre by dirty-yellowish brown crusts; tip and edges red. A slight rash covers the body, being best seen on the chest and abdomen, and less seen on the back and extremities; temperature 106°; pulse 120, full and hard. Patient is excited and restless. Bowels just been moved—a copious, slimy bilious stool. Urine has been passed freely; it is dark in colour and albuminous. Patient complains bitterly of his pains, and suffers much from palpitation. Turpentine stupes ordered; 15 grs. of calomel and 20 grs. sulphate of quinine administered. Saline draught was again ordered. Night—Bowels have acted well and patient has had a fair day; but to-night the "white vomit" began. An effervescing mixture, containing acid. hydrocyanic. dil., to be given every two hours.

Patient very thirsty. Acidulated gum water ordered, and cold to head; temperature 105°; pulse 120, bounding in character.

Third day, noon.—Notwithstanding the vomiting, which has been severe, patient maintains his strength. Night—Pains less; vomiting has ceased, and patient is more easy; temperature, 104·4°. Pulse 100, regular, and rather softer. Patient is sweating slightly. 10 grs. of Dover's powder ordered.

Fourth day.—Patient had a good night, slept for four hours. He feels quite comfortable this morning. 6 p.m.—Patient is not so well, complains of great nausea; headache is much worse; photophobia. His nose bled freely this afternoon; temperature, 104·6°; pulse 118, full, and has lost its soft character. Tongue very foul; breath foul; skin dusky yellow. Turpentine in 15 drop doses ordered, with sweet nitre and camphor mixture.

Fifth day, 3 a.m.—Patient commenced the black vomit. He is delirious, noisy, and very restless, clamouring for drink which he cannot retain. He is quite powerless, and can hardly raise his head from the pillow. Urine drawn off, albumen a half (nearly). Patient has a wild look; his colour is nearly that of mahogany; temperature, 106·2; pulse very weak and very rapid. Turpentine enema and small doses of champagne to be given.

Sixth day, 4 a.m.—Patient was so much exhausted that death was thought inevitable. He was well rubbed with brandy and quinine, and ether injected. He was quite unconscious for over two hours; he then rallied somewhat, and is now (9 a.m.) lying quietly, conscious, and not complaining of pain. The vomiting has quite ceased. Nutritive enema given. He gets a little brandy and milk or beef-tea every half-hour. Night—Little change in patient's condition; if anything, he is a shade stronger. Turpentine stopped, and quinine in 3 grain doses given; temperature 104°. Enema repeated.

Seventh day.—Patient had a quiet night; temperature lower, 103·2. Since last note no urine has been passed.

Loins dry-cupped. Night—Rather more than an ounce of almost black urine drawn off; almost solid on boiling. Spirit. ether. nit., 1 drachm every two hours.

Eighth day. — Temperature, 102°. Patient was rather restless in the night; passed a few drops of urine at 7 a.m. Treatment continued and a warm pack given, after which patient slept for a few hours.

Ninth day.—Since last note patient has much improved. Slept well; temperature, 100·4°; pulse 100, still weak, but regular. No pain, no nausea; the tongue cleaner, but he experiences some difficulty in swallowing. Glycerine and borax for mouth. Nourishment increased, and champagne to be given frequently.

Tenth day.—Much better to-day. Urine passing freely; lighter in colour and less albumen; many casts seen.

Eleventh day. — This morning temperature was 102·6, probably due to a large boil which is forming just below his jaw on the left side.

Twelfth day. — Temperature still up. Complains much of pain in boil, which was opened. Evening.—Temperature, 100°; pulse 90, stronger. Enema was ordered, as bowels have not been opened to-day. Patient still very weak and listless. Nose bled slightly this afternoon.

Thirteenth day.—Patient not quite so well; heart sounds feeble. Digitalis ordered every three hours; thick soup and rice to be given.

Fourteenth day.—Temperature, 99·2°. Boil almost healed. Urine normal in colour, sp. gr. 1024, albumen a quarter; few casts seen.

Fifteenth day.—Patient much better to-day; was able to be raised in bed. Slight diarrhœa.

Sixteenth day. — Temperature normal; pulse 82, rather stronger. Digitalis stopped; citrate of iron and quinine and liquor strychnia ordered.

Twentieth day.—Patient going on fairly well, but he is still very weak. Colour of skin normal.

Twenty-third day.—Two large boils forming on abdomen; temperature, 101°. Port wine substituted for champagne; 10 grs. of quinine to be given night and morning in addition to the medicine.

Twenty-fourth day, evening.—Temperature, 102°. Boils opened.

Twenty-fifth day.—Temperature 99°. Patient is quite comfortable and cheerful to-day. He is taking his food well. Urine plentiful, but albumen still present.

Twenty-seventh day.—Patient allowed up for half-an-hour.

Twenty-ninth day.—Temperature normal. Patient doing well, but his mouth is still rather sore.

Thirtieth day.—Patient disturbed by slight diarrhœa through the night. Ordered ferri. per. nit.

Thirty-second day.—Diarrhœa better. Patient up for two hours. The patient's further progress was good, but very slow.

LITERATURE CONNECTED WITH MICRO-ORGANISMS.

Ca. Finlay:—El Mosquito Hipoteticamente considerado como Agente de Transmission de la Fiebre Amarilla: Pathogenia de la Fiebre Amarilla, la Havane, 1881–1882; Arch. de Med., Nav. xxxiv., 67–307, et xli., 421.

Apuntes:—Sobre la Historia Primitiva de la Fiebre Amarilla, 1884. Hematimétria en la Fiebre Amarilla, Cron. Med. Chir., la Havane, 1885.

Apuntes:—Lettre dans les Arch. de Med., Nav. de Juin 1885, p. 262.

Domingo Freire:—Et Experim. sur la Contagion de la Fièvre Jaune, Rio de Janeiro, 1883: Mem. sur les Inoculations Preventives, 1884: Doctrine Microbienne de la Fièvre Jaune et ses Inoculations Preventives. Rapport presenté au Government Imperial du Bresil, 1885.

De Arango Goes:—Sobre e Vaccinacao Contra a Febre Amarilla, Uniao Medica Maio, 1884; Arch. de Med. Nav. xli., 263; xlii., 315. Bulletin de l' Acad. de Méd., Sêance du 6 Mai 1884.

Rangé:—Et. sur l'epid de Fièvre Jaune ayant sevi aux îles du Salut. (Guyane) du 12 Fev. 1885 au 25 Juillet. Archiv. de Med. Nav. xlv., et lettre particulière.

Le Dantec:—Recherches sur la Fiévre Jaune, Critique des Theories Microbiennes Emisés en Amerique, th. de Paris, 1886.

Cornil et Babès:—Les Bacterieres.

Domingo Freire:—Archiv. de Med. Nav. 1882, xxxvii. Notice sur la Regeneration de la Virulence des Cultures Attenuées, du Microbe de la Fièvre Jaune.

The following support Freire:—

Issartier:—Rev. San. de Bord, 25 Fevrier 1886.

Ramon Suret:—Genio Med. Chir. Mars 1886.

E. Trouessart:—Rev. Scient, 8 Janvier 1887.

Domingo Freire:—P. Gibier et Rebourgeon, Ac. des Sc. 21 Mars et 4 Avril 1887.

Edinburgh: E. & S. Livingstone, Printers.

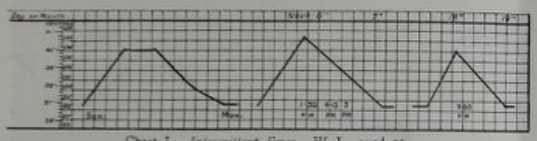

Chart I.—*Intermittent Fever.* W. L., aged 21.

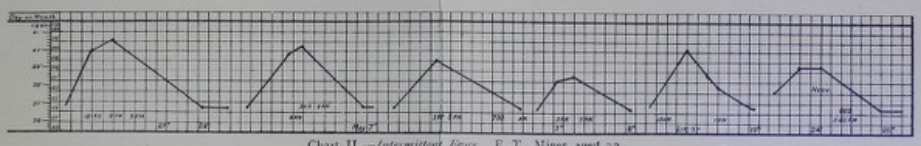

Chart II.—*Intermittent Fever.* E. T., Miner, aged 33.

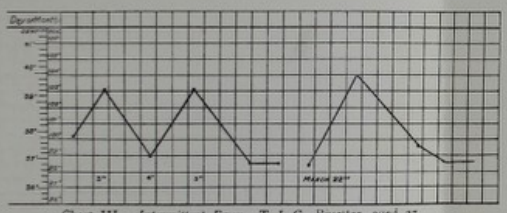

Chart III.—*Intermittent Fever.* T. J. C., Rivetter, aged 27.

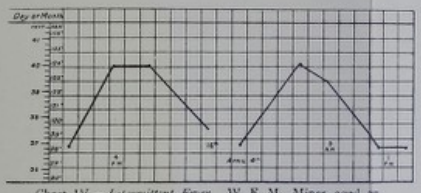

Chart IV.—*Intermittent Fever.* W. F. M., Miner, aged 37.

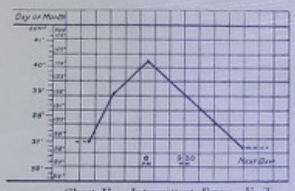

Chart V.—*Intermittent Fever.* E. T.

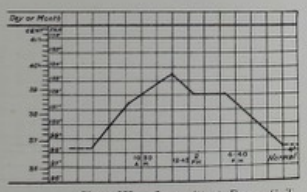

Chart VI.—*Intermittent Fever.* E. T.

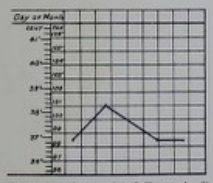

Chart VII.—Small Attack of Fever, in Patient inured to Malarious Country.

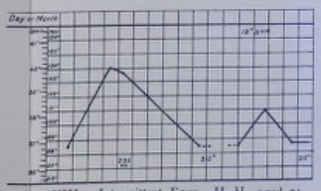

Chart VIII.—*Intermittent Fever.* H. H., aged 21.

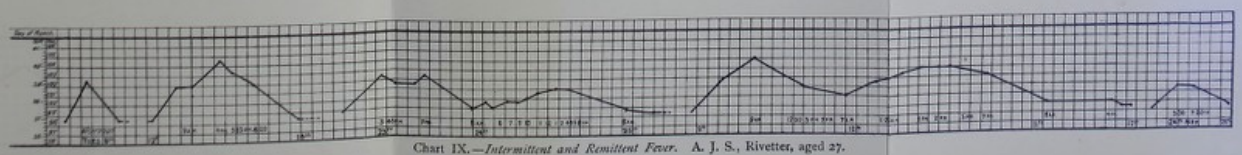

Chart IX.—*Intermittent and Remittent Fever.* A. J. S., Rivetter, aged 27.

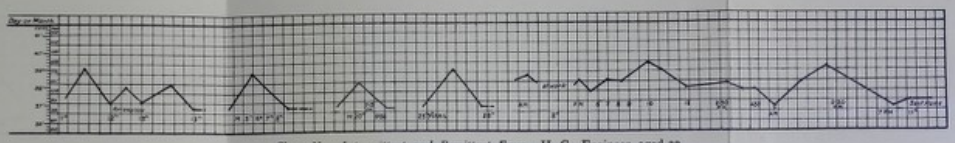

Chart X.—*Intermittent and Remittent Fever.* H. C., Engineer, aged 39.

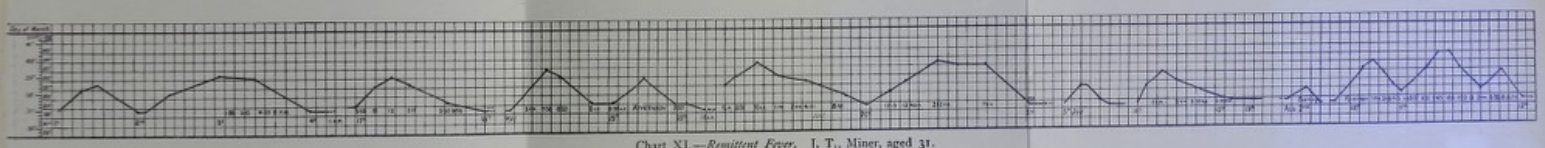

Chart XI.—*Remittent Fever.* J. T., Miner, aged 31.

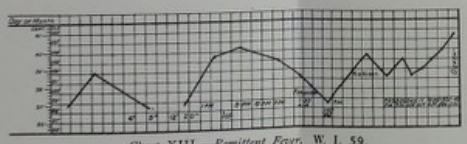

Chart XIII.—*Remittent Fever.* W. I. 59.

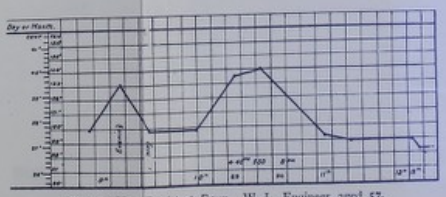

Chart XII.—*Remittent Fever.* W. L., Engineer, aged 57.

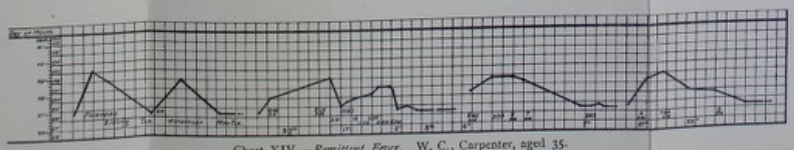

Chart XIV.—*Remittent Fever.* W. C., Carpenter, aged 35.